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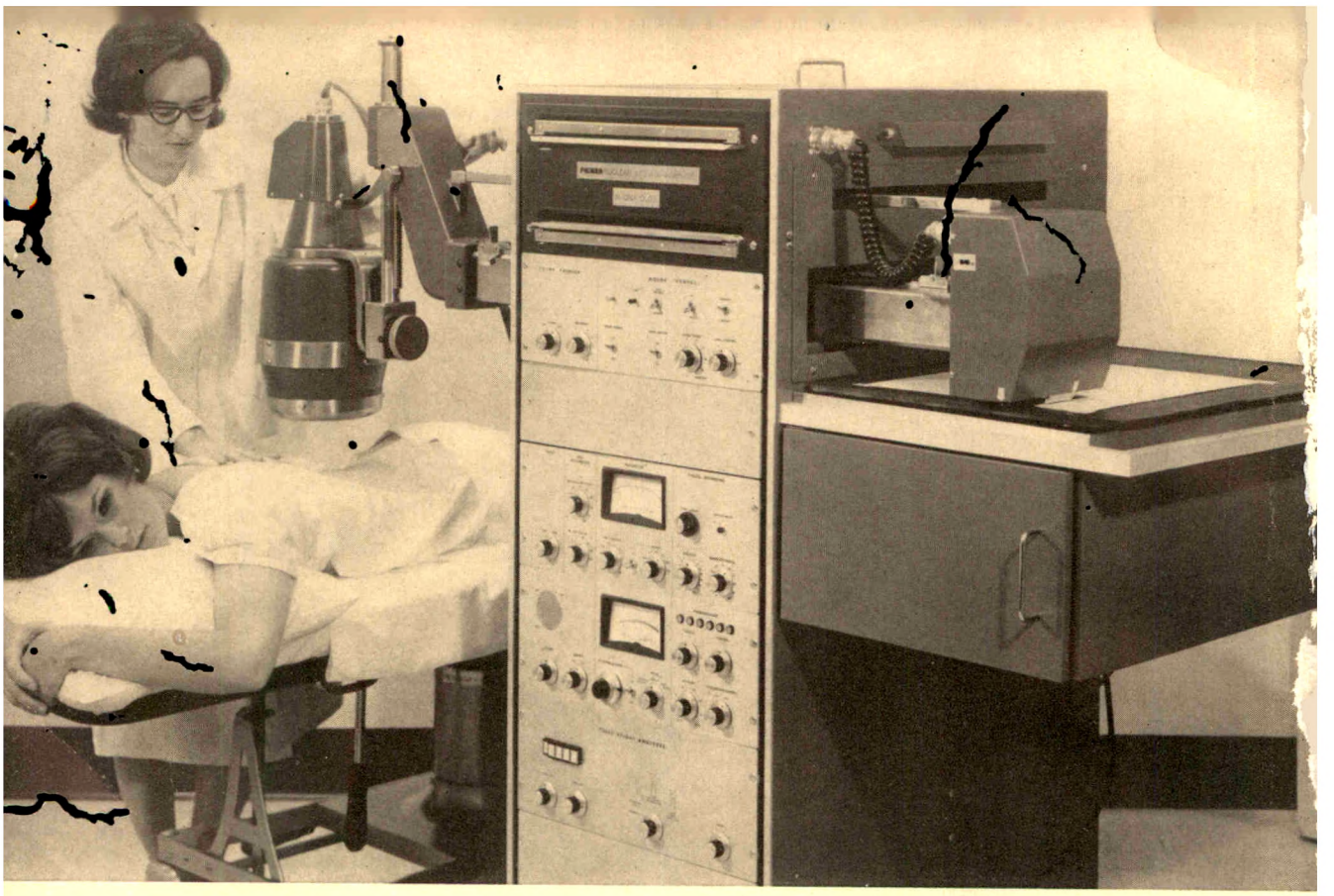
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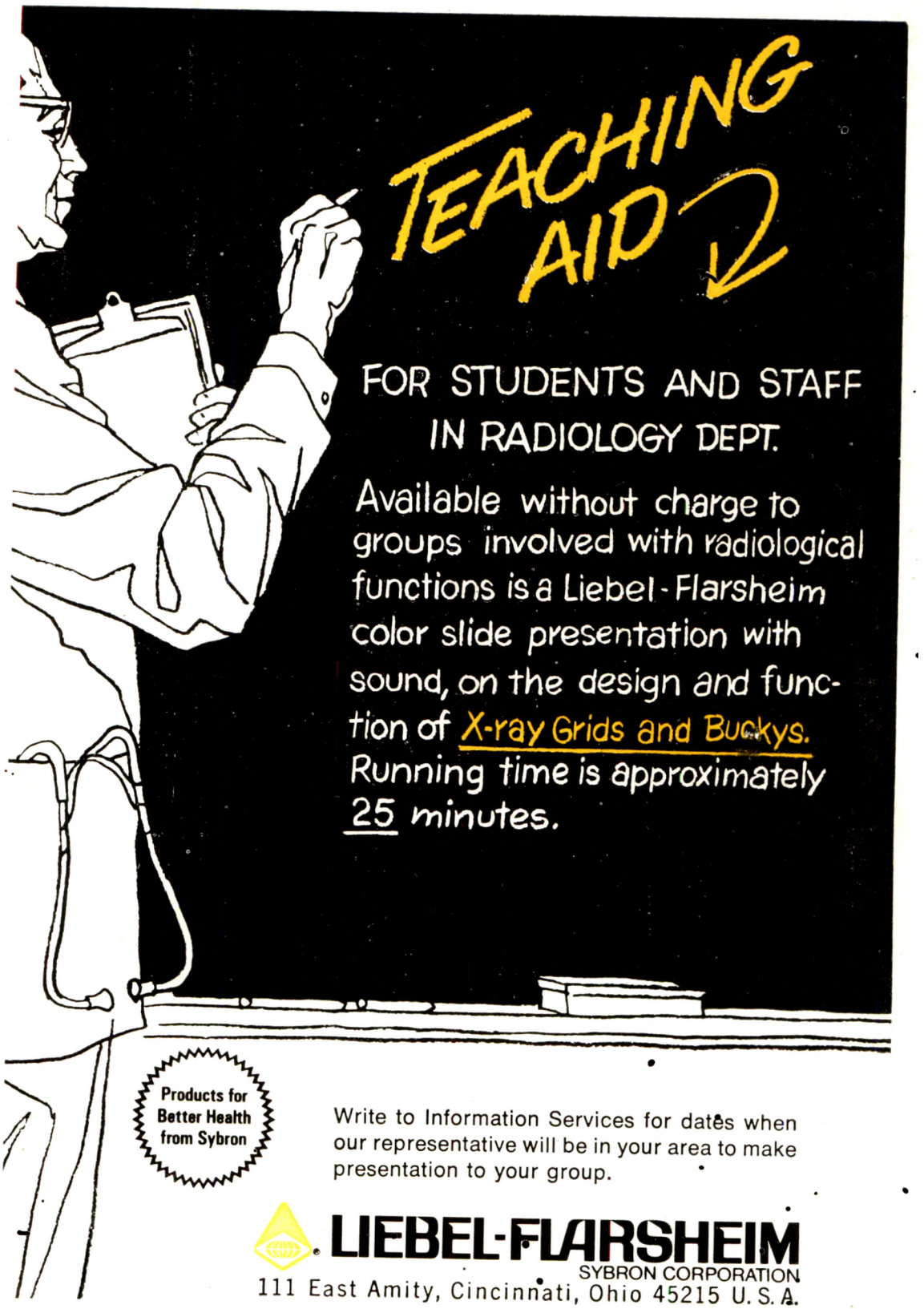
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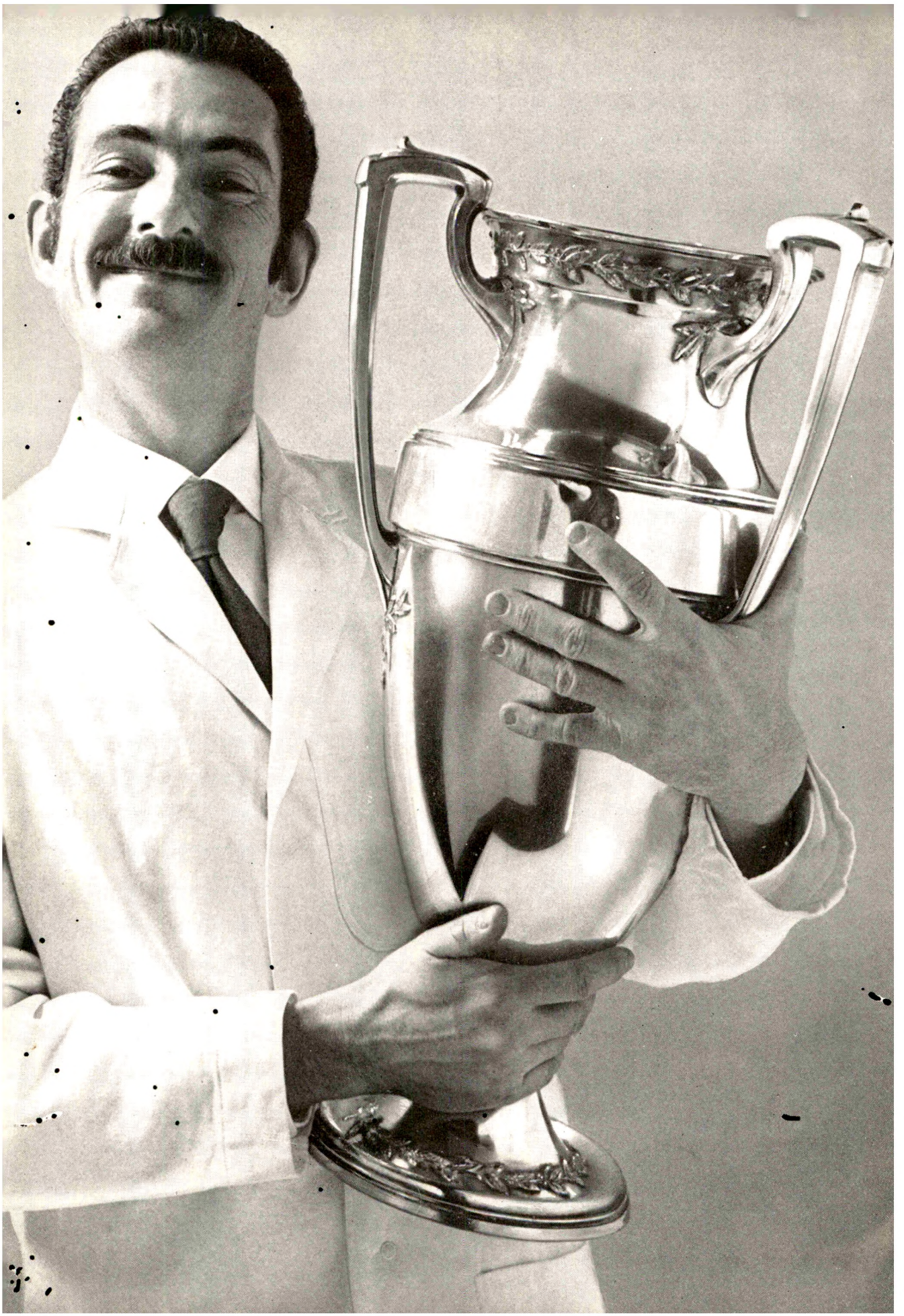
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of the left ventricle (Fig. 4, *A* and *B*). The postoperative angiogram shows an excellent recuperation of contraction in these segments of the left ventricular wall (Fig. 4, *C* and *D*).

DISCUSSION

Patency of aorto-coronary grafts was assessed early after surgery, and pre and postoperative ciné-ventriculograms were compared. Recuperation of a more or less vigorous and sometimes normal left ventricular contraction from previous hypokinetic or akinetic areas could be evidenced in nearly two-thirds of our patients with patent aorto-coronary grafts. This shows that left ventricular function can often be re-established when adequate perfusion of blood to the myocardium is supplied.

Direct observation of left ventricular contraction, on the operating table, has also permitted us to identify areas of asynergy noted on the left ventricular angiogram and to ascertain that these areas contain viable muscle and do not necessarily correspond to an infarcted or fibrosed area.

Our study shows that hypokinesis is, in most instances, reversible with return to an essentially normal pattern of left ventricular contraction. This seems to indicate that viable ischemic muscle is totally responsible for left ventricular dysfunction in these patients. Akinesis, on the other hand, is not completely reversible. Two different situations can be observed following successful by-pass surgery in these patients. In approximately half of them, a mild or moderate improvement of left ventricular contraction can be seen with persistence of some degree of hypokinesis. This partially reversible type can presumably be accounted for by the presence in the area of fibrosed muscle fibers intermingled or surrounded with viable ischemic muscle. In the other half, conversely, akinesis remains unchanged in spite of a potentially adequate perfusion of the area. With this irreversible type, it is logical to think that there has been predominant scar tissue replacement of the area.

Reversibility of left ventricular asynergy also appears to be influenced by its localization on the left ventricular wall. Inferior or diaphragmatic asynergy is less reversible than asynergy located to the anterior wall or apex of the left ventricle. It has not been possible, from this relatively small series of cases, to identify the factors that could explain why direct revascularization of the anterior descending artery should be more successful than by-passes of the right or circumflex coronary artery.

It remains a difficult task to foresee whether an akinetic, or perhaps a dyskinetic ventricular segment will regain contraction following direct coronary artery surgery. Predominant symptoms of angina pectoris during stress favor myocardial ischemia, whereas overt chronic left heart failure with an enlarged heart should suggest the presence of myocardial fibrosis. A myocardial infarction pattern on the electrocardiogram is often in association with ventricular asynergy and does not always indicate myocardial scar formation. On the other hand, lack of vascular supply of the area, on the coronary angiogram, from collateral circulation in the presence of complete obstruction and the demonstration of a thin left ventricular wall, on the ventriculogram, strongly suggest myocardial fibrosis.

Recently, changes in the contraction pattern of the asynergic areas have been demonstrated on left ventricular angiograms obtained before and after catecholamine and propanolol administration.[3] We have also obtained modifications of asynergic ventricular segments following exercise and cardiac pacing.[7,8] Such dynamic studies of the left ventricular angiogram will permit us, we hope, to determine variable, thus reversible, alterations of left ventricular contraction.

SUMMARY

Left ventricular asynergy, a frequent cause of myocardial dysfunction in subjects with ischemic heart disease, was studied on ciné-ventriculograms obtained before and after direct coronary artery surgery in 41 patients.



Following successful aorto-coronary bypass, hypokinesis of the left ventricle is completely reversible in most instances. Akinesis, on the other hand, is not totally reversible.

Improvement of the contraction pattern of the ventricle, from akinesis to hypokinesis, can be noted in approximately half of the patients, whereas the others remain unchanged. Similarly, alterations of left ventricular contraction located to the inferior wall of the ventricle are not usually reversible after surgery, whereas asynergy of the anterior wall of the ventricle can be abolished or improved in most instances.

The importance, difficulties and possible means of assessing the reversibility of left ventricular asynergy prior to direct coronary artery surgery are stressed.

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TRANSPOSITION OF THE GREAT ARTERIES*

DETERMINATION OF THE POSITION OF THE GREAT ARTERIES IN CONVENTIONAL CHEST ROENTGENOGRAMS

By RONALD GUERIN, M.D.,† BENIGNO SOTO, M.D., ROBERT B. KARP, M.D., JOHN W. KIRKLIN, M.D., *and* ALBERTO BARCIA, M.D.
BIRMINGHAM, ALABAMA

THE heart of patients with transposition of the great arteries is classically described as having a narrow vascular pedicle placed in front of the vertebral column as seen in conventional posteroanterior chest roentgenograms. The mediastinum is narrow because the aorta and pulmonary artery are superimposed in the anteroposterior plane.[3,4]

It was our experience in a series of 114 cases previously studied at the Mayo Clinic that the great arteries are not always located in the same anteroposterior plane.[1] With the purpose of analyzing the different possible positions of the great arteries in all types of transpositions, we correlated the conventional roentgenograms and angiocardiographic findings from 70 patients studied at the University of Alabama Medical Center.

METHOD OF STUDY AND DEFINITIONS

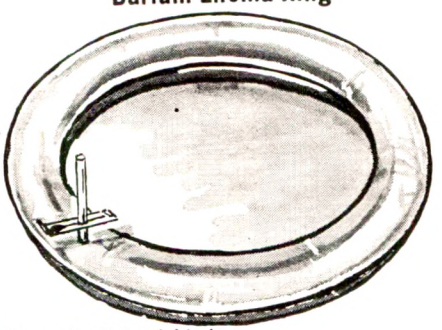
The conventional roentgenograms were taken in posteroanterior and lateral projections using a high kilovoltage technique of 150 kv. and 3 phase x-ray generators. The angiocardiographic studies were done with biplane large roll film technique by injecting the contrast media selectively into both ventricular chambers. The roentgenograms were taken at a rate of 12 per second in each plane during the first 2 seconds of the series. Patients studied only by cineangiography were excluded due to lack of detail and the impossibility of a quantitative analysis. The size of the pulmonary artery and aorta was measured immediately above the sinuses of Valsalva in the anteroposterior and lateral projections. The positions of the pulmonary artery and the aorta on the posteroanterior chest roentgenogram were reproduced on transparent paper prior to comparison with the angiographic findings.

Our series includes cases of classic complete transposition, corrected transposition, and double outlet right ventricle of the type in which the aorta is transposed in relation to the pulmonary artery. The generalizations concerning localization of the positions of the great arteries are applicable to both transpositions and double outlet right ventricle, and on conventional roentgenograms the two cannot be distinguished. A classic complete transposition is present when the aorta arises from the morphologic right ventricle and the pulmonary artery from the morphologic left ventricle and the atria and ventricles are concordant, that is, both in situs solitus or inversus. Most frequently, complete transposition occurs with situs solitus of the atria, situs solitus of the ventricles (dextroventricular loop), right-sided subaortic conus and the aorta to the right of the pulmonary artery at the semilunar valve level. A corrected transposition is present when the location of the ventricles relative to the atria is discordant. Most frequently, corrected transposition occurs with situs solitus of the atria, inversion of the ventricles, left-sided subaortic conus, and aorta to the left of the pulmonary artery at the semilunar valve level. Van Praagh's[6,8] loop rule states that the position of the ventricles can be identified by the location of the aorta. An aortic valve located to the right of the pulmonary valve indicates a

* From the Departments of Diagnostic Radiology and Surgery, School of Medicine, University of Alabama in Birmingham, Birmingham, Alabama.
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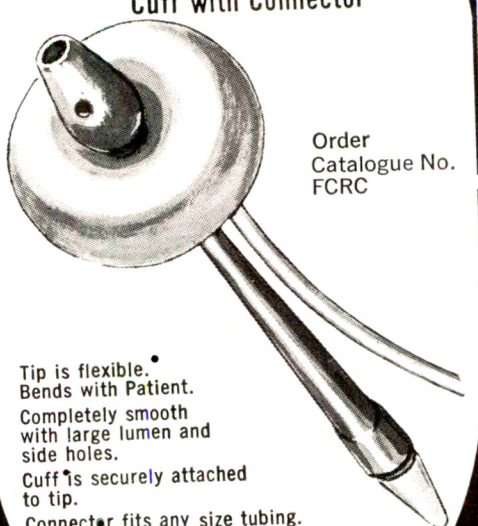
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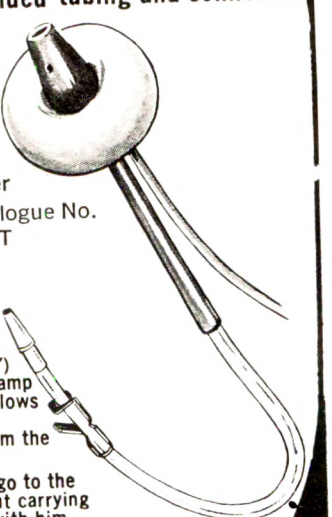
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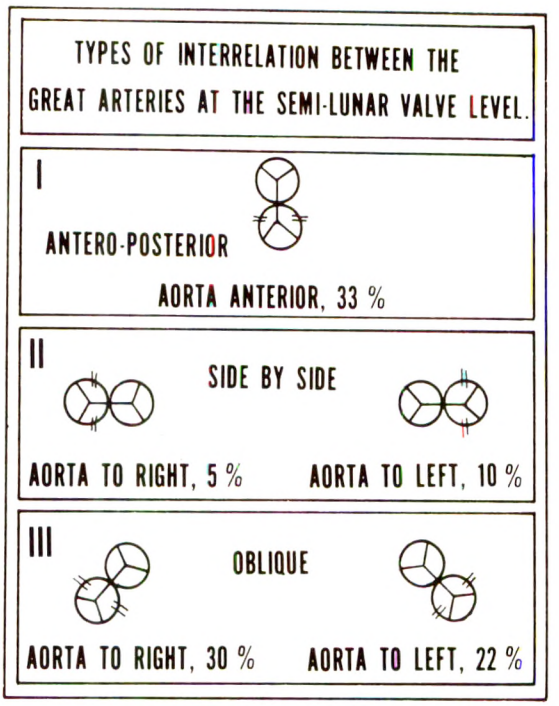


FIG. 1. Summary of the types of interrelation between the great arteries, and their relative incidence in this series of 70 patients.

right-sided morphologic right ventricle (d-ventricular loop). An aortic valve to the left indicates a left-sided morphologic right ventricle (l-ventricular loop).[8]

RESULTS

We found that the great arteries at the semilunar valve level may be in 3 main positions in patients with transposition and related malformations: (1) anteroposterior; (2) side-by-side; and (3) oblique. We had previously described these positions in double outlet right ventricle.[2] In the first group the aorta was always located anterior to the pulmonary artery. In the second group, the aorta was to the right or to the left of the pulmonary artery. When the great arteries were in an oblique position the aorta was almost always anterior to the right or anterior to the left of the pulmonary artery. In only 1 case was the aorta posterior to the pulmonary artery.

The percentage distribution of the 3 types of interrelation between the great arteries is shown in Figure 1. The frequency of these 3 types in complete transposition, corrected transposition and double outlet right ventricle is detailed in Figure 2. In classic complete transposition, the most common interrelationships were the oblique with the aorta to the right (Fig. 3, *C* and *D*; and 4, *A* and *B*), and the anteroposterior (Fig. 3, *A* and *B*). In 5 cases of complete transposition, the aorta was located anteriorly and to the left in relation to the pulmonary artery. These exceptions to Van Praagh's loop rule were due in each case to the marked right-to-left rotation of the heart produced by right ventricular hypertrophy and dextroconvex dorsal scoliosis. In corrected transposition the most common interrelationship was either the side-by-side (Fig. 5, *A–D*) or oblique (Fig. 6, *A* and *B*), in both instances with the aorta to the left. The anteroposterior relation was less frequent than in complete transposition (Fig. 5, *C* and *D*). In double outlet right ventricle there also frequently was a side-by-side (Fig. 3, *A* and *B*) or oblique relationship. As shown in Figure 2, the aorta was to the left in 6 (1+5) cases and to the right in 5 (3+2). Of the 3 patients with anteroposterior relation, 2 had pulmonary atresia.

Figure 7 shows the position of the great arteries at the semilunar valve level related to the visceroatrial situs, the ventricular loop, the size of the great arteries and the heart axis. The visceroatrial situs was solitus in 90 per cent of the patients and inversus in 10 per cent. No patients with indeterminate visceroatrial situs were studied in our series. The ventricular loop and the position of the ascending aorta was concordant in all cases of complete transposition except in 5 patients with d-loop, in which the aorta to the left of the

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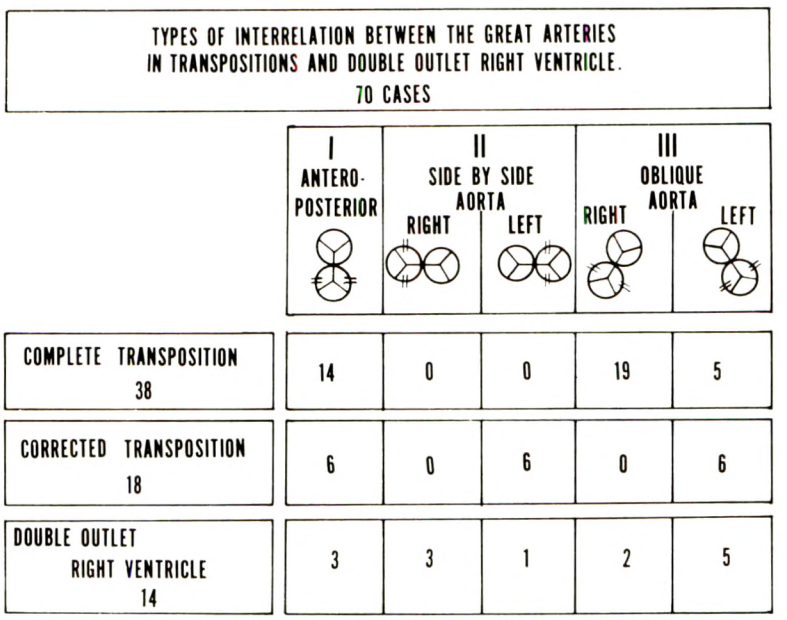


FIG. 2. Detail of the frequency of the 3 types of complete transposition, corrected transposition, and double outlet right ventricle.

pulmonary artery was an exception to the loop rule.

The position of the great arteries and their interrelationship could be predicted accurately in the majority of the cases from chest roentgenograms done with high kilovoltage technique. Difficulties arose when there was a marked discrepancy in the size of the vessels, particularly when the aorta was small and the pulmonary artery large.

DISCUSSION

The high kilovoltage chest roentgenographic technique allows better visualization of the heart and mediastinum by increasing the contrast between air and soft tissue and decreasing the contrast between soft tissue and bones.[5] With this technique it is possible to outline the ascending aorta (Fig. 3–6) in the majority of the cases even when the artery is not border-forming.

The ascending aorta may be directly localized on conventional chest roentgenograms made in this way when it is identified on the right (Fig. 3, *A–D*; and 4, *A* and *B*) or left (Fig. 5, *A–D*; and 6, *A* and *B*) mediastinal border. When the ascending aorta is not outlined, we try to identify the main pulmonary artery. The main pulmonary artery directly visualized on the left mediastinal border (Fig. 4, *A* and *B*) indicates a right-sided aorta. To locate the main pulmonary artery when it is not directly outlined in hearts with a wide vascular pedicle, one identifies the right and left pulmonary branches. If the pulmonary branches are at the same level and of the same length, the main pulmonary artery was medially placed indicating a left-sided aorta (Fig. 5, *A–D*). When the right pulmonary artery is higher than the left (Fig. 6, *A* and *B*), the ascending aorta was likewise located in the left upper mediastinum. All except 1 of the patients with these arrangements had corrected transposition.

When there is a narrow vascular pedicle and one cannot identify the ascending aorta or the pulmonary artery, the relative position of the left and right main branches may help to locate the main pulmonary artery. If the left pulmonary branch is


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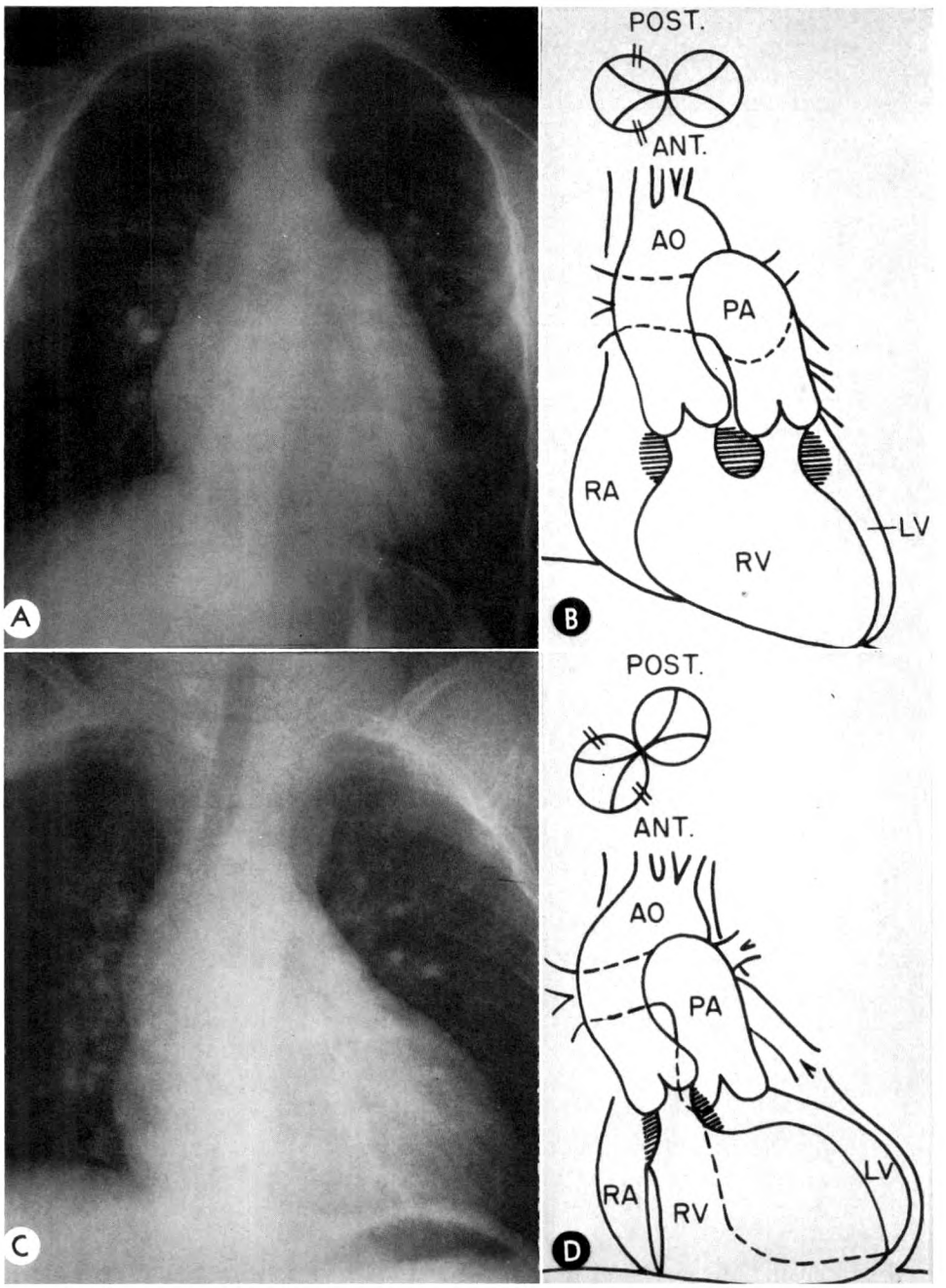


FIG. 3. (In this and subsequent Figures, (*A*) and (*C*) are frontal conventional chest roentgenograms, and *B* and *D* are schematic diagrams of corresponding angiocardiograms.) (*A* and *B*) Double outlet right ventricle with both subaortic and subpulmonary conus and ventricular septal defect. The gastric air bubble to the left indicates that the visceroatrial situs is solitus. The vascular pedicle is wide as the great arteries are side-by-side. The ascending aorta to the right of the prominent main pulmonary artery identifies the normal (solitus) position of the right ventricle, according to the loop rule. (*C* and *D*) Complete transposition of the great arteries with ventricular septal defect and oblique interrelation between the great arteries. The wide vascular pedicle is due to prominence of the ascending aorta on the right mediastinal border and to the main pulmonary artery on the left. There is situs solitus of viscera, atria and ventricles. AO = aorta; PA = pulmonary artery; RA = right atrium; RV = right ventricle; LV = left ventricle.

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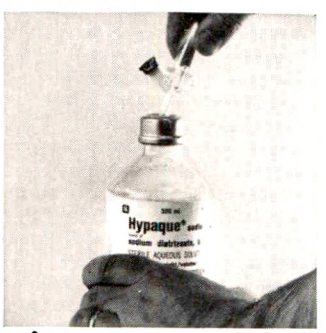
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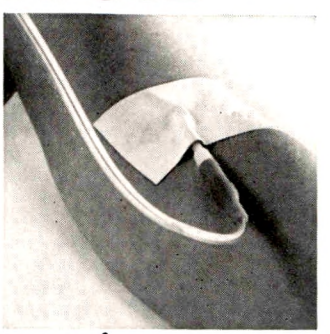
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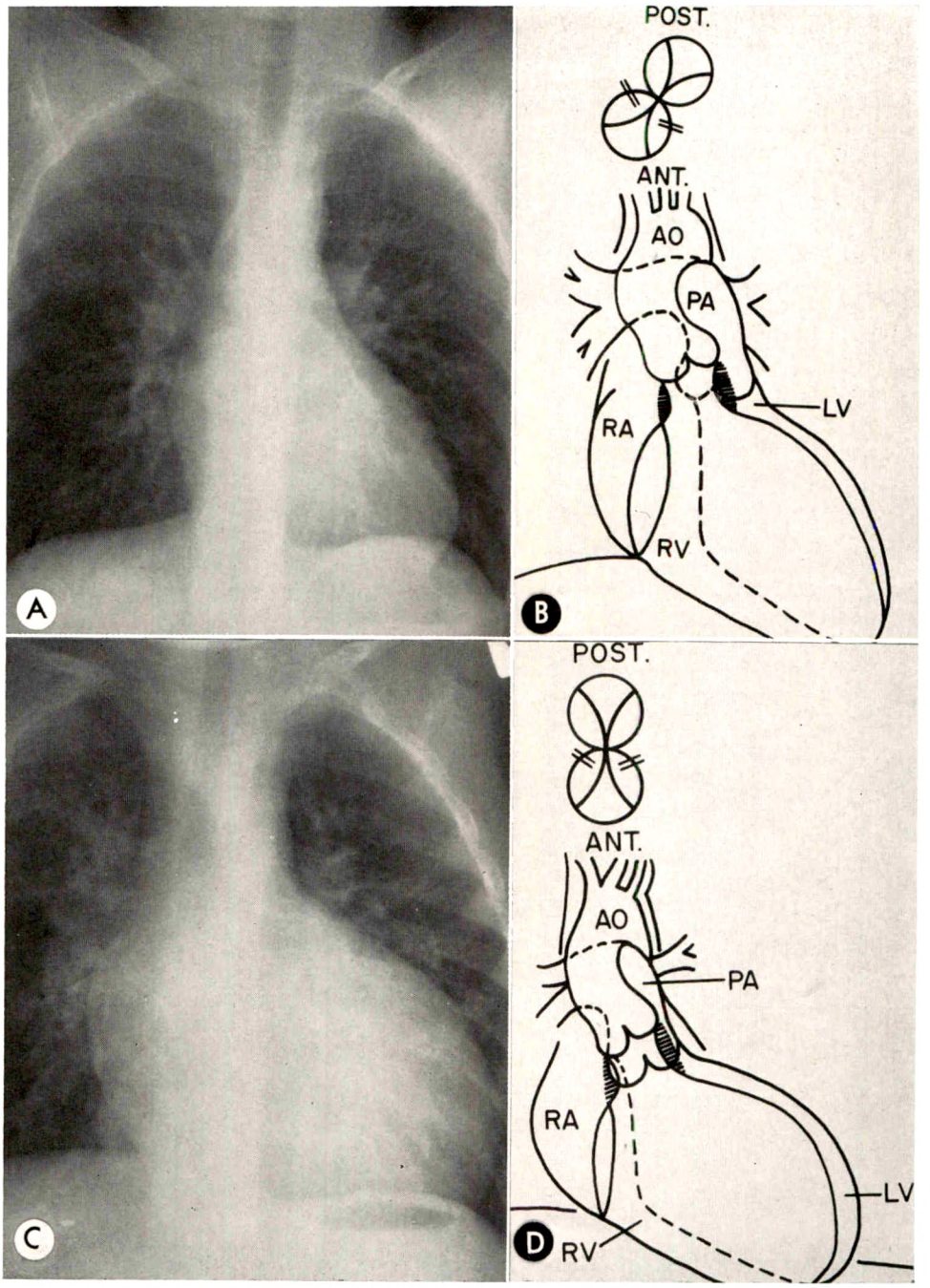


FIG. 4. (*A* and *B*) Complete transposition of the great arteries with situs solitus of the viscera, atria and ventricles. Compared to Figure 2, *C* and *D*, there is a lesser degree of obliquity of the great arteries. The aorta to the right and the pulmonary artery to the left are identified forming the borders of the mediastinum. (*C* and *D*) Complete transposition with anteroposterior relationship of the great arteries. The vascular pedicle is narrow. The ascending aorta and main pulmonary artery are not border-forming.

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PRECAUTIONS

Before a contrast medium is injected, the patient should be questioned for a history of allergy. Although a history of allergy may imply a greater than usual risk, it does not arbitrarily contraindicate the use of the medium. Premedication with antihistamines to avoid or minimize possible allergic reactions may be considered. **Benadryl® (brand of diphenhydramine hydrochloride) however, may cause precipitation when mixed with Hypaque sodium.** The intravenous injection of 0.5 ml. to 1 ml. of the contrast medium approximately 15 minutes prior to infusion of the full dose is frequently used in an effort to detect patient sensitivity. The absence of a reaction to this test dose is not entirely reliable for predicting the patient's response to the full diagnostic dose. Severe reactions, including fatalities, have occurred with a test dose as well as with larger doses. Adequate facilities for treating severe reactions should be available.

Caution is advised in patients with severe cardiovascular disease and in patients with a history of bronchial asthma or other allergic manifestations or of sensitivity to iodine.

Contrast media have been shown to promote the phenomenon of sickling in individuals who are homozygous for sickle cell disease when the material is injected intravenously or intraarterially.

In myelomatosis, urography should only be performed with caution. If a weak protein-binding agent such as a diatrizoate is used for the procedure it is essential to omit preparatory dehydration, administer fluids, and attempt to alkalinize the urine.

The results of PBI and RAI uptake studies, which depend on iodine estimations, will not reflect thyroid function for up to 16 days following administration of iodinated urographic media. However, function tests not depending on iodine estimations, e.g., T_3 resin uptake or direct thyroxine assays, are not affected.

Immediate adverse reactions to drip infusion pyelography have not been reported to occur at a higher frequency or greater severity than with routine excretory urography. However, sequelae of this procedure are possible some hours after the examination. Drip infusion pyelography imposes not only a sudden osmotic load, but also may present as much as 160 meq. of sodium (3.7 Gm.) to patients with established, decreased glomerular filtration and renal tubular damage. In addition, these patients may also have coexisting or associated cardiovascular disease. Therefore, the possibility of the development of congestive heart failure hours after the procedure should be considered.

Because of the possibility of inducing temporary suppression of urine, it is wise to allow an interval of at least 48 hours to pass before repeating drip infusion pyelography in patients with unilateral or bilateral reduction of normal renal function.

Preparatory dehydration or abdominal compression may not be necessary in infusion pyelography. In infants and young children and in azotemic patients, especially those with oliguria, dehydration is considered dangerous.

ADVERSE REACTIONS

Reactions accompanying the use of contrast media may vary directly with the concentration of the substance, the amount used, the technique used, and the underlying pathology.

Adverse reactions, usually of a minor nature, have occurred in 10-14 per cent of patients who have received Hypaque intravenously. Reactions due to **technique** include hematomas and ecchymoses following extravasation from the vein, and pyrogenic reactions. **Hemodynamic reactions** include vasodilatation with flushing, hypotension and, rarely, vein cramp or thrombophlebitis. Serious **cardiovascular reactions** include rare cases of cardiac arrhythmias (e.g., ventricular fibrillation), shock, and cardiac arrest. Transient proteinuria may occur occasionally following the injection of radiopaques and, rarely, oliguria and anuria (renal shutdown) have been reported as a secondary effect of a hypotensive reaction. **Allergic reactions** include asthmatic attacks, nasal and conjunctival symptoms, dermal reactions such as urticaria and, rarely, anaphylactic shock, sometimes with fatal outcome. Severe reactions may also be manifested by signs and symptoms relating to the **respiratory system** (dyspnea, cyanosis, pulmonary or laryngeal edema), or to the **nervous system** (restlessness, confusion, or convulsions). **Other reactions** include nausea, vomiting, excessive salivation, anxiety, headache, and dizziness. Infrequently, "iodism" (salivary gland swelling) from organic compounds appears two days after exposure and subsides by the sixth day.

DOSAGE AND ADMINISTRATION

The recommended dose is calculated on the basis of 2 ml. of Hypaque sodium 25 per cent per pound of body weight. The average dose of the solution for adults is 300 ml.; for optimum results, a minimum dose of 250 ml. should be used. A maximum dose of 400 ml. is generally sufficient for the largest of subjects.

The solution is administered intravenously through an 18-gauge needle over a period of three to ten minutes. Pyelographic films are taken at 10, 20, and 30 minutes from the beginning of the infusion. Early 2, 3, 4, and 5 minute films are obtained when indicated for the evaluation of hypertension. Nephrotomographic sections are best taken just at the end of the infusion, and voiding cystourethrograms when desired are usually made at 30 minutes.

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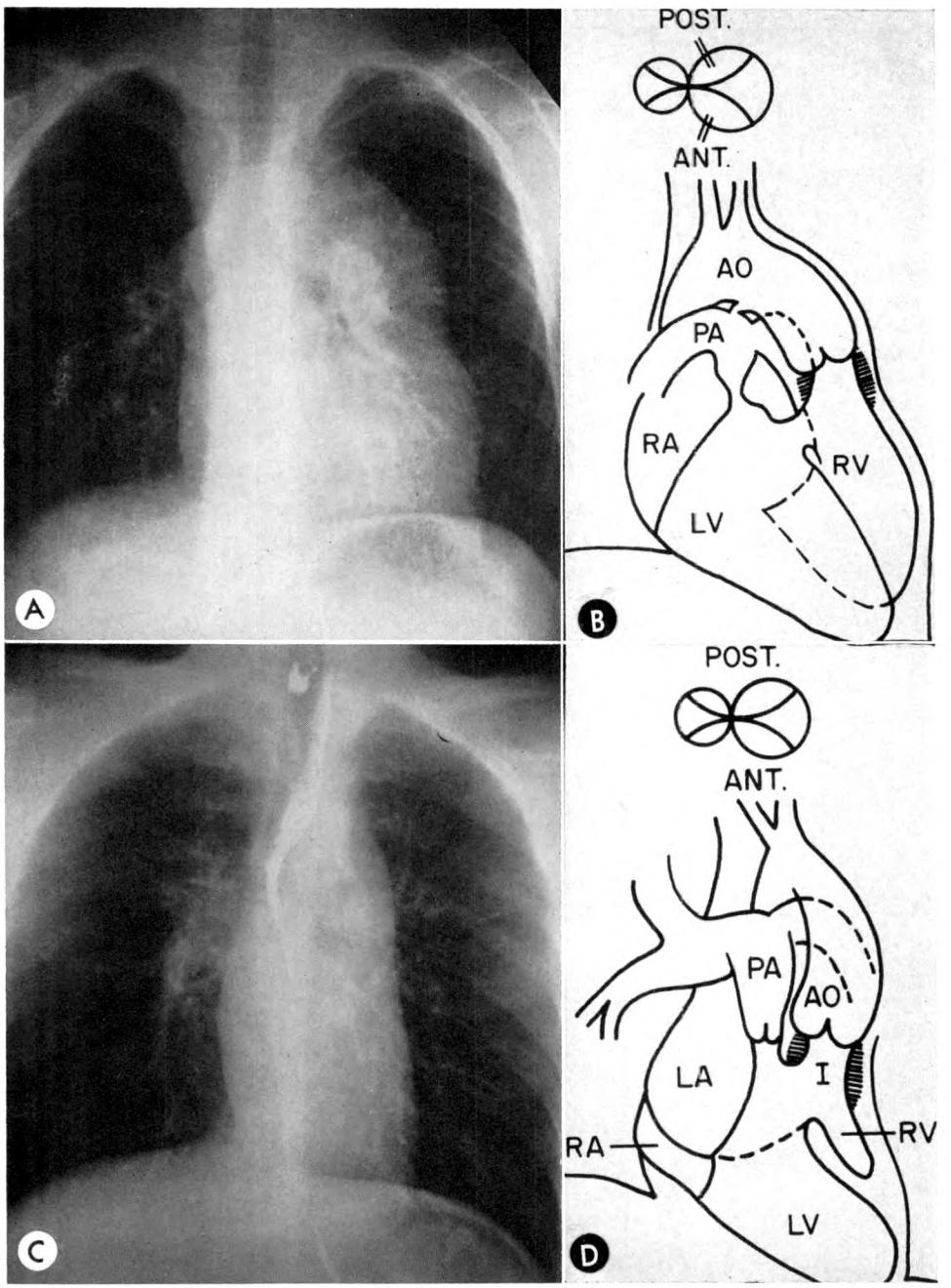


FIG. 5. (*A* and *B*) Corrected transposition with ventricular septal defect and side-by-side interrelation between the great arteries. The dilated aorta is prominent on the left mediastinal border. The main pulmonary artery is medially placed with right and left branches at the same level. The left-sided location of the aorta indicates a left-sided right ventricle. The viscera and atria are in situs solitus. The atria and ventricles are in discordant position. (*C* and *D*) Common ventricle with underdeveloped right ventricular sinus and large right-sided morphologic left ventricle. The great arteries are transposed and side-by-side with the ascending aorta to the left of the pulmonary artery, forming a wide vascular pedicle. LA = left atrium; I = infundibulum.



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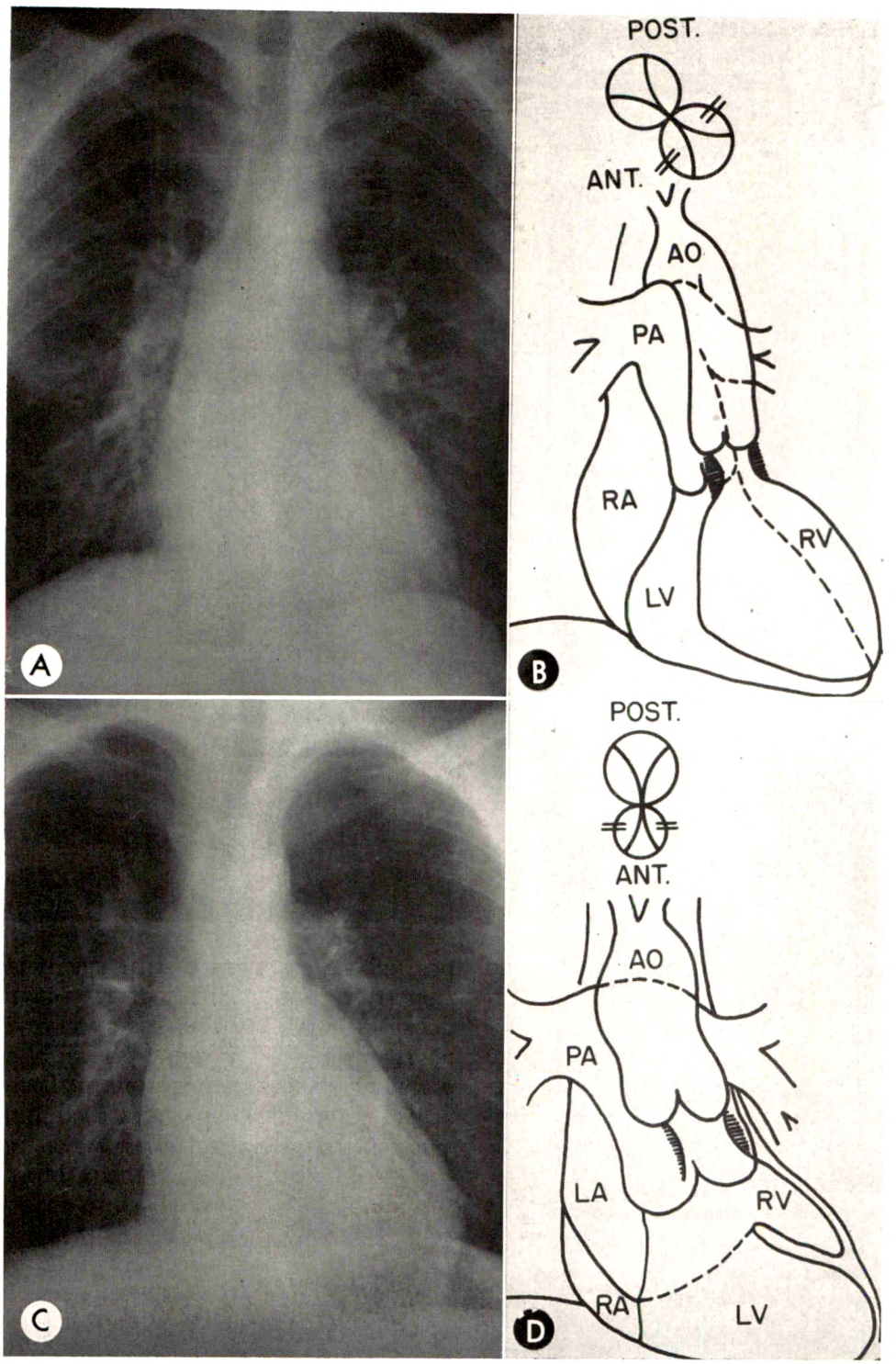


FIG. 6. (*A* and *B*) Corrected transposition with oblique interrelation between the great arteries. The ascending aorta is located on the left mediastinal border. The main pulmonary artery is medially placed and the right pulmonary branch is higher than the left. (*C* and *D*) Corrected transposition with anteroposterior interrelation between the great arteries. The vascular pedicle is narrow with both great arteries medially placed. The right and left pulmonary arteries are at the same level. There is situs solitus of the viscera and atria with discordant position of the ventricles.

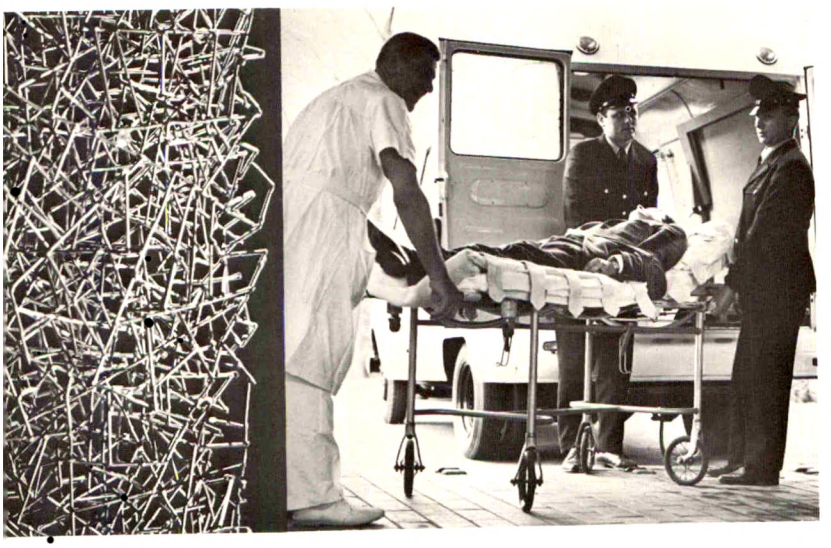


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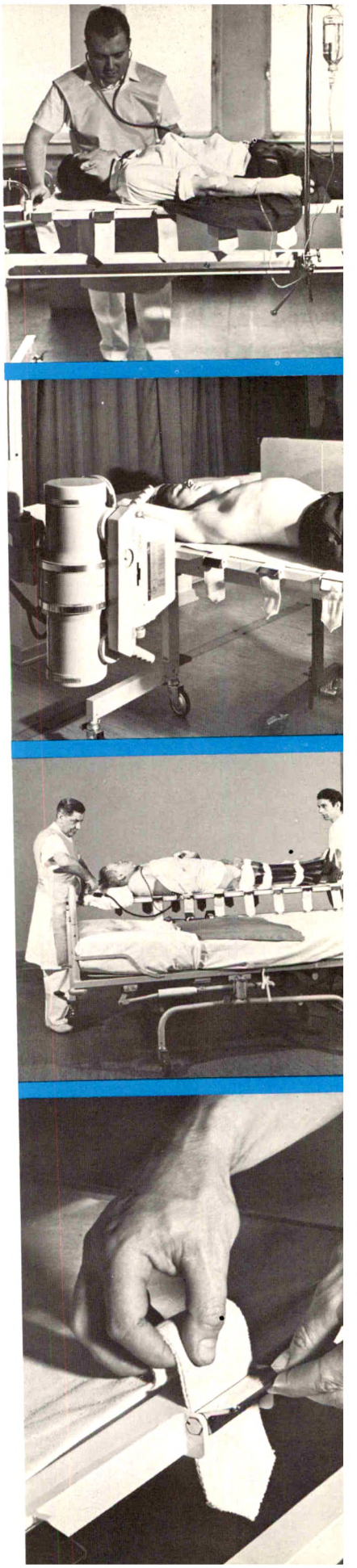
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POSITION OF GREAT ARTERIES IN RELATION TO VISCERO-ATRIAL SITUS, VENTRICULAR LOOP, GREAT ARTERY SIZE, AND HEART AXIS. 70 CASES

			I ANTERO-POSTERIOR	II SIDE BY SIDE AORTA RIGHT	II SIDE BY SIDE AORTA LEFT	III OBLIQUE AORTA RIGHT	III OBLIQUE AORTA LEFT
			23	3	7	21	16
VISCERO ATRIAL SITUS	SOLITUS	63	21	3	6	20	13
	INVERSUS	7	2	0	1	1	3
VENTRICULAR LOOP	D. LOOP	46	16	3	0	21	6
	L. LOOP	24	7	0	7	0	10
GREAT ARTERY SIZE	AO > PA	30	12	0	6	5	7
	AO = PA	7	2	1	1	1	2
	AO < PA	33	9	2	0	15	7
HEART AXIS	LEVOCARDIA	55	18	3	5	21	8
	DEXTROCARDIA	15	5	0	2	0	8

FIG. 7. Position of the great arteries at the semilunar valve level related to viscero-atrial situs, the ventricular loop, the size of the great arteries and the heart axis.

higher than the right, the main pulmonary artery is usually left-sided and the aorta right-sided. All such cases which we reviewed were complete transpositions. If both pulmonary branches are at the same level and there is an anteroposterior relationship of the great arteries, complete transposition occurred more frequently than corrected transposition.

The identification of the position of the great arteries in patients with complex forms of congenital heart disease is a step in the segmental approach described by Van Praagh.[7,9,10] The 4 major cardiac segments to be analyzed are: (1) the visceroatrial situs; (2) the ventricular segment; (3) the conus or infundibulum; and (4) the great arteries. From a practical standpoint, it is helpful to obtain as much of this information as is possible from simple studies. It is important, then, that on conventional chest roentgenograms it is possible to determine the position of the viscera, the atria, and the great arteries in the majority of the cases. There was always correspondence in the position of the viscera and atria in our cases.

From the loop rule, knowing the position of the aorta, we can suspect the ventricular interrelation. On the angiocardiograms we confirm the position of the great arteries, the location of the morphologic right and left ventricle, and the position and degree of development of the conus or infundibulum.

There were no exceptions to Van Praagh's loop rule in our cases of side-by-side and anteroposterior interrelation between the great arteries. Five exceptions to the loop rule occurred in patients with complete transpositions with dextroventricular loop and oblique interrelated great



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arteries with the aorta anterior and to the left. The other exception was a case of double outlet right ventricle, situs solitus of atria and ventricles, and left-sided aorta.

Thus it is of prime importance to locate the ascending aorta on the conventional chest roentgenograms to establish the interrelation between the great arteries and the position of the ventricles. The following are the factors affecting the identification of the ascending aorta in the conventional chest roentgenograms: (1) the position of the aorta in relation to the main pulmonary artery; (2) the relative size of the great arteries; (3) the position of the heart axis; and (4) the rotation of the heart and degree of right ventricular hypertrophy.

When an anteroposterior interrelationship of the great arteries occurs, the relative size of the arteries is the most important factor in the visualization of the aorta. In every case of pulmonary atresia and in most cases with severe pulmonary stenosis, the aorta was directly identified on the chest roentgenograms. When the great arteries are side-by-side, the position of the vessels is more important than their relative size in the identification of the aorta. In oblique interrelationship the aorta is more easily seen as a border-forming structure when it is on the left. This is a consequence of the degree of right ventricular hypertrophy and the clockwise rotation of the heart. Thus the aorta is prominent even when it is not enlarged and smaller than the pulmonary artery.

The position of the heart axis (levocardia or dextrocardia) was not an important factor in the identification of the aorta.[1] In dextrocardias with levoventricular loop, the aorta was more often directly identified to the left in 10 of 12 cases studied, because the side-by-side and extreme oblique positions were more frequent.

SUMMARY

To determine the relative position of the aorta and pulmonary artery in conventional chest roentgenograms, a correlative study with large roll film angiocardiograms was made in 70 cases of transposition of the great arteries, including 14 cases of double outlet right ventricle of the transposition type.

We found 3 main types of interrelation between the great arteries: anteroposterior, oblique and side-by-side.

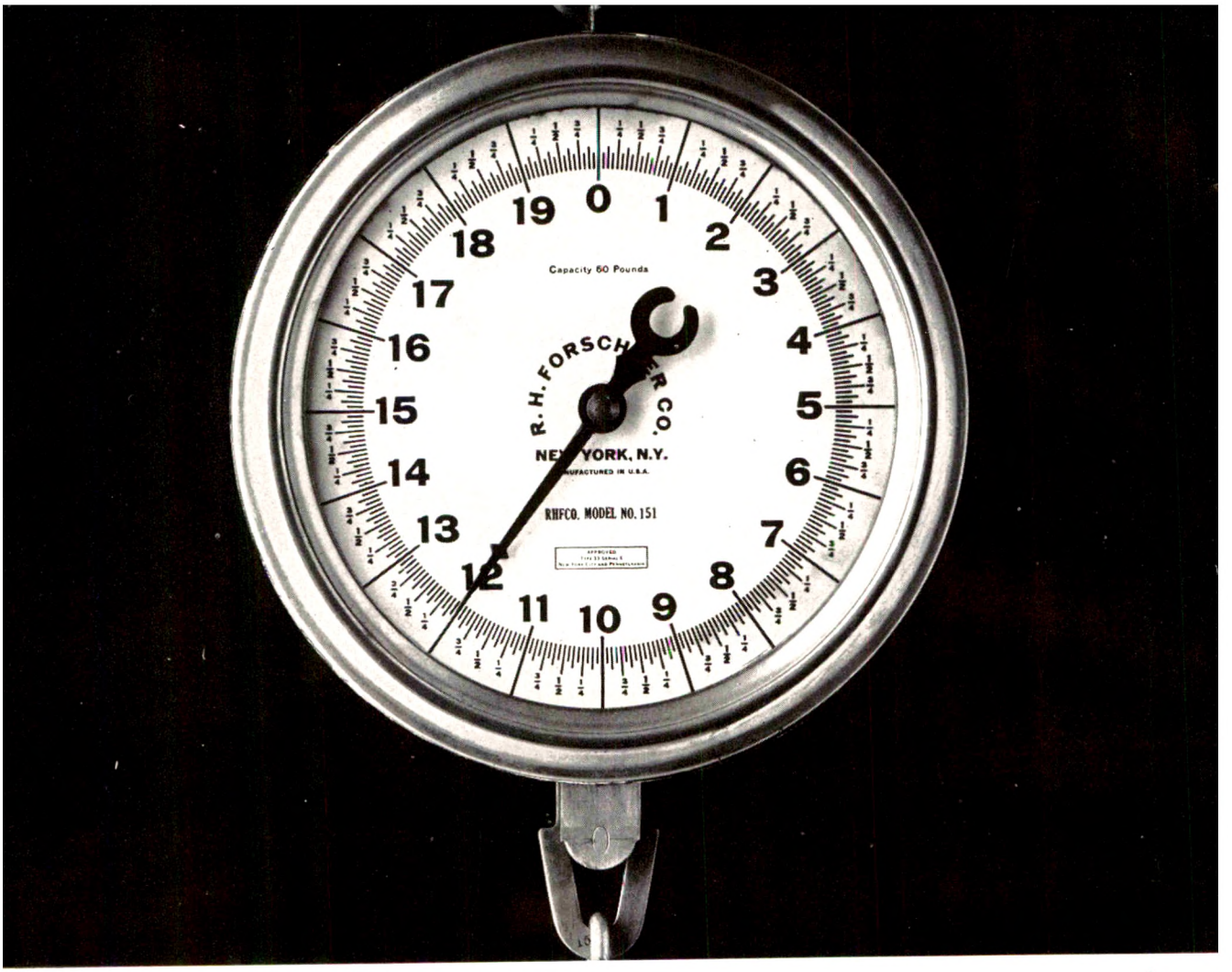
From the interpretation of the conventional high kilovoltage roentgenogram it was possible to determine the position of the great arteries in the majority of the cases with levoventricular loop and left-sided aorta. In patients with dextroventricular loop and anteroposterior relationship between the great arteries, it was more difficult to identify the position of the arteries.

Six exceptions to the loop rule occurred in 70 cases.

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University of Alabama Medical Center
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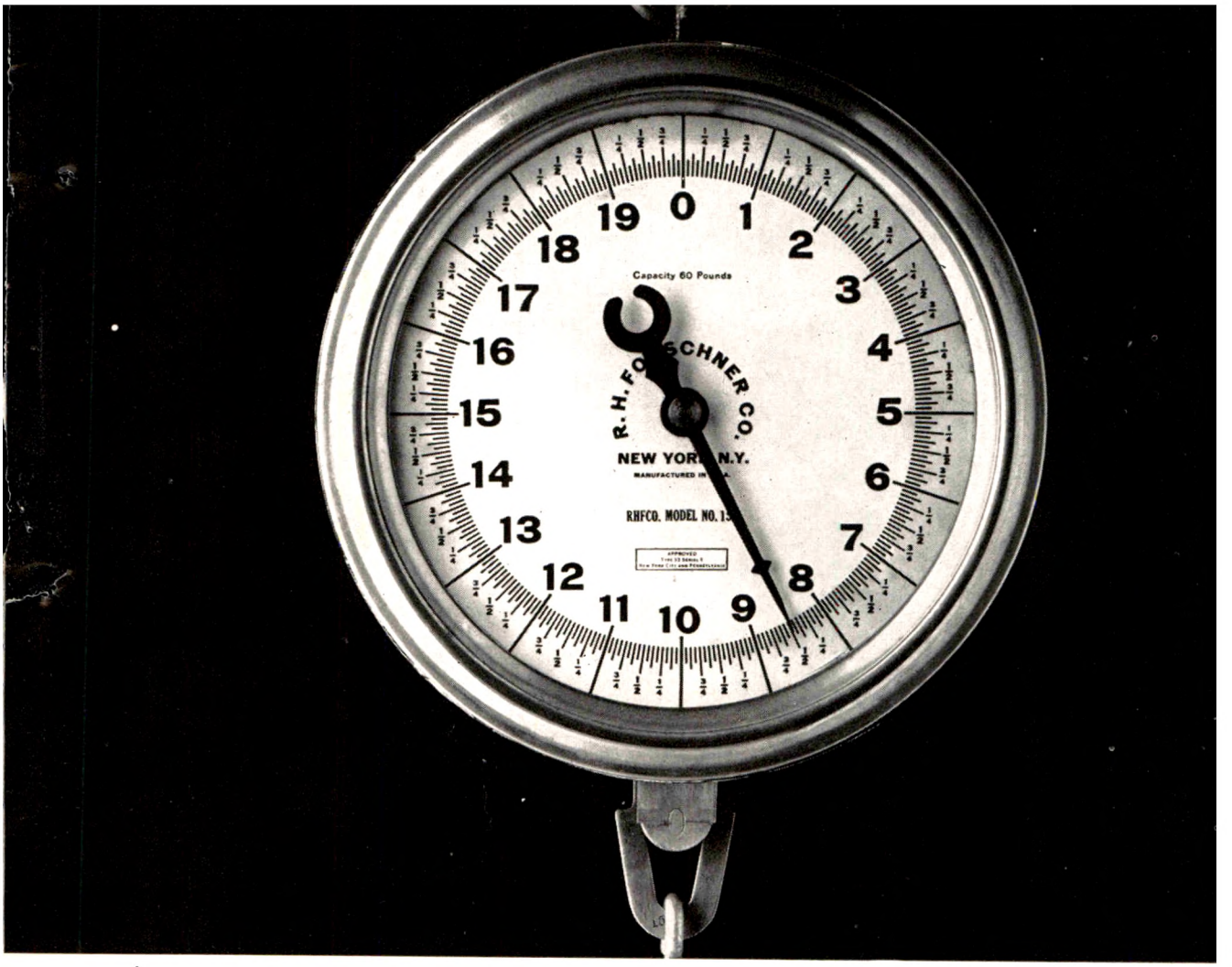
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ROENTGENOGRAPHIC MEASUREMENT OF ARTERIAL NARROWING*

By F. A. BERNE, M.D., W. P. LAWRENCE, M.D., *and* W. H. CARLTON, Ph.D.
AUGUSTA, GEORGIA

ANGIOGRAPHIC demonstration of the degree of arterial stenosis has routinely been calculated as a percentage of the normal lumen diameter.[1,2] Our purpose is to demonstrate the conceptual errors associated with occlusions and to make suggestions for minimizing these errors.

MATERIAL AND METHOD

Phantoms simulating occluded arteries were constructed from glass tubing with paraffin plaques secured to the wall by gentle heating. Phantom A was designed to simulate a recanalized thrombus (Fig. 1, *A* and *B*) and Phantom B was formed to occlude more than 50 per cent of the lumen of the tube (Fig. 2, *A* and *B*). The glass tubes were filled with conray and placed in a water phantom. Roentgenograms (70 kv., 80 mas.) were taken at 45° intervals in a plane perpendicular to the longitudinal axis of the tubes. The actual occluded areas were measured graphically from photographs taken along the longitudinal axis of the phantom.

RESULTS

Per cent occlusion with respect to diameter was calculated by conventional means for each individual view, then by averaging 2 measurements 90° apart, 3 measurements 45° apart, and 4 measurements 45° apart. Only the minimum diameter of the main column of the contrast medium was measured, even for roentgenograms with multiple interfaces. Table 1 contains the per cent occlusion calculated by a ratio of lumen diameters for Phantoms A and B. It also contains per cent area occlusion for Phantom A (recanalized)

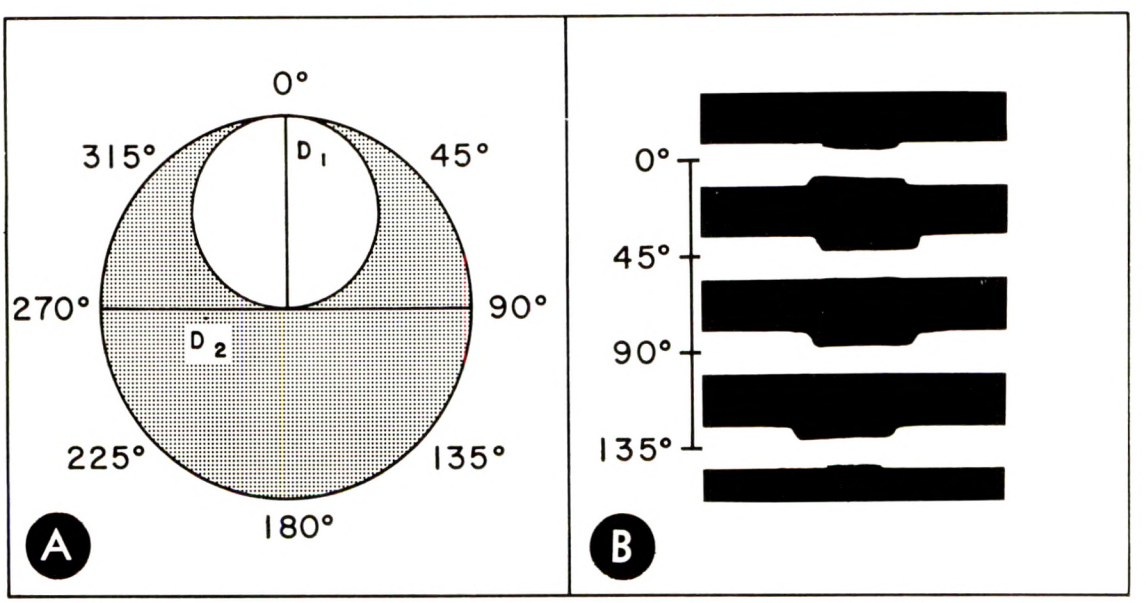
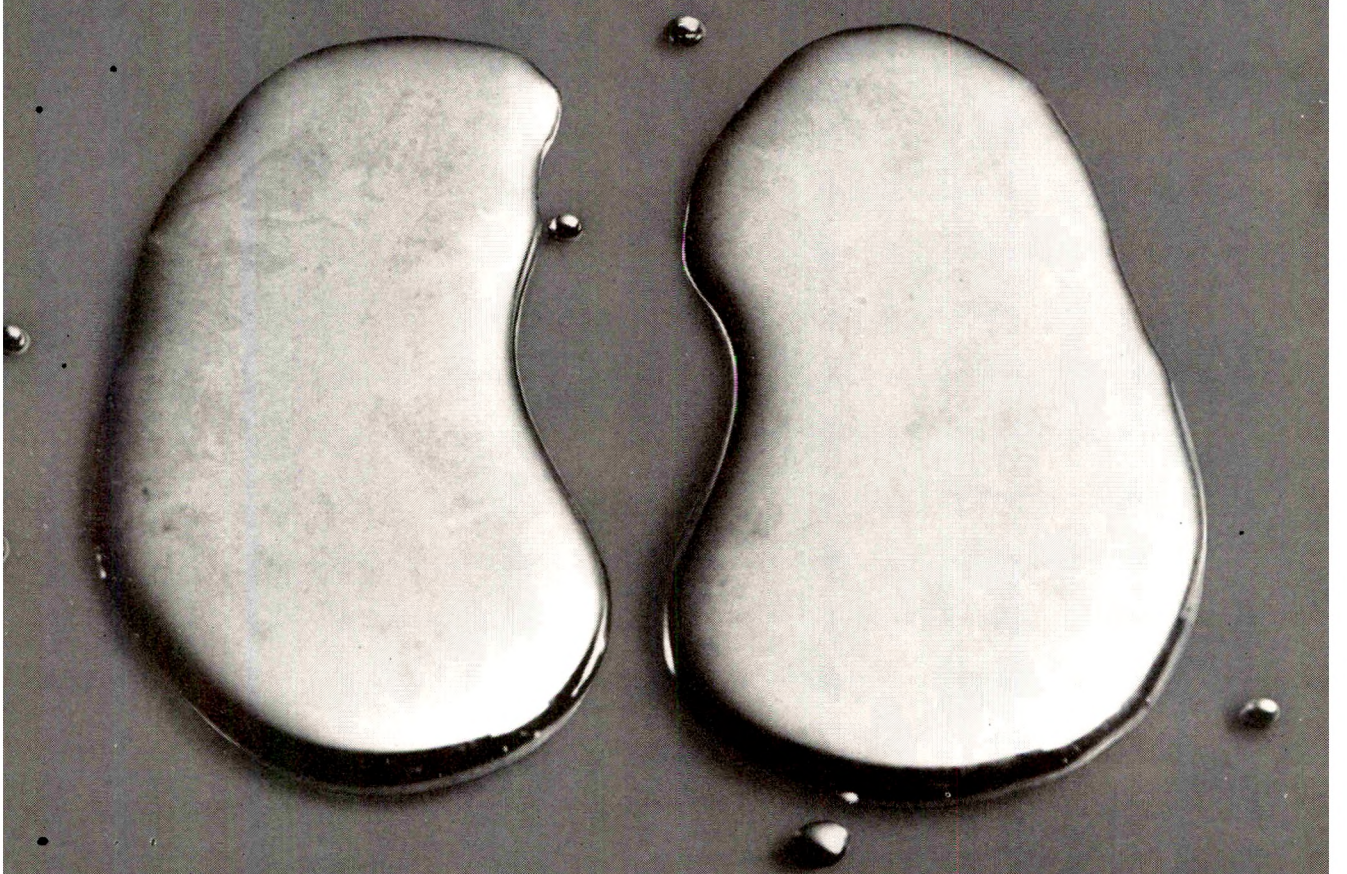


Fig. 1. (*A*) Diagram simulating annular narrowing of the lumen of an artery. (*B*) Roentgenograms taken at 45° rotations in a plane perpendicular to the longitudinal axis of Phantom A.

* From the Department of Radiology, Medical College of Georgia, Augusta, Georgia.

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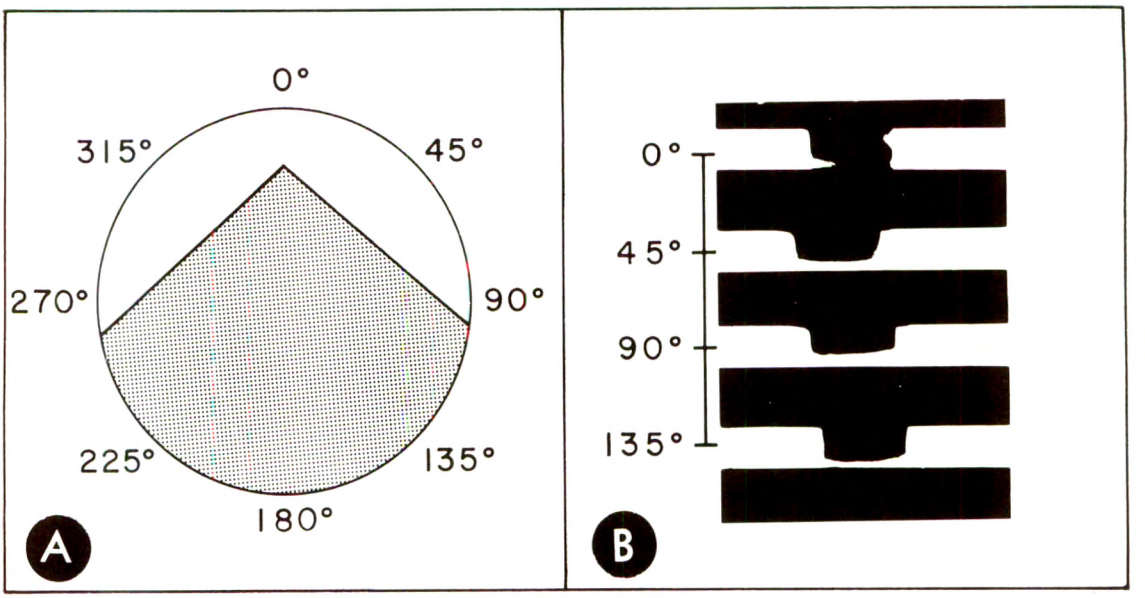


Fig. 2. (*A*) Diagram of a stylized plaque in an artery. Note that the apparent occlusion depends on the orientation. (*B*) Roentgenograms taken at 45° rotations in a plane perpendicular to the longitudinal axis of Phantom B.

calculated by a ratio of the cross sectional areas ($A = D^2/4$).

The actual cross sectional occluded area for Phantom A (Fig. 1, *A* and *B*) is 61 per cent. Calculation of a conventional ratio of diameters gives the impression that the occlusion is approximately 35 per cent while the ratio of the cross sectional areas calculated by finding the average diameters is much closer to the true value.

It may be seen that a single view of Phantom B (Fig. 2*B*) gives the impression of a 35 per cent variation in occlusion. Averaging multiple views results in a variation of 16 per cent for 2 views at 90°, 10 per cent for 3 views at 45° intervals, and 3 per cent for 4 views at 45° intervals.

Discussion

The data presented above serve to demonstrate some of the fallacies inherent in the usual practice of reporting arteriographic occlusion in terms of a ratio of luminal diameters. No method presently exists for demonstrating the actual area of occlusion with complete accuracy. Figure 1*A* shows diagrammatically an "artery"

Table I

Measurement of Blood Vessel Occlusion

Position (degrees)	Phantom A Per Cent Occlusion (ratio of diameters)	Phantom A Per Cent Occlusion (area)	Phantom B Per Cent Occlusion (ratio of diameters)
0	34	57	51
45	34	57	79
90	36	58	86
135	32	54	84
0, 90	35	57	68
45, 135	33	55	81
90, 180	35	57	71
135, 225	32	54	84
0, 45, 90	35	57	72
45, 90, 135	34	58	82
90, 135, 180	34	58	76
135, 180, 225	35	57	75
0, 45, 90, 135	35	57	75
45, 90, 135, 180	33	55	77
90, 135, 180, 225	34	58	78
135, 180, 225, 270	34	56	77
Actual value	61	61	75

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See next page for brief summary.

with a "recanalized" diameter equal to ½ the artery diameter. An arteriogram in this case would imply an occlusion of only 50 per cent if compared as a ratio of diameters. It is important to realize that this would be obtained for any orientation of the artery. The area actually occluded in this example is 75 per cent. It may be seen that a simple ratio of diameters in this case will always underestimate the actual cross sectional occlusion. For example, when the ratio of diameters implies 80 per cent occlusion the actual value is 96 per cent and when the ratio is 20 per cent the real occlusion is 36 per cent. It is our opinion that in the case of annular narrowing (*e.g.*, recanalized thrombus), arteriograms should thus be interpreted in terms of a ratio of cross sectional areas rather than a ratio of diameters.

A different situation occurs when the occlusion is due to a single plaque. The degree of occlusion may be either underestimated or overestimated depending on the orientation of the artery and the shape of the plaque. This is demonstrated in Figure 2*A*. The case of a plaque occluding the lumen does not lend itself to a simple roentgenographic determination of area. The per cent occlusion may be established by taking multiple views and averaging the values obtained by the ratio of diameters method. It is our opinion that in order to minimize error, at least 3 projections, including the anteroposterior, lateral, and one oblique should be obtained.

SUMMARY

Arteriograms demonstrating annular narrowing should be interpreted in terms of area rather than a ratio of diameters. In the case of a plaque an anteroposterior, lateral, and one oblique projection should be obtained in order to make a reasonably accurate estimate of the per cent occlusion using the ratio of diameters method.

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Formula feedings should be substituted for breast feedings if these agents must be administered to the mother during lactation.

^{99m}Tc-DTPA, ^{99m}Tc-S colloid, and sodium pertechnetate ^{99m}Tc should not be administered to persons less than 18 years of age unless the expected benefit outweighs the hazards. It should be noted that although radiopharmaceuticals are not generally used in individuals under 18, procedures using ^{99m}Tc-DTPA or ^{99m}Tc-S colloid are occasionally necessary in such patients. The low internal radiation dosage of ^{99m}Tc-DTPA makes it a very satisfactory agent when scans of the kidney, brain, or blood vessels are necessary in young patients. The low internal radiation dosage of ^{99m}Tc-S colloid makes it a very satisfactory agent when liver or spleen scans are necessary in young patients.

Radiopharmaceuticals, produced by nuclear reactor or cyclotron, should be used only by physicians who are qualified by specific training in the safe use and safe handling of radioisotopes and whose experience and training have been approved by the appropriate federal or state agency authorized to license the use of radioisotopes.

When obtaining elutions from Technetope II (Technetium 99m) Sterile Generator, proper radiation safety precautions should be maintained at all times. The column containing ^{99}Mo need not be removed from the lead shield at any time. There is a high radiation field surrounding an unshielded column. Solutions of sodium pertechnetate ^{99m}Tc withdrawn from the generator should always be adequately shielded. The early elutions from the generator are highly radioactive. **Important:** Since material obtained from the generator may be intended for intravenous administration, aseptic technique must be strictly observed in all handling. The stoppers of the eluent bottle, of the elution tube, and of the collecting vial, as well as both rubber closures in the generator column, should be swabbed with a suitable germicide before each entry. All entries into the generator column must be made aseptically with sterile needles. Only the eluent provided should be used to elute the generator. Use a fresh milking tube and collecting vial for each elution; sufficient equipment is provided for this purpose. All equipment used to collect or administer sodium pertechnetate ^{99m}Tc must be sterile. Do not administer material eluted from the generator if there is any evidence of foreign matter. NOTE: The Renotec Kit and the Tesuloid Kit are not radioactive. However, after the eluted ^{99m}Tc is added, adequate shielding of the resulting preparation should be maintained.

Precautions: When using radioactive material, care should be taken to insure minimum radiation exposure to the patient (*i.e.*, by using the smallest dose of radioactivity consistent with safety and validity of data) as well as to all personnel directly or indirectly involved with the patient. Before a test is repeated in the same patient, the need should be carefully evaluated; this is especially true in younger patients.

Each elution from Technetope II (Technetium 99m) Sterile Generator should be assayed before use for ^{99m}Tc activity and for the possible presence of ^{99}Mo. Material containing more than 5 microcuries of ^{99}Mo per dose of ^{99m}Tc pertechnetate exceeds Atomic Energy Commission limits and should not be administered. Poor gastrointestinal absorption of an oral dose of pertechnetate and resultant low blood radioactivity levels have been observed in the postprandial state, in seriously ill patients, and in a small number of normal, fasting individuals. Since pertechnetate is concentrated by the gastric mucosa and the salivary glands, secretions of the digestive tract are radioactive and may cause artifacts on the cranial scan. Therefore, all possible care should be taken to avoid extracranial contamination, not only for the protection of patients and of hospital personnel but also to avoid obtaining a falsely positive scan due to extracranial radiation. Any condition which alters the blood-brain barrier or the normal cranial vasculature may cause abnormal areas of increased radioactivity. The brain scan with sodium pertechnetate ^{99m}Tc is therefore likely to be abnormal in patients with scalp contusions or acute head injuries. Following a craniotomy, uptake of radioactivity is increased throughout the operative field, usually for only a few weeks but in some instances for prolonged periods. Since cerebral radiographic techniques temporarily affect the blood-brain barrier, brain scanning with sodium pertechnetate ^{99m}Tc should precede cerebral angiography when possible, or should be postponed for several days thereafter. A negative brain scan does not rule out the possibility of a lesion and should therefore never be considered diagnostically conclusive. Because the normal vascular structures are more apparent on a ^{99m}Tc pertechnetate scan than on a radiochlormerodrin scan, and because the choroid plexus may be visible, it is particularly important to recognize the appearance of a normal brain scan when ^{99m}Tc pertechnetate is used, in order to avoid incorrect interpretation.

NOTE: The Renotec Kit and the Tesuloid Kit were designed for use with the sodium pertechnetate eluate obtained from a Technetope II Sterile Generator. It is recommended that only Technetope II be used as the source of sodium pertechnetate with the Renotec Kit and the Tesuloid Kit unless the user has demonstrated that other sources of ^{99m}Tc are consistently compatible and meet the standards of Technetope II.

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PITFALLS IN THE ULTRASONIC DIAGNOSIS OF PERICARDIAL EFFUSION*

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NEW YORK, NEW YORK

ALTHOUGH Edler and Hertz,[3] the Swedish pioneers of ultrasonic cardiography, first advanced the theory behind the ultrasonic detection of pericardial effusion in 1955, 10 years passed before Feigenbaum *et al.*[5,6] introduced the technique in the United States. Since then several studies have appeared extolling the validity of the procedure[2,8,9,14,16,22] or sharply criticizing its usefulness.[11,15]

We undertook the technique of echocardiography in a clinical setting to determine its accuracy and to explain the origin of any pitfalls in interpretation and technique. It was hoped that enumeration of our sources of error would be helpful in avoiding them in the future. We found that we encountered pitfalls that others had described previously and also found other sources of error which could frequently be troublesome.

METHOD

One hundred consecutive patients referred for the evaluation of possible pericardial effusion constitute the study group. The Physionic Portascan model MDU-661 BU-B equipped with a storage oscilloscope was used for all echocardiograms. The time-motion mode was employed in all patients, and Polaroid photographs of the electronically stored scans were made.

In all cases the examination was done at the bedside without patient discomfort or interruption of medical care. Clinical suspicion of pericardial effusion was based on unexplained cardiomegaly, decreased voltage on electrocardiography, pericardial friction rub, pulsus paradoxus, or congestive failure of unknown etiology. None of the patients had a definitive diagnosis of pericardial effusion before the ultrasound scan was performed.

The patients were all studied in the supine position using a one megacycle barium titanate crystal transducer coupled to the skin by a water soluble transmission gel. The transducer was usually placed in the fourth or fifth interspace to the left of the sternum and aimed directly posterior. The sensitivity control was always started at zero and gradually increased until a major echo was obtained from the posterior heart wall. This usually appeared at a depth of 9–14 cm. from the anterior chest wall. If the dominant echo is seen to be beating synchronously with the pulse, it

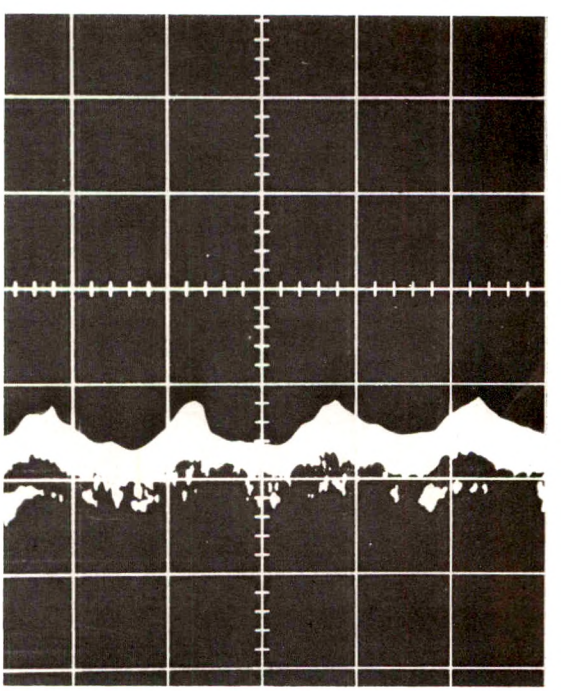
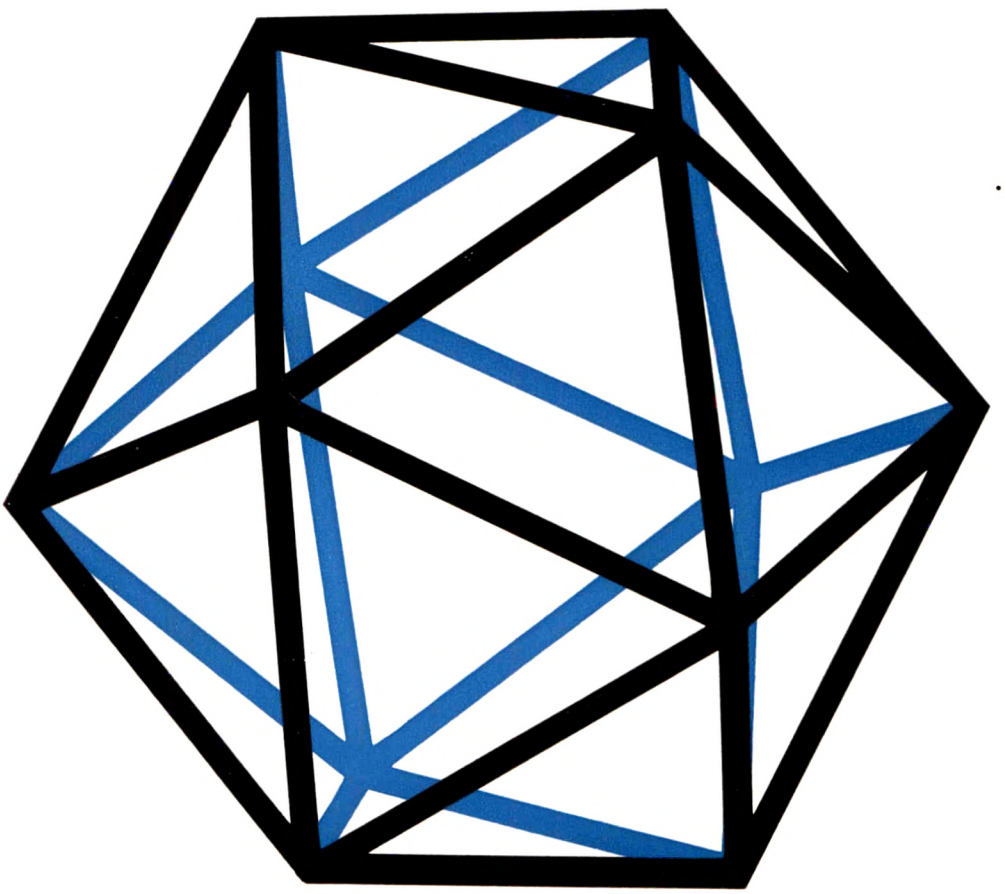


FIG. 1. A normal echocardiogram showing a pulsatile left ventricular wall. No separate pericardial echo is seen.

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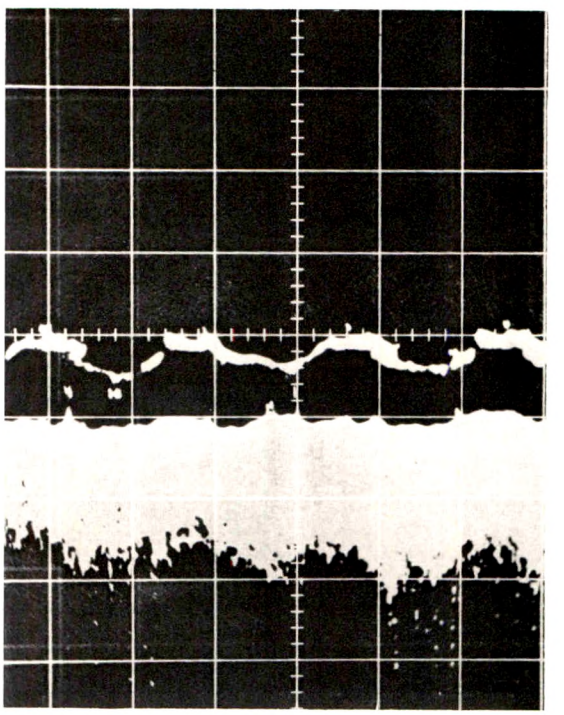


Fig. 2. A typical pericardial effusion. The pulsatile left ventricular echo is separated from the dominant stationary posterior pericardium-lung interface by an echo-free clear space of pericardial fluid.

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will inscribe a sinusoidal curve when the vertical sweep time-motion mode is used on the storage oscilloscope. When a single pulsating echo was obtained, a pericardial effusion was considered to be absent (Fig. 1). A dominant stationary echo from this area may represent the pericardium separated from the pulsatile myocardium by acoustically clear fluid. In this situation, further increase in the strength of the input signal will detect the posterior wall of the left ventricle pulsating anterior to the static pericardium (Fig. 2).

The choice of final position of the trans-

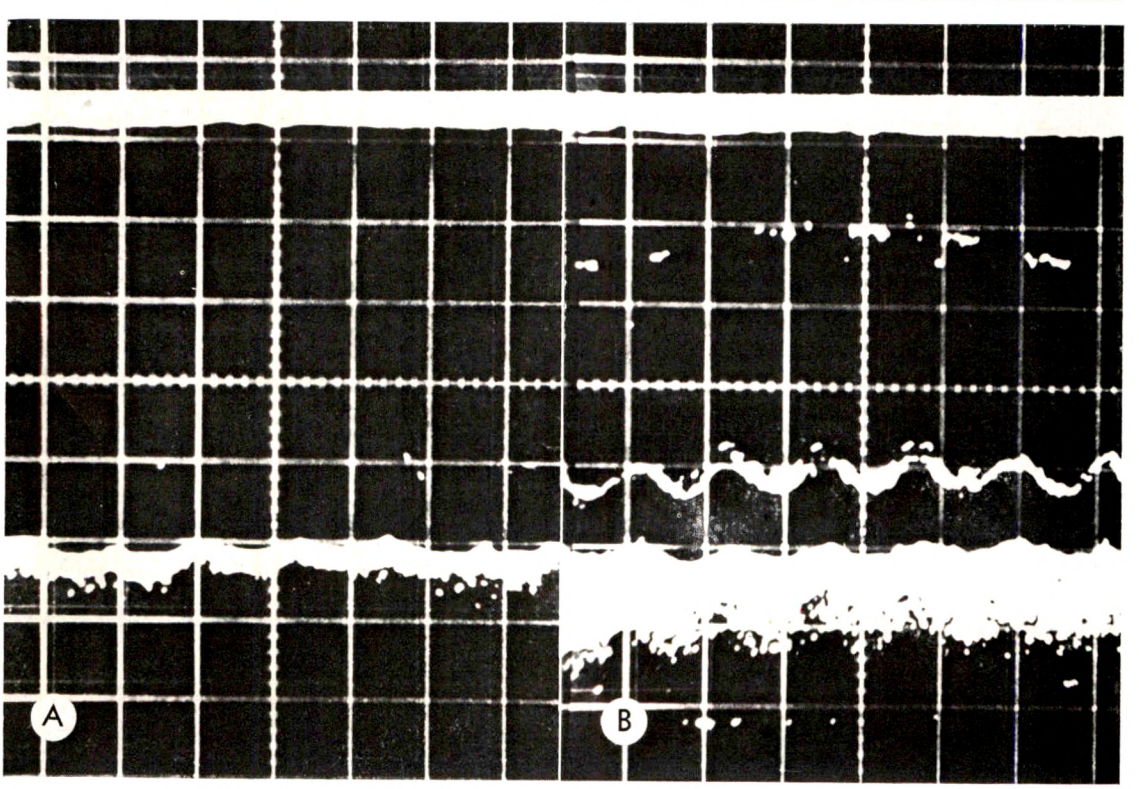
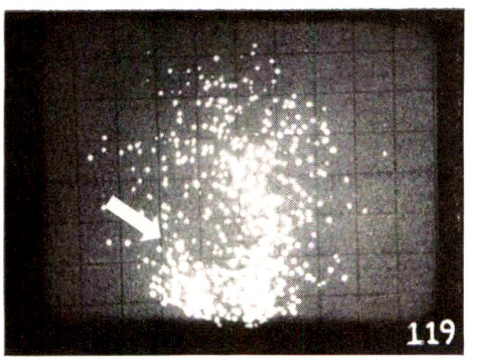
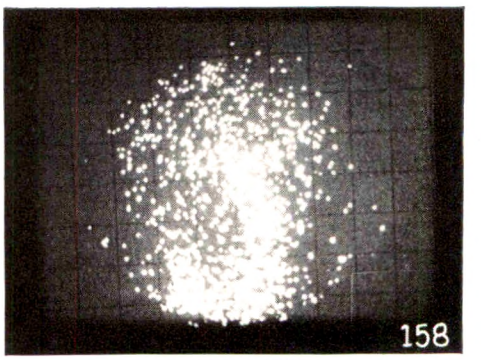


Fig. 3. The pericardium may show definite pulsation. (*A* and *B*) These 2 scans were done on the same patient with lupus erythematosus. (*A*) The pulsatile pericardium is seen. (*B*) By increasing the gain of the ultrasonic signal, the myocardium is detected anterior to the pericardium in this patient with proven pericardial effusion.

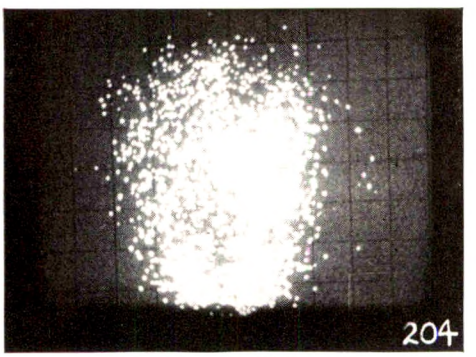
SUPER-8 MOTION-PICTURE STUDY. SELECTED FRAMES. ANTERIOR VIEW.



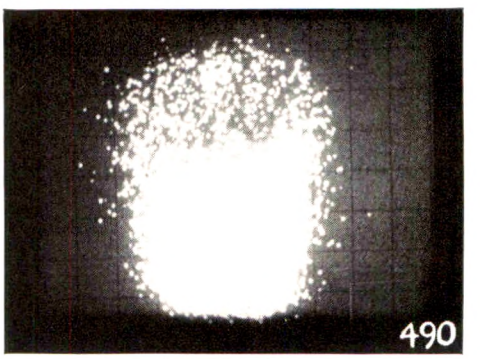
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R ~1.2 SEC. LATER. L



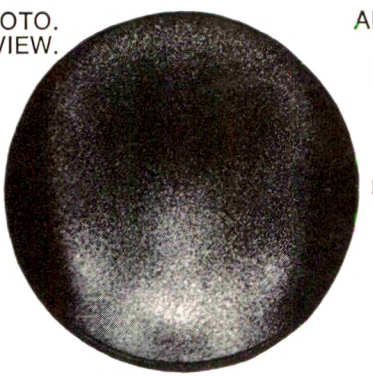
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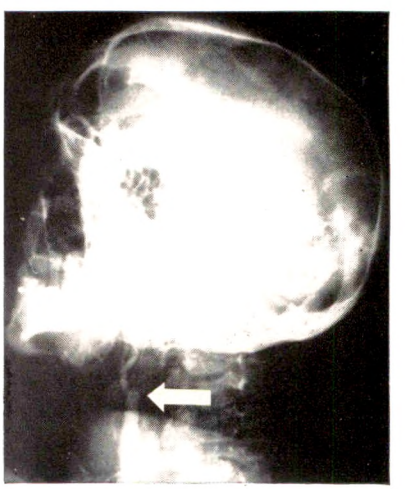
STATIC SCINTIPHOTO. ANTERIOR VIEW.

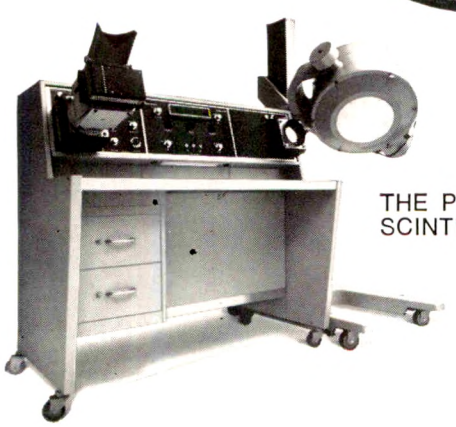
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ARTERIOGRAPHIC STUDY. RIGHT OBLIQUE VIEW.





THE PHO/GAMMA SCINTILLATION CAMERA.

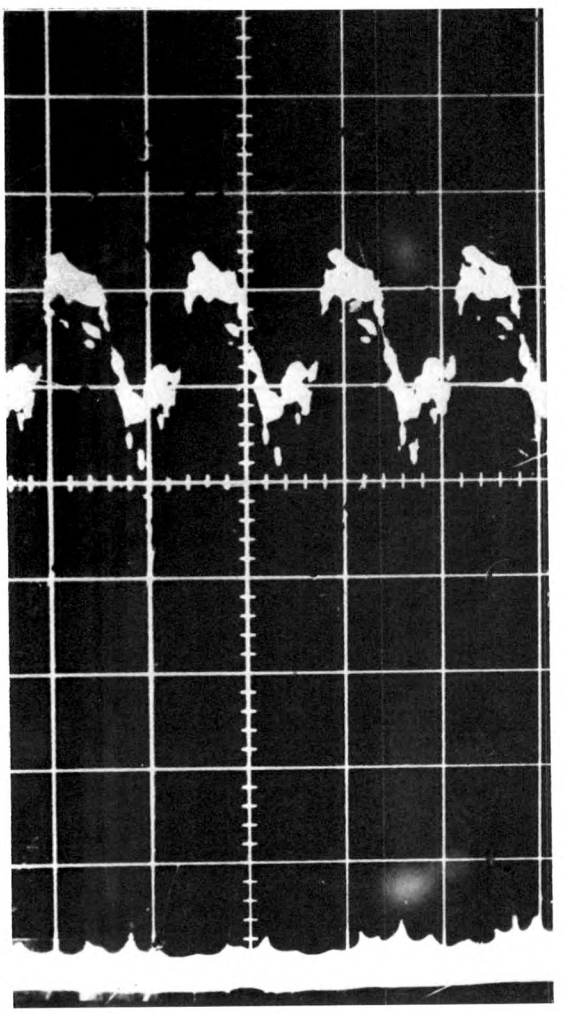


FIG. 4. The echocardiogram of a patient with loculated anterior pericardial effusion. A single echo is obtained from the posterior wall. The strongly pulsatile echo represents the anterior surface of the right ventricle displaced far posterior and masquerading as a stenotic mitral valve.

ducer must be made on the basis of trial and error. The position of the vertical or horizontal cardiac axis can necessitate changes in transducer placement. In a patient with hyperinflated lungs secondary to emphysema or asthma, an adequate scan may be obtainable only in full expiration when the air filled lung is no longer interposed between the chest wall and anterior pericardium.

RESULTS

In judging the value of the echocardiogram, only the initial study was considered although many separate scans were often done on the same patient. The scans done prior to any other confirmatory study were felt to be a more objective test than those performed for follow-up purposes. Therefore in all 100 cases the diagnosis was in doubt at the time of echocardiography. Of these patients, 36 had positive ultrasonic evidence of pericardial effusion, and 64 were negative. Of the 36 positive cases, 21 were confirmed by CO_2 angiography, angiocardiography, pericardiocentesis, surgery or autopsy. The remaining 15 were not studied further because the other clinical data were deemed sufficient to provide the correct diagnosis or because the presence of pericardial fluid was of secondary importance, and no specific intervention was contemplated. Of the 21 positive echocardiograms that had confirmatory studies, 2 were falsely positive.

Of the 64 negative cases, only 15 were studied by other means. In 13 of 15 cases, the lack of pericardial effusion was confirmed. Two instances of false negatives were found. Aside from these 4 false results, we encountered several potential errors in interpretation and technique which could have led to an incorrect diagnosis. Analysis of these pitfalls resulted in changes in technique which hopefully will improve the accuracy of echocardiography.

The following pitfalls in the interpretation of the echocardiogram were encountered:

1. THE PULSATILE PERICARDIUM

This potential error is the result of a pericardium which does not oblige the examiner and remains stationary in the presence of pericardial effusion. Errors in interpretation occurred when a single beating structure was seen in the area of the posterior wall of the left ventricle and mistaken for myocardium when it in fact represented a pulsatile pericardium. Since the pericardium-lung interface produces the dominant posterior wall echo,[9,21] it will always appear first as the gain sensitivity is

Th C r bral "Fl..w" -tudy

Evaluation of Cerebral Vascular "Flow" with the Nuclear-Chicago Pho/Gamma® Scintillation Camera

In this technique using ^{99m}technetium pertechnetate for dynamic study of vascular "flow" pathways (both intra- and extra-cranially), the Pho/Gamma Scintillation Camera is equipped with the Nuclear-Chicago Super-8/Persistence Scope.

SETTING-UP. The standard 4000 parallel-hole collimator is used. The area to be visualized includes the patient's neck and head. With the patient in the supine position, the Pho/Gamma detector is positioned touching the tip of the nose. This orientation can be readily achieved, because of the Pho/Gamma detector's positioning flexibility.

ISOTOPE AND DOSE. An intravenous injection of 10 mC of ^{99m}technetium pertechnetate is administered, preferably in one of the antecubital veins. No attempt is made for a bolus injection.

DATA ACCUMULATION AND DISPLAY. At the first detection of events on the persistence scope (which displays data in live "fluoroscopic" fashion), the scope display is filmed with the Super-8 movie camera. Frame rate is 32 per second. Filming is stopped when the recirculation phase is detected — usually about one minute after the beginning of the study.

Then, approximately one hour later, conventional scintiphotos are taken, in a variety of viewing positions, each representing approximately 250,000 counts.

The motion-picture film is subsequently viewed with the Super-8 Analyst projector in slow, fast, or stop-motion, as necessary for evaluation.

These Pho/Gamma-generated data can also be recorded, in high-resolution digital form, on the Nuclear-Chicago Data-Store/Playback Accessory or on the CDS-4096 Clinical Data System. With either of these system accessories, patient data can be stored and then re-played, processed, and manipulated at the clinician's discretion. The result is an increased range of analysis, yielding additional qualitative and quantitative data.

CASE HISTORY. The clinical study illustrated on the opposite page is of a patient with the following history: Male, 51 years old. Three-month history of intermittent episodes (one to three minutes duration) of right visual-field constriction. Physical examination negative, except for slight blurring of right optic disc.

EVALUATION. In the selected frames from the Super-8 motion-picture film shown at left, these clinically relevant indications are seen: Frame 119, there is no isotope flow through right carotid artery pathway (arrow); note also outline of anterior and middle cerebral artery pathways, with relatively decreased concentration in right hemisphere. In Frame 158 (capillary phase), block in right carotid pathway is still evident. In Frame 204 (venous phase), delayed arterial perfusion in the right hemisphere begins. And, in Frame 490, recirculation with evident delayed arterial perfusion in right hemisphere is seen.

The static scintiphoto shown is essentially negative for any evidence of abnormal isotope accumulation, as were a number of other scintiphotos taken following the Super-8 study.

CONCLUSIONS. In this case, detection and localization of an abnormal "flow" pattern in the Super-8 dynamic study—but not in the static scintiphotos—led to a meaningful differential diagnosis. To this end, a serial arteriographic study was performed. The radiograph selected from that study reveals complete occlusion (arrow) of the right internal carotid artery at the bifurcation with the external carotid on the right. The intra-cranial problem was therefore shown to be the result of extra-cranial pathology.

Thus the Pho/Gamma Scintillation Camera permits the use of a relatively innocuous, yet rapid, technique to produce supplementary diagnostic information. This information can provide direction for the use of other investigative techniques and make possible a more definitive diagnosis. 0-225

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slowly increased. If the study is stopped at this point, the left ventricle pulsating more anterior to the pericardial lung interface will be missed. This situation will lead to a false negative reading. We have noted this phenomenon in both cases of tuberculous pericarditis in our series, and it was the cause of one false negative report. Figure 3, *A* and *B*, shows an example of this problem in a patient with systemic lupus erythematosus.

2. THE LOCULATED EFFUSION

In cases of pyogenic or tuberculous pericarditis there may be areas of adherence between epicardium and pericardium. If this area lies directly posterior over the left ventricle, a false negative result may be obtained. Exactly this situation was the cause of a false negative result in a patient with tuberculous pericarditis. In this case the effusion was loculated anteriorly and laterally. The amount of anterior fluid was so great that the anterior wall of the right ventricle was displaced 8 cm. posterior and was mistaken for a stenotic mitral valve (Fig. 4). This potential pitfall underlines the necessity for carefully checking both the anterior and posterior walls of the heart as well as localizing the mitral valve position in every case. At surgery this patient had adherence of the pericardium and left ventricle to such a degree that they could not be separated (Fig. 5).



FIG. 5. A right-sided angiocardiogram of a patient with a loculated pericardial effusion secondary to tuberculous pericarditis. The anterior surface of the right ventricle is displaced posteriorly and straightened by the loculated pericardial fluid. The posterior heart wall was tightly adherent to the pericardium.

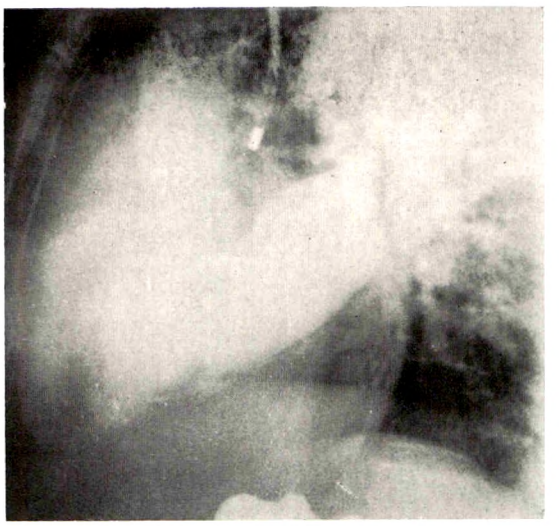


FIG. 6. The levophase of a venous angiocardiogram in a patient with pericardial effusion. The posterior heart surface is seen to course obliquely in the pericardial sac. The amount of separation of myocardium and pericardium depends on the position of measurement as well as the amount of fluid present.

3. THE UNEVEN DISTRIBUTION OF FLUID

It is clear from angiographic studies and experimental work[21] that pericardial fluid does not encase the heart in a shell of uniform thickness. In some areas the pericardium and epicardium may be almost in contact although there is no fixed adherence of the two surfaces. This caused some confusion in one case and renders virtually useless any attempt to accurately estimate the amount of effusion present by measuring the width of the effusion at one point. In most angiocardiographic studies of pericardial effusion the left ventricle was seen to lie obliquely in the pericardial sac

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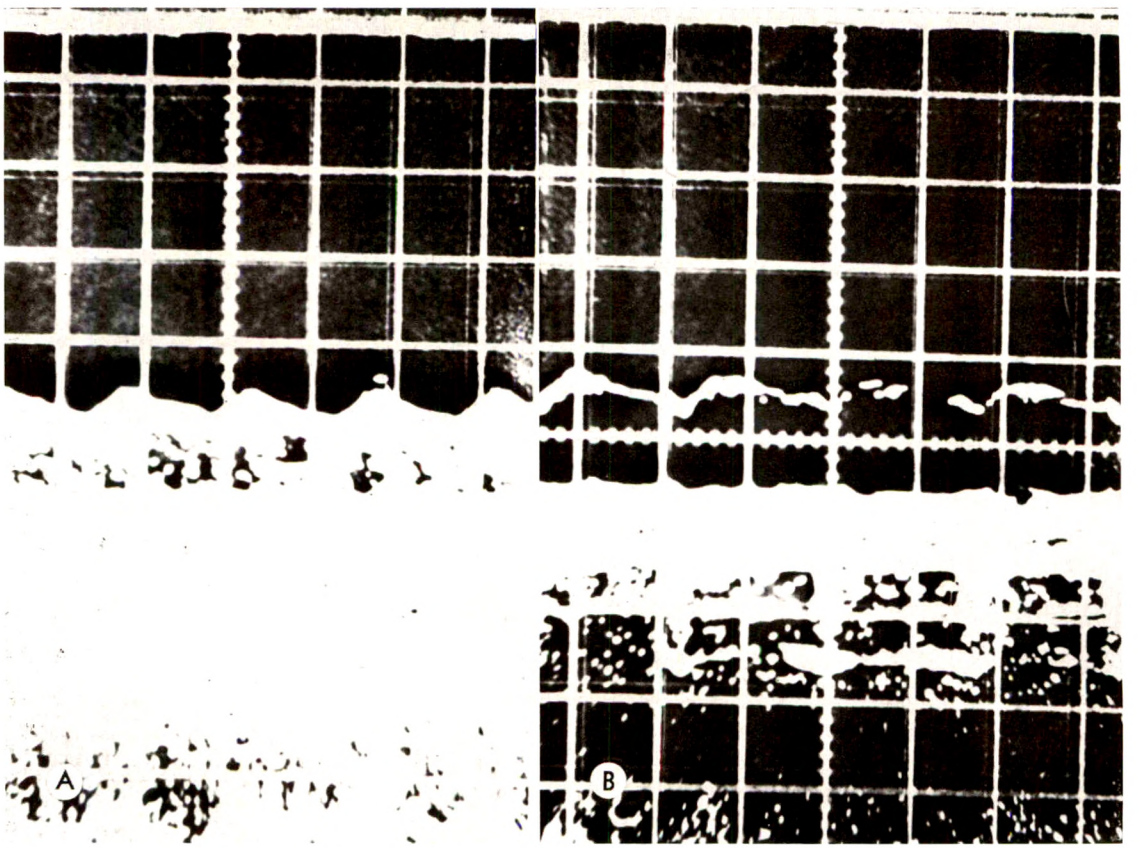


FIG. 7. (*A* and *B*) Using an overly strong signal can artifactually obliterate an effusion. (*A*) The scan gives the impression of a false negative. (*B*) On reducing the signal the true nature of the pericardial effusion is seen.

resulting in varying degrees of separation between the epicardium of the left ventricle and the posterior pericardium (Fig. 6).

4. THE OVERLY STRONG ECHO INPUT SIGNAL

We have already discussed the possibility of false negative results from a pulsatile pericardium. This is a consequence of using too weak an input signal with the result that the less reflective myocardial surface is missed.

False negative results may also be obtained from using an input signal that is excessively strong (Fig. 7, *A* and *B*). This causes the echo-free clear space between the epicardium and pericardium to be filled in by extraneous static thereby obscuring a pericardial effusion.

5. THE IMPRESSION OF EXTRACARDIAC MEDIASTINAL STRUCTURES

As Feigenbaum *et al.*,[6] Goldberg *et al.*,[9] and Soulen *et al.*[22] have described, mediastinal structures can produce false negative or positive scans because of interfering echoes from the spine, pulmonary veins, or aorta. Also a medial placement may result in confusion of the mitral annulus with the posterior wall of the left ventricle. We obtained false negative scans in cases where the transducer was angled very slightly medial to a point where a positive pattern was obtained. Mediastinal structures such as the aorta may indent the pericardium thereby displacing fluid and apposing it to the epicardium (Fig. 8, *A* and *B*). To avoid this possibility, the transducer must be placed lateral to the aorta. This can be done by locating the mitral valve and directing the ultrasound beam well lateral to it. In patients with a tortuous aorta, the beam may have to be directed more laterally than usual.


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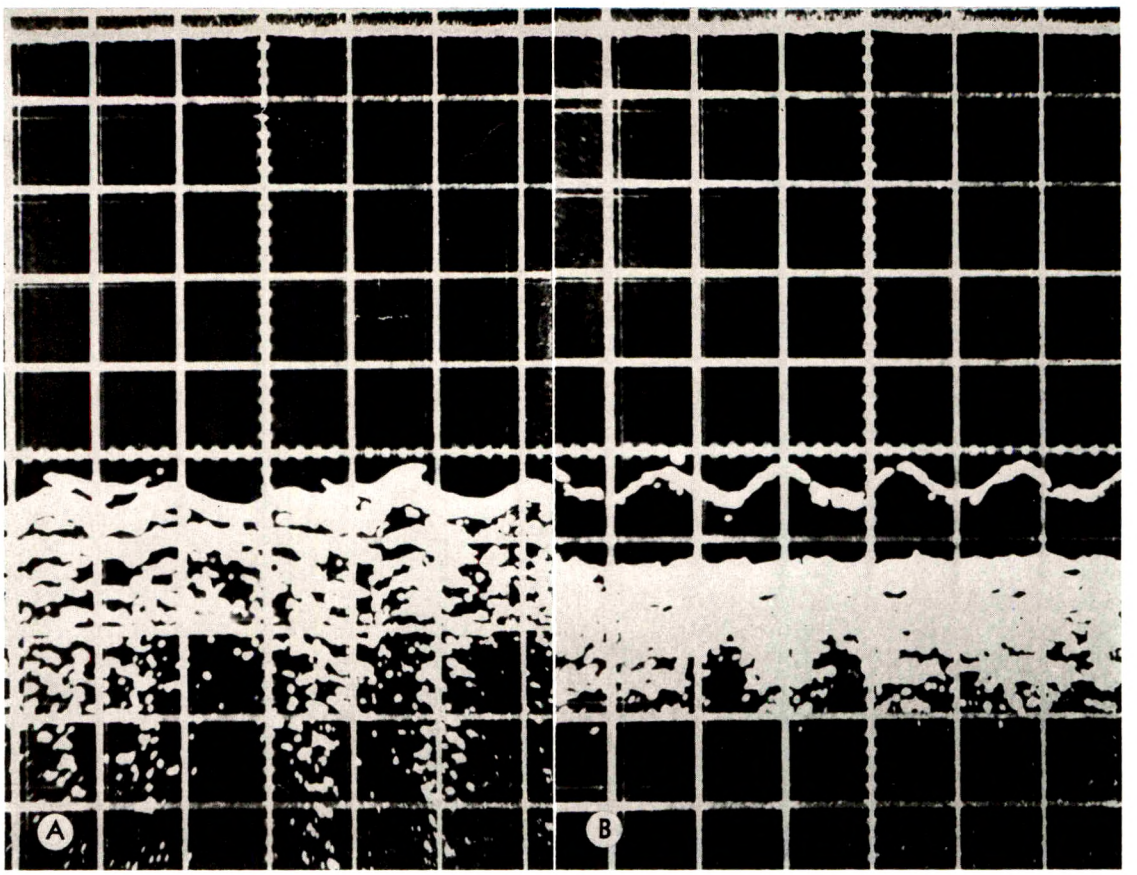


Fig. 8. (*A*) The echocardiogram could easily be misinterpreted as negative. By angling the transducer only a few millimeters medially, the effusion pattern was obliterated by mediastinal structures such as the aorta impressing the pericardium against myocardium. (*B*) The scan reveals a typical pericardial effusion pattern in a patient with subacute bacterial endocarditis.

6. LEFT PLEURAL EFFUSION

A false positive study may be obtained in the presence of left pleural effusion if the pleural fluid insinuates itself between the heart and pleural surface[9,11] (Fig. 9). If pleural effusion is present, the patient should be examined in the upright or lateral decubitus position.

DISCUSSION

Recognition of these major pitfalls has led to the evolution of the following procedure in performing an echocardiogram.

1. Evaluate the chest roentgenogram to ascertain the presence of left pleural effusion or a markedly tortuous aorta.
2. Locate the mitral valve and annulus and note its position as well as its distance from the anterior chest wall.
3. Direct the ultrasonic beam well lateral of the mitral valve.
4. Turn the gain sensitivity to zero and gradually increase it until posterior structures are seen. Continue to increase the gain to rule out the possibility of the pulsatile pericardium artifact.
5. Direct attention to the anterior heart surface where a clear separation of chest wall and anterior epicardium of the right ventricle may give supporting evidence to the diagnosis of pericardial effusion. It may be the only positive evidence of pericardial effusion if the fluid is loculated.

The echocardiogram is a useful adjunct in the diagnosis of pericardial effusion. Its innocuous nature and safety are great ad-

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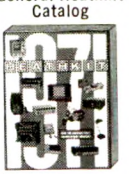
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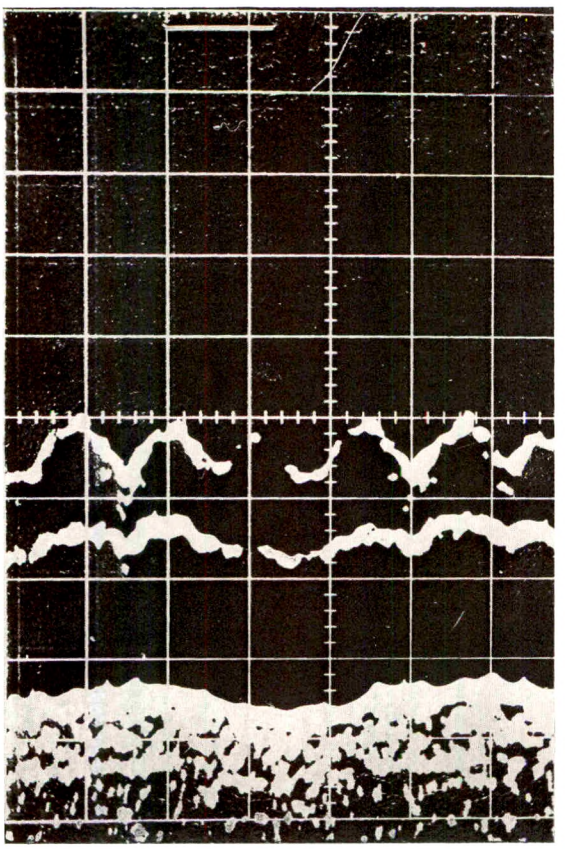


FIG. 9. The echo appearance of a patient with lupus erythematosus who had both pleural and pericardial effusions. The pleural fluid is seen posteriorly between pericardium and pleural surface. This shows how pleural fluid can insinuate itself between pleura and pericardium and produce false positive echocardiograms.

vantages. Its accuracy approached 90 per cent in our series, but serious errors and potential pitfalls were encountered. Recognizing the 6 pitfalls described above should increase the accuracy of the procedure and further establish its usefulness.

SUMMARY

In a series of 100 consecutive patients, the echocardiogram was found to have an accuracy approaching 90 per cent in the diagnosis of pericardial effusion. Analysis of errors and recurring problems revealed 6 pitfalls in interpretation. They are the pulsatile pericardium, the loculated effusion, the uneven distribution of fluid, the overly strong ultrasonic input signal, the impression of extracardiac mediastinal structures, and left pleural effusion.

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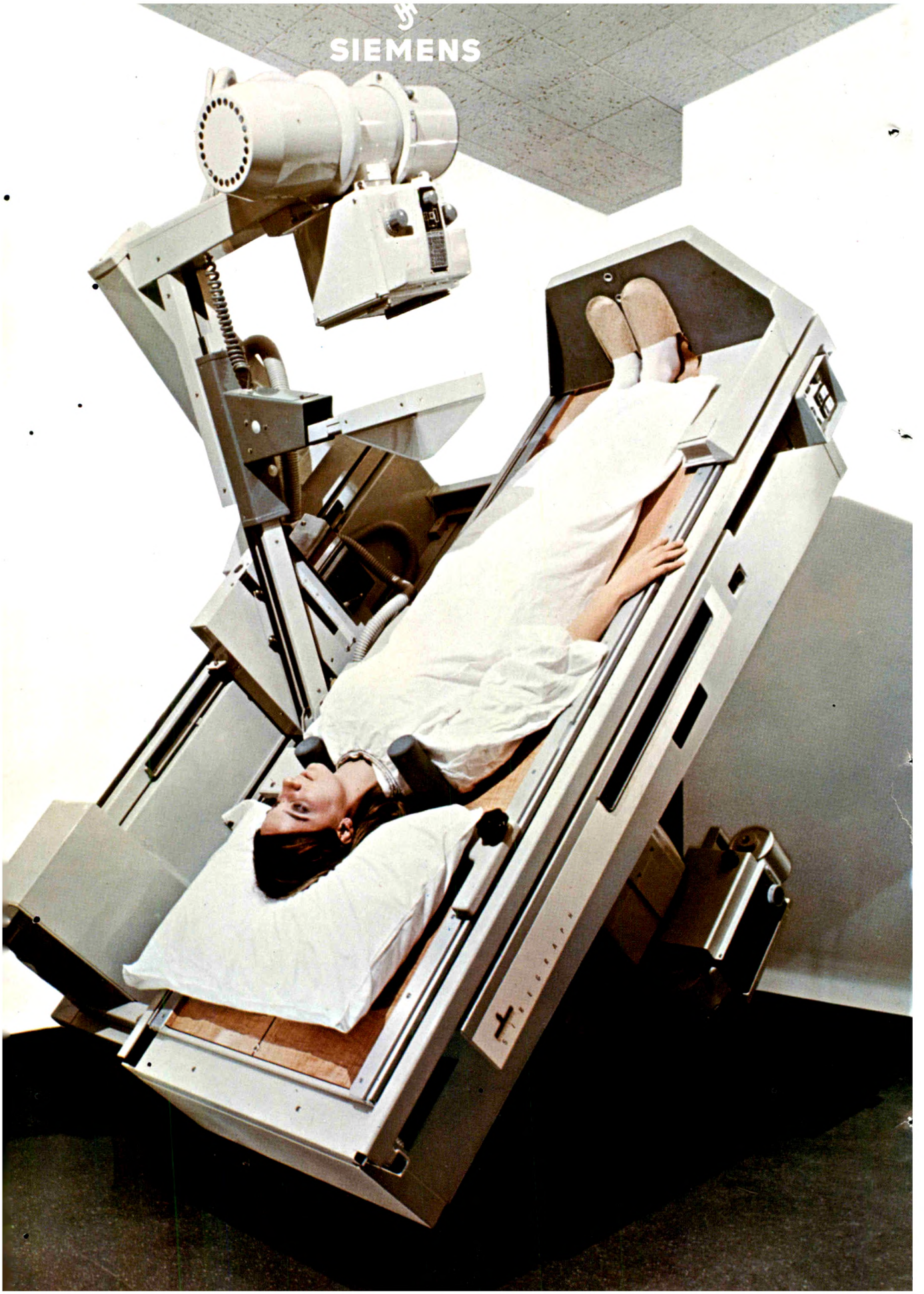
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TRANSPOPLITEAL VENOGRAPHY: AN ALTERNATE METHOD FOR EXAMINATION OF THE ILIOFEMORAL VEINS AND INFERIOR VENA CAVA*

By BRIAN R. SCHNIER, M.D., HARVEY M. GOLDSTEIN, M.D., *and* RONALD A. CASTELLINO, M.D.
STANFORD, CALIFORNIA

TRANSPOPLITEAL venography is an alternate method for evaluating the iliofemoral venous system and inferior vena cava. Since this technique was introduced by Harrell and Grollman,[2,3] no other reports have appeared evaluating its simplicity and usefulness.

The purpose of this communication is to describe our experience with transpopliteal venography and reaffirm its value in selected cases.

METHOD

The popliteal artery is palpated in the popliteal crease while the patient is in the prone position and the knee slightly flexed. After sterile preparation, the skin lateral to the artery is infiltrated with a local anesthetic. The subcutaneous tissues and underlying fascia are carefully spread with a hemostat after nicking the anesthetized skin. An 18 gauge arterial needle is used to puncture the popliteal vein which usually lies *lateral* to the popliteal artery. Attaching a saline-filled syringe to the needle and aspirating while withdrawing aids in determining when the needle is positioned in the vein. Using the Seldinger technique,[4] a radiopaque catheter with side holes is inserted into the vein and advanced under fluoroscopic control to the desired level in the femoral or iliac vein. Percutaneous puncture of the popliteal vein with an Intracath* was used successfully in 1 case,

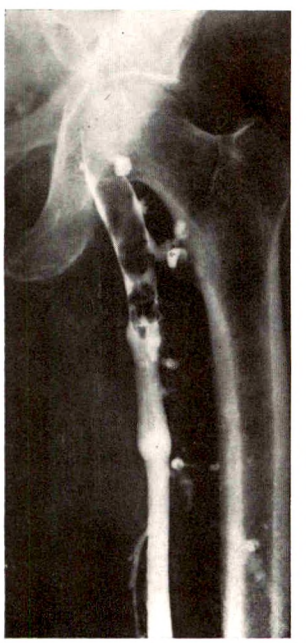


FIG. 1. A 78 year old male developed progressive pitting edema of the left lower extremity and calf tenderness. Peripheral venography did not adequately visualize the femoral and iliac veins. The transpopliteal venogram demonstrated filling defects in the femoral and iliac veins interpreted as thrombi.

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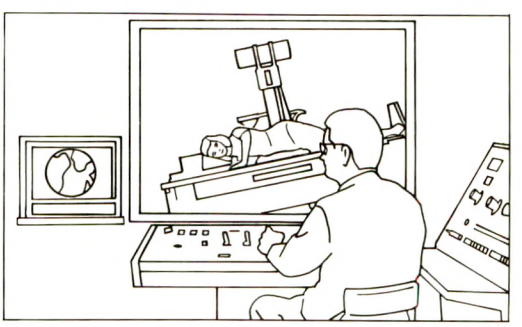
FIG. 3. (*A* and *B*) A 43 year old male underwent inferior vena caval plication and right iliofemoral thrombectomy 2 years previously for recurrent pulmonary emboli and iliofemoral thrombophlebitis. Because of a 5 day history of pain and swelling in both calves and substernal pain with dyspnea, bilateral peripheral venographies were performed (not shown). The femoral and pelvic veins were not adequately visualized. Transpopliteal venograms (above) demonstrated obstruction of the distal inferior vena cava, iliac, and femoral veins bilaterally. The proximal inferior vena cava was patent and filled via extensive collateral channels.

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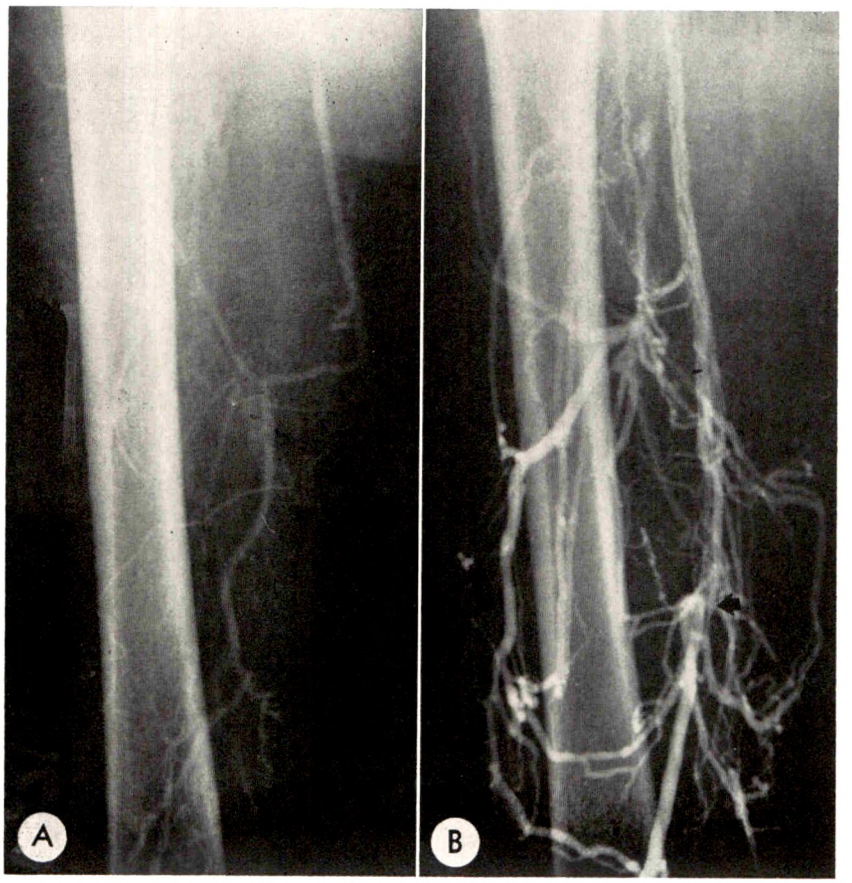
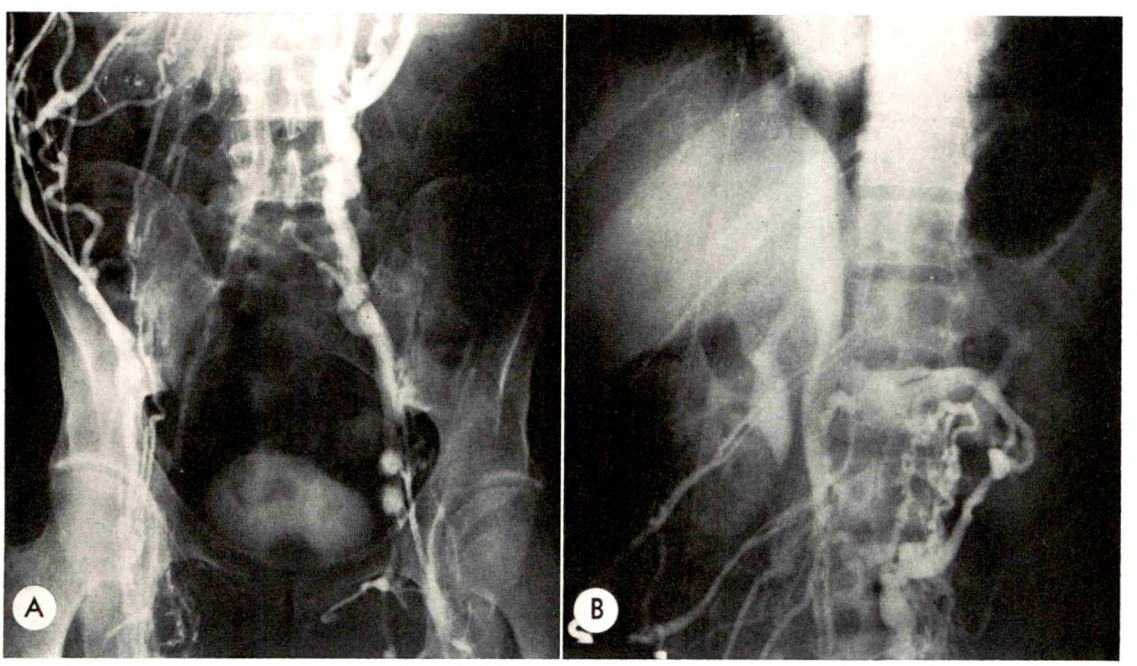
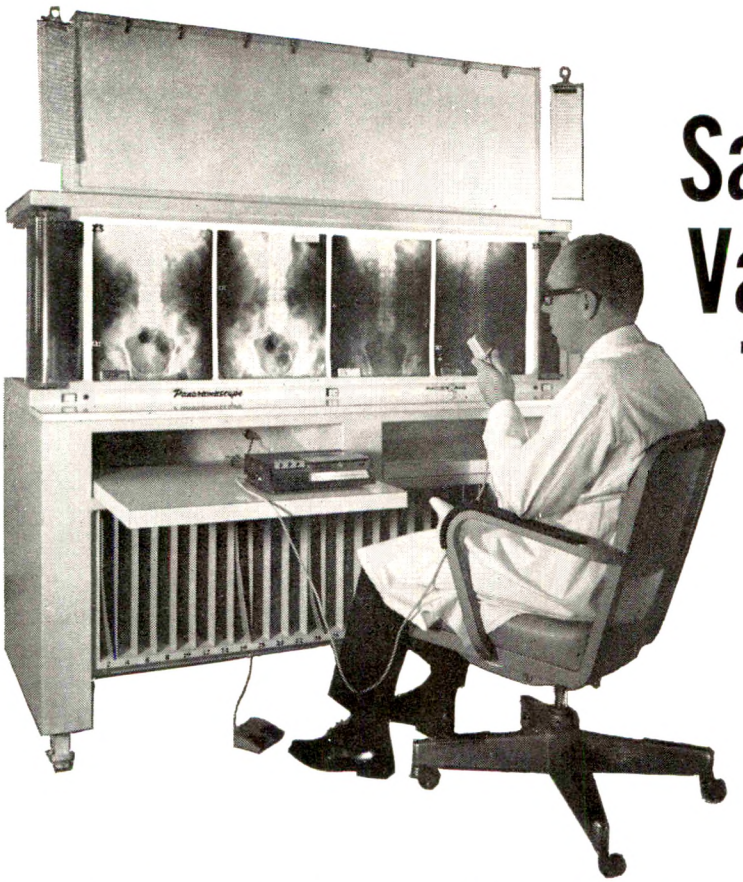


FIG. 2. A 58 year old male developed swelling of the right leg over a 2 week period. (*A*) Peripheral venography failed to visualize the femoral veins adequately. (*B*) A transpopliteal venogram demonstrated partial occlusion of the femoral vein (arrow) with multiple filling defects proximal to this point and multiple collateral veins.





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DISCUSSION

Ten popliteal vein catheterizations were performed in 8 patients. In a ninth patient the popliteal vein could not be punctured. The transpopliteal approach was utilized 5 times because previous peripheral foot venography provided poor evaluation of the femoral and iliac veins (Fig. 1; 2, *A* and *B*; and 3, *A* and *B*). In 2 instances a dorsal foot vein could not be cannulated because of edema (Fig. 4). In another case lymphadenopathy and recent surgery in one groin and a palpable thrombotic vessel in the other precluded femoral vein puncture. The remaining 2 transpopliteal venograms were performed electively to obtain optimal visualization of the iliofemoral junction, since this was the area of clinical suspicion. No complications related to the procedure were encountered.

Transpopliteal venography is particularly valuable when evaluation of the iliofemoral venous system is desired. With conventional peripheral venography performed via a dorsal foot vein, visualization of femoral and pelvic veins is often inadequate due to dilution of the contrast material with nonopacified blood. The transpopliteal technique enhances roentgenographic visualization and detail, since the contrast material is placed at or near the area of interest without significant dilution. This approach is a good alternate method of evaluating the iliac veins and inferior vena

FIG. 4. A 49 year old male with pancreatic carcinoma developed right lower extremity edema and right groin pain. A dorsal foot vein could not be cannulated because of edema. Femoral vein puncture was not attempted because of a palpable, thrombotic vein. A transpopliteal venogram demonstrated obstruction of the femoral vein with multiple filling defects and collateral vessels.

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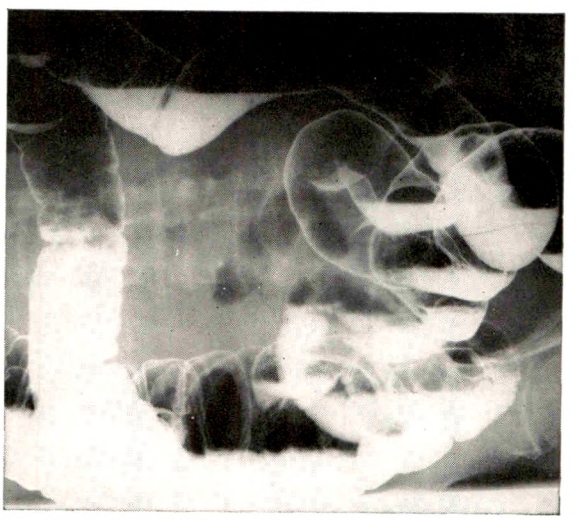
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cava when puncture of the femoral vein cannot be accomplished because of abnormality in the femoral triangle. For example, previous groin surgery or injury, local vein thrombosis, or inguinal lymphadenopathy or infection may make puncture of the femoral vein undesirable or impossible.

We have found it more difficult to puncture the popliteal vein than the femoral vein. This may in part be due to our relative inexperience with this technique. In addition, the vascular anatomy of this region is more variable than in the femoral triangle. The junction of the anterior tibial, posterior tibial, and peroneal veins occurs at varying levels with respect to the knee joint. These veins join above the level of the knee joint so that multiple venous channels in the popliteal fossa is the rule.[5] As the popliteal veins ascend the popliteal fossa, they pass superficially from medial to lateral in relation to the popliteal artery. Although the exact point of crossing from medial to lateral in relation to the popliteal crease and popliteal pulse is variable, the popliteal vein usually lies lateral to the artery at the popliteal crease.

Slight knee flexion facilitates palpation of the arterial pulse making the subsequent venous puncture easier. In addition, knee flexion prevents popliteal vein compression between the femoral condyles and the plantaris muscle, which may occur with full knee extension.[1]

CONCLUSION

Transpopliteal venography provides excellent opacification of the iliofemoral venous system and inferior vena cava. Its greatest value is in cases where femoral vein puncture is undesirable or not possible, or when evaluation of the femoral vein following foot vein venography is inadequate.

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RENAL AGENESIS—ROENTGENOLOGIC PROBLEM*

By D. M. HYNES, M.A., M.R.C.P., F.F.R.,† *and* E. M. WATKIN, M.B., B.S.
SHEFFIELD, ENGLAND

NONVISUALIZATION of a kidney during excretion urography is a common problem confronting the radiologist. There are many causes for this appearance and the final diagnosis rests largely upon roentgenologic techniques. The extent to which further investigation should proceed depends upon the clinical problem, but frequently these patients undergo arteriography.

Such extensive investigation might be considered unnecessary, especially if the diagnosis can be achieved without resorting to arterial studies. It is with this in mind that the authors present a report of a group of patients in whom it was thought that a kidney was either congenitally absent or aplastic.

MATERIAL

All 16 patients in this group have been seen during the past 9 years. Each was discovered initially during routine urography as a "nonfunctioning" kidney. Further investigations included cystoscopy with a view towards retrograde pyelography.

Details of the clinical findings and investigations are outlined in Table 1.

It is evident from Table 1 that the apparent absence of a kidney was an incidental finding, although the patients did present with symptoms referable to the genitourinary tract. The age spectrum was wide–ranging from 7 to 69 years at the time of investigation. There was a preponderance of males to females (11 M:5 F) although most reports indicate that the sex distribution is uniform. The left kidney was absent in 10 of the 16 patients. This tendency for the left kidney to be absent more than the right has been suggested in a previous review.[7]

RESULTS

When confronted with this problem, it is the duty of the radiologist to find the cause for nonvisualization of the kidney. Frequently this is comparatively simple if there is evidence of an opaque calculus or other obstructive lesion. Occasionally, it may be much more difficult, particularly if the presenting symptom strongly suggests active renal disease. Therefore, a diagnosis of an absent kidney is not easy to make with confidence. This group of patients presents several diagnostic features which might lead one to suspect renal agenesis or aplasia.

(A) ABSENT RENAL SOFT TISSUE SHADOW

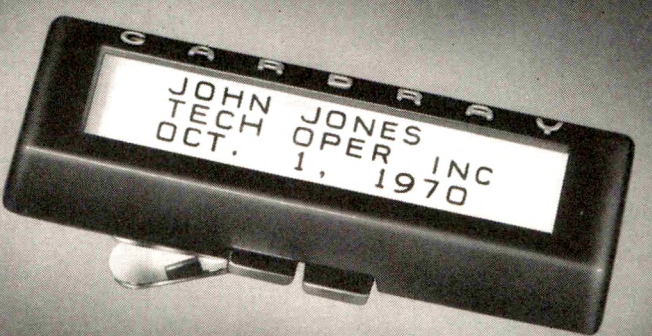
In the majority of normal examinations it is possible to identify both renal shadows on a plain roentgenogram of the abdomen, especially if this examination is combined with tomography. In all the patients described the normal renal silhouette could be identified but there was no evidence of a renal soft tissue shadow on the opposite side. Braasch and Merricks[3] suggested that the psoas shadow might be wider on the affected side, and they reported this finding in 6 out of a total of 36 (16 per cent) cases. We were unable to identify this sign in any of our patients.

(B) ABSENT EXCRETION DURING UROGRAPHY

There was no visualization of the kidney during urography in this series. Furthermore, there was no pyelographic evidence of any etiologic factor. Assuming that the kidney has not been removed surgically and that it does not lie within the pelvis, the possibility of it being absent should be considered. However, it must be remembered that a seriously diseased kidney may

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present these features and that further investigation will be necessary.

(c) hypertrophy of the normal kidney

Good detail of the normal kidney was obtained in every case. This kidney was hypertrophied (Table 1) and appeared bulky, otherwise it presented a normal contour. There was no roentgenographic evidence of bilateral renal disease; *i.e.*, chronic pyelonephritis, hydronephrosis or polycystic disease. There was 1 exception (M.L.) in which the solitary kidney contained a large hypernephroma. This case has been described in a previous communication.[5]

(d) cystoscopy

Fifteen of the 16 patients underwent cystoscopy, and an attempt was made to pass a ureteric catheter for retrograde pyelography. Details of this examination are documented in Table 1. Every case presented an absent ureteric orifice and 3 had definite asymmetry of the trigone. It is interesting to note that 3 other patients had an abnormal bulge at the anatomic site of the ureteric orifice. This was thought to represent an enlarged seminal vesicle or possibly a pelvic kidney.

Case 1. T.S., a male aged 33 years, presented with back pain associated with frequency. Clinical examination was negative but the pyelogram showed no function on the left side. The right kidney was normal and showed some hypertrophy. On cystoscopy there was a small mass indenting the bladder which was obscuring the site of the left ureteric orifice. The top of this mass was removed with a resectoscope. As a result, whitish fluid escaped which contained spermatozoa.

(e) associated anomalies of genito-urinary tract

When there is a congenital absence or aplasia of a kidney it is hardly surprising that other abnormalities of the genito-urinary tract may coexist. Ten of the 16 patients demonstrated such an anomaly and details are presented in Table 1. Both the urinary and genital systems arise from common mesoderm which forms the urogenital ridge and develop in close approximation. However, the final anatomic arrangement depends upon several transitory stages of development. Arrest or delay in the formation of a kidney could possibly disrupt or be associated with some other minor maldevelopments in the genito-urinary system.

(f) arteriographic features

A percutaneous transfemoral renal arteriogram was obtained in each case. This examination confirmed the pyelographic findings of a normal hypertrophied solitary kidney. There were 2 exceptions–M.L. who had a hypernephroma and A.H. who had developed chronic pyelonephritis by the time the arterial studies were carried out. There was no arteriographic evidence of the opposite renal artery or a nephrogram in the abdomen or pelvis (Fig. 1). Apart from this the arteriograms were not remarkable. It should be emphasized that it is important to make a pelvic injection to exclude a pelvic kidney.

Discussion

Renal agenesis is a well recognized condition but its precise incidence is uncertain. Estimates have varied between 1 in 480[8] and 1 in 4,000.[4] Whatever the true incidence, it seems possible that cases will be seen fairly regularly in most general x-ray departments. The 16 patients in this communication were gathered from a total of 18,000 pyelographic examinations. It is stated that the sex incidence is almost equal and that there is a tendency for the left kidney to be absent more than the right.

Associated anomalies of structures arising from the urogenital ridge are documented.[1,2,6] These anomalies may be minor and when present tend to be more common in females. The true incidence of such defects will be higher than indicated in this report. This applies particularly to minor variations in the form of the female genital tract, which can only be examined adequately at operation or post mortem.

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TABLE I

SUMMARY OF CASES

No.	Name	Sex	Age (yr.)	Hy. es and Watkin Clinical Presentation	Blood Pressure (mm. Hg)	Excretion Urography: Visualized Kidney: Side	Excretion Urography: Visualized Kidney: Length	Excretion Urography: Visualized Kidney: Outline	Cystoscopy Contralateral Ureteric Orifice	Aortography: Renal Artery and Kidney: Visualized Side	Aortography: Renal Artery and Kidney: Contralateral Side	Associated Anomalies of the Urogenital Tract
1	A.H.	F	7	Imperforate anus; hypertension	260/170	Right	9.0 cm.	Lobulated	Not performed	Normal	Absent	Imperforate anus
2	J.S.V.	M	12	Left loin pain	120/74	Right	15.0 cm.	Normal	Possible pin-point opening	Artery large	Absent	Ectopic right testis in superficial inguinal pouch
3	C.I.	M	14	Nocturnal enuresis; urinary infection	115/75	Right	15.5 cm.	Normal	Absent	Artery large	Absent	Bladder trabeculated; wide mouthed saccule above right ureteral orifice
4	H.K.M.	M	17	Nocturnal enuresis	120/70	Right	14.5 cm.	Normal	Absent	Normal	Absent	Prostatic bar; short posterior urethra
5	M.P.	F	19	Right loin pain; dysuria	140/80	Left	15.5 cm.	Normal	Absent	Artery large	Absent	None
6	T.B.	M	21	Right-sided abdominal pain	125/70	Left	15.0 cm.	Normal	Absent	Artery large	Absent; small artery probably lumbar	Cystic swelling of upper pole of right epididymis
7	M.W.	M	23	Urethral pain	135/80	Right	15.5 cm.	Normal	Absent	Two arteries	Absent	Soft mass in region of left seminal vesicle
8	H.R.	M	27	Left epididymo-orchitis	140/90	Left	16.0 cm.	Normal	Absent	Normal	Absent	None

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No.	Name	Sex	Age (yr.)	Hynes and Watkin Clinical Presentation	Blood Pressure (mm. Hg)	Excretion Urography: Visualized Kidney Side	Length	Outline	Cystoscopy Contralateral Ureteric Orifice	Aortography: Renal Artery and Kidney Visualized Side	Contralateral Side	Associated Anomalies of the Urogenital Tract
9	T.S.	M	33	Left loin pain; frequency of micturition	125/80	Right	15.0 cm.	Normal	Absent	Normal	Absent*	Swelling containing spermatozoa projecting into left side of trigone
10	S.B.	F	34	Absent vagina	160/95	Right side of pelvis	12.0 cm.	Normal	Absent	Large artery from left common iliac	Absent	Absent left side of trigone; absent vagina; left ovarian cyst; rudimentary uterus
11	D.B.	M	38	Symptomless albuminuria	180/95	Left	17.0 cm.	Normal	Doubtful	Normal	Absent	None
12	A.K.P.	F	43	Right loin pain; frequency of micturition	210/100	Left	14.0 cm.	Normal	Absent	Normal	Absent	None
13	M.L.	M	44	Painless hematuria	140/80	Right	22.0 cm.	Mass of lower pole	Absent	Vascular mass lower pole	Absent	Absent left side of trigone; hypernephroma
14	D.M.	F	47	Hematuria	130/75	Right	16.0 cm.	Normal	Absent	Normal	Absent	None
15	C.C.	M	55	Left scrotal pain	170/100	Right	15.0 cm.	Normal	Absent	Normal	Absent*	None
16	F.M.	M	69	Carcinoma of lung	140/90	Left	16.5 cm.	Normal	Absent	Artery tortuous	Absent	Absent right side of trigone; absent right vas deferens and right epididymis; globular swelling of upper pole of right testis

* No pelvic injection.



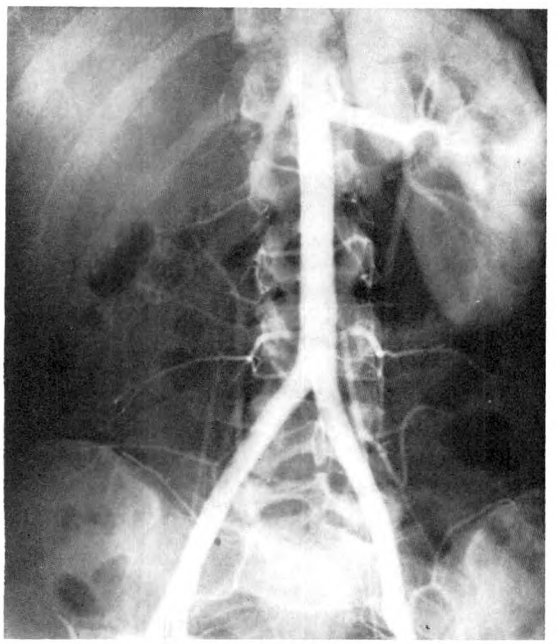


Fig. 1. Case 1. T. B., male, age 21 years. Retrograde aortogram, midstream injection, shows an absent right kidney with a hypertrophied normal left kidney.

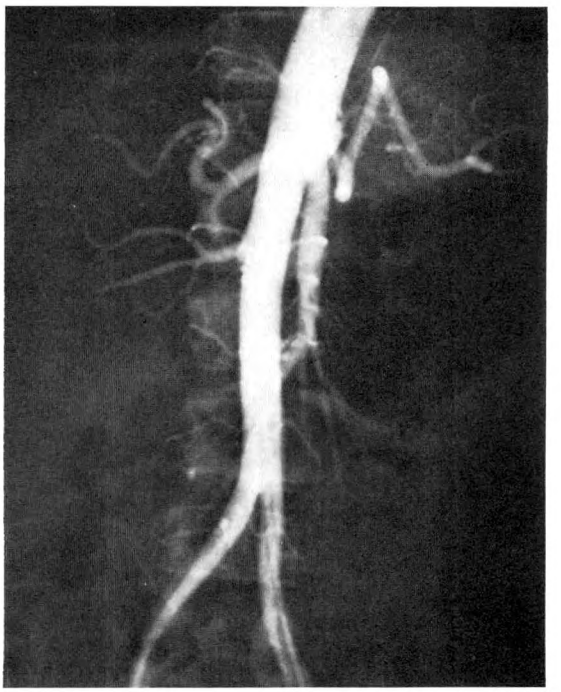


Fig. 3. Case 11. A.H. Retrograde aortogram. Midstream injection reveals no evidence of a left kidney. The right renal artery is present but details of the right kidney are poor.



Larger series describing renal agenesis have been based to some extent on postmortem and occasionally surgical material, which gives an opportunity to examine the genitourinary system in detail. The anomalies in this communication were all identified on clinical or cystoscopic examination.

The combination of plain film roentgenographic and pyelographic features with cystoscopic findings may suggest agenesis or aplasia of a kidney. A significant number of the patients described also had an anomaly of the genitourinary system. In retrospect this information would have been sufficient to make the diagnosis without resorting to arteriography. The neces-

Fig. 2. Case 11. A.H., female, age 6 years. An intravenous pyelogram, 20 minute roentgenogram. No kidney is seen on the left. The right kidney is large (9 cm.) with irregular calyces but normal renal cortex.

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sity of proceeding to arterial studies is open to question and perhaps they need not be carried out if the clinical picture does not suggest severe renal disease.

The difficulty in determining the difference between agenesis and a severely diseased kidney can be exemplified by considering the following case.

Case II. A.H. was a girl born on March 9, 1959 without complications. Routine physical examination after birth revealed an imperforate anus which was repaired by dissection and suture. Shortly afterwards she developed a rectovaginal fistula and had repeated attacks of urinary tract infection.

In 1961 the rectovaginal fistula was repaired successfully. She remained well with sterile urine and developed normally. In 1965 she was investigated following a blackout. Excretion urography showed a large, rather irregular right kidney, but no kidney on the left (Fig. 2). She had another blackout in 1966 when she was found to be hypertensive (240/180 mm. Hg). Neurologic investigation failed to reveal any intracranial abnormality and aortography was performed to exclude a remediable renal cause for the hypertension. This confirmed the pyelographic findings of an absent left kidney. On the right side the renal artery was normal but the details of the right kidney were poor (Fig. 3).

One month later she developed acute renal failure and died. A postmortem examination revealed: (1) an absent left kidney—the kidney was replaced by a small fibrous mass. Histology showed no evidence of renal elements; (2) a large pale right kidney which contained multiple small abscesses. Histology revealed chronic pyelonephritis and did not suggest fusion of 2 renal elements; and (3) both ureters were present. The left ureter was a thin cord but a small probe could just be passed in its lower third.

This was, therefore, a patient in whom there was a congenital aplasia of the left kidney and an imperforate anus which was complicated by the development of chronic pyelonephritis and hypertension during the following 7 years. The diagnostic problem was to decide whether the left kidney was absent or severely diseased by pyelonephritis. The differentiation was extremely difficult, but the fact that the right kidney was large, apparently normal with an associated imperforate anus and a negative aortogram led to the correct interpretation of congenital absence or aplasia of the left kidney.

SUMMARY

Sixteen cases suspected of agenesis or aplasia of a kidney are presented. Some characteristic roentgenographic and clinical features are outlined and it is suggested that arteriography may not always be necessary.

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RENAL EMPHYSEMA*

A CASE REPORT AND REVIEW OF THE LITERATURE

By CHARLES S. LANGSTON, M.D.,† *and* RICHARD C. PFISTER, M.D.‡
BOSTON, MASSACHUSETTS

RENAL emphysema is a rare but frequently fatal complication of renal infection in the diabetic patient. The diagnosis can be made only roentgenographically.

This report describes a successfully treated case and reviews the roentgenology, therapy, and pathology of the 27 cases which have been reported in the literature since 1898.

REPORT OF A CASE

J.M., a 65 year old white male diabetic, was admitted to the Massachusetts General Hospital on May 11, 1970 with a complaint of fever and chills of 3 days' duration. The patient was a known diabetic of 14 years' duration who had been well controlled on chlorpropramide. In 1954 he underwent total colectomy for ulcerative colitis following which he remained in good health except for frequent lower urinary tract infections which cultured multiple *coliforms* but appeared to respond to antibiotic therapy.

In September 1968, he presented with hematuria, hesitancy and frequency. An intravenous pyelogram revealed slight irregularity of the minor calyces consistent with the diagnosis of papillary necrosis, more prominent on the left side, and prostatic enlargement. *Klebsiella aerobacter* was cultured from the urine. At this time he underwent the first of 2 prostatectomies. The postoperative course was complicated by *Klebsiella* septicemia.

In August 1969, a second prostatectomy was performed to remove residual prostatic tissue. A repeat intravenous urogram was unchanged. The postoperative course was again complicated by *Klebsiella* urinary tract infection and septicemia. The organism was sensitive only to colistin, and was also cultured from the sputum. In November 1969, the patient underwent circumcision for phimosis following which urine cultures were still positive for *Klebsiella aerobacter*. In December 1969, he was admitted for a *Klebsiella* pneumonia, septicemia and urinary tract infection from which he recovered uneventfully and was discharged in January 1970 with a sterile urine culture.

He did well until May 7, 1970 when he was seen in the Emergency Room with fever and pyuria for which chloramphenicol was prescribed. A plain roentgenogram of the abdomen obtained at this time was normal. He was admitted 4 days later with persistent fever and chills.

Physical examination revealed a lethargic white male with a temperature of 102.4° F.; pulse 90; respiration 40; blood pressure 120/74 mm. Hg; and moderate dehydration. There was voluntary guarding of the abdomen without definite tenderness. The remainder of the examination was unremarkable for an elderly diabetic with an ileostomy.

Initial laboratory values were: hematocrit 32 per cent, white blood cell count 22,000, with a shift to the left; urine loaded with red blood cells, white blood cells and bacteria; urine sugar 4+ but negative for acetone; blood sugar (BS) 630; blood urea nitrogen (BUN) 78; sodium 120; chloride 89; potassium 4.9; carbon dioxide 17; and pH 7.36. Six blood cultures grew *Klebsiella aerobacter*. Medical therapy for urinary tract infection and sepsis was begun.

On the morning following admission the patient was referred for evaluation of moderate uremia, renal infection, and possible necrotizing papillitis. The patient was prepared for infusion nephrotomography.

After the initial roentgenogram was seen (Fig. 1), nephrotomography was carried out revealing a nonfunctioning left kidney which was diffusely mottled by gas-containing abscesses (Fig. 2). The kidney was outlined by a crescent of gas within Gerota's fascia. Gas

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‡ Instructor in Radiology at the Massachusetts General Hospital, Harvard Medical School; Assistant Radiologist, Massachusetts General Hospital.

The great visual-izer

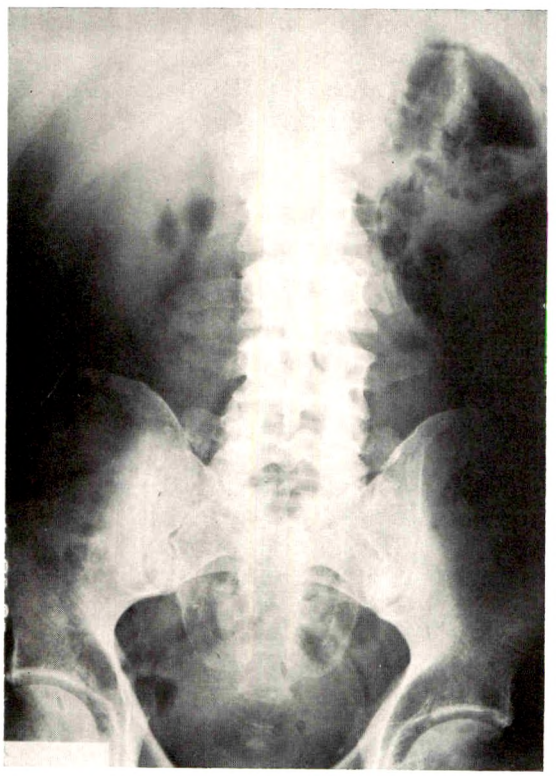


Fig. 1. Preliminary abdominal roentgenogram showing extensive air overlying the left renal bed.

was also seen within the renal pelvis (Fig. 3). The right kidney was essentially normal in function. A retrograde pyelogram prior to surgery revealed no evidence of obstruction, little change in the minor calyces, and a filling defect in the renal pelvis which was thought to represent necrotic tissue or blood clot (Fig. 4).

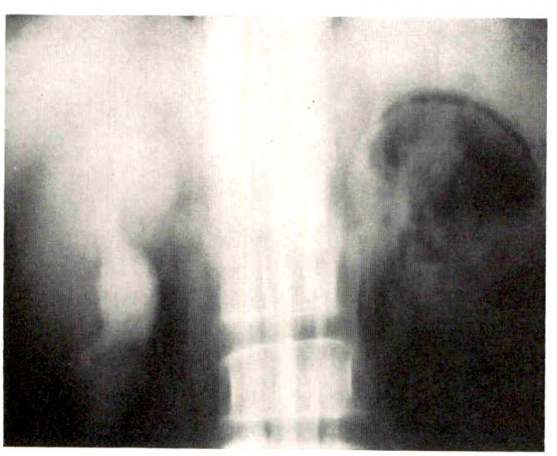


Fig. 2. Frontal nephrotomogram. Nonfunctioning left kidney is replaced and outlined by air. Normal right kidney.



Fig. 3. Oblique tomogram (detailed view) of spontaneous renal emphysema. Mottled intra- and perirenal gas is well seen. Renal margin (white arrows) is delineated by air within Gerota's fascia. Air is also present in the renal pelvis (black arrows).



Fig. 4. Retrograde pyelogram showing necrotic debris within the renal pelvis and absence of ureteral obstruction. Artifact overlies middle minor calyx.

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1. **Aortogram:**
   Ectopic left kidney
2. **Selective Renal Arteriogram:**
   Adenocarcinoma of kidney
3. **Aortogram:**
   Dual Renal Blood Supply
4. **Aortogram**
5. **Excretory Urogram**
6. **Selective Renal Angiogram:**
   Renal cyst

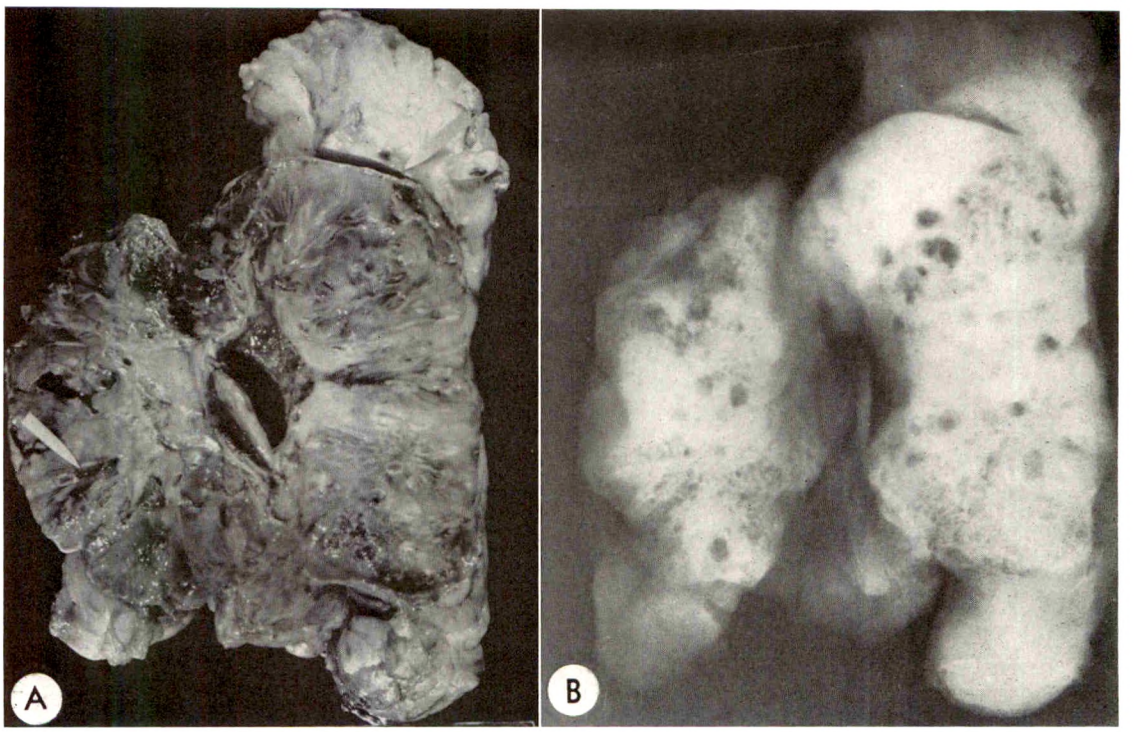


FIG. 5. (*A*) Opened surgical specimen. Diffuse, small, confluent *Klebsiella* abscesses are present. (*B*) Roentgenogram of specimen correlates closely with the pathologic findings.

A diagnosis of spontaneous renal emphysema was made. That evening an emergency nephrectomy was performed. Pathologic examination confirmed the roentgenologic findings and revealed the kidney parenchyma to be almost totally destroyed by yellow and black purulent abscesses (Fig. 5, *A* and *B*). The abscess formation extended locally into the perinephric fat for 2–3 mm. at both poles. Air could be palpated between the kidney and the surrounding fat. There was no evidence of vascular thrombosis. The renal pelvis was hemorrhagic and contained purulent material which extended 2.5 cm. down the ureter. The ureteropelvic junction was normal.

Postoperatively the patient was treated with colistin and then gentamycin. His clinical and metabolic status improved rapidly, and by the fourth postoperative day he was able to sit in a chair, although his wound continued to drain purulent material culturing *Klebsiella*.*

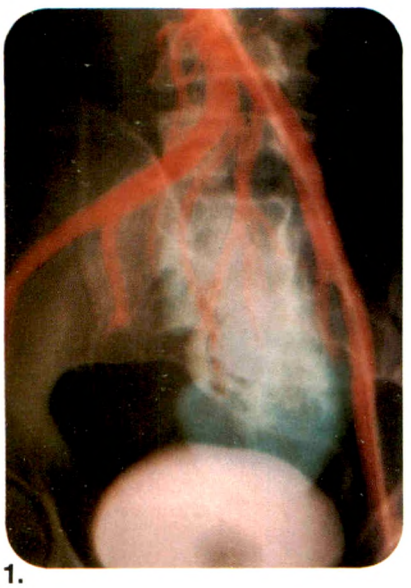
DISCUSSION AND REVIEW

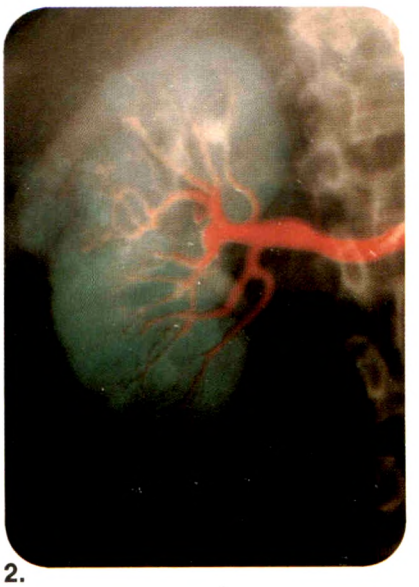
Spontaneous generation of gas within the renal parenchyma and surrounding tissues occurs only rarely and appears to be of grave prognostic significance. The underlying pathophysiologic process is the production of carbon dioxide and hydrogen through fermentation of sugar by aerobic non-spore-forming bacilli. In the cases reported in the literature (Table 1), the organisms have been those normally found in the gastrointestinal tract: *Escherichia coli*, *Pseudomonas*, *Aerobacter aerogenes* and, in this report, *Klebsiella aerobacter*, all of which break down many carbohydrates into acid and gas.

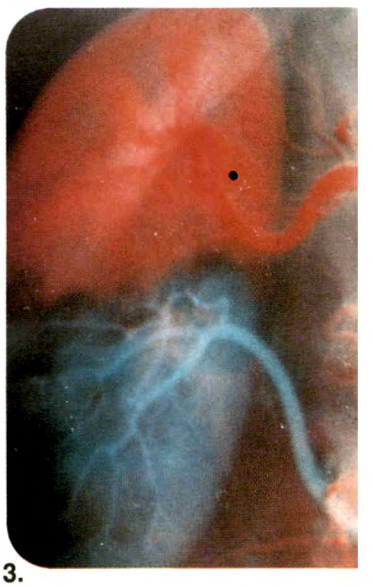
It has been postulated in the past that the triad of diabetes mellitus, obstruction, and coliform organism is necessary for the production of renal emphysema[21] and this must at least be true in part. Certainly, the overwhelming majority of the reported cases (82 per cent) have been diabetics and all have cultured enteric bacteria. However, urinary obstruction has not been a constant feature, being found in only 46 per

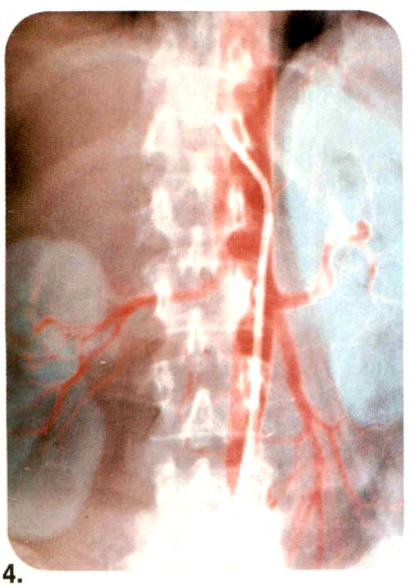
* The patient died 10 weeks following nephrectomy, as a result of *Klebsiella* tonsillar abscess and pneumonia which developed 4 weeks postoperatively. Postmortem examination was refused.

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TABLE I

CLINICAL FINDINGS

Authors	Age	Sex	Diabetes	Side	Obstruction	Function	Organism	BS	BUN	Duration PTA	Duration Hosp.	Presentation	Treatment and Result
Kelly and MacCallum[14] 1898	36	F	No	L	—	—	—	—	—	—	—	—	I&D; lived
Randall[26] 1927	44	F	No	L	—	—	*E. coli* *Proteus*	—	—	—	—	—	Nephrectomy; lived
Mathe and De la Pena[20] 1933	40	M	No	L	Yes	—	*E. coli*	—	—	—	—	—	Nephrectomy; lived
Nogueira[22] 1935	38	M	No	L	Yes	—	—	—	—	—	—	—	Medical; lived
Olsson[24] 1939	69	F	Yes	L	No	No	*E. coli*	—	—	3 mo.		Coma	Medical; died
Jensen[13] 1939	37	F	No	R	Yes	—	*E. coli*	—	—	—	—	—	Nephrectomy; lived
Alexander[1] 1941	63	F	Yes	L	No	No	*E. coli*	350	50	—	—	Fever, flank pain	Nephrectomy day 6; died
Gillies and Flocks[10] 1941	52	F	Yes	Both	Yes	Yes	*E. coli*	Elev.	Elev.	3 da.	3 wk.	Fever, flank pain	I&D; died
Harrison and Bailey[11] 1942	46	F	Yes	Both	Yes	No	*E. coli*	Elev.	Elev.	2 da.	3 wk.	Flank pain	Nephrectomy; died
Plagzemeyer *et al.*[25] 1947	62	M	Yes	Both	Yes	—	*Proteus*	Elev.	—	—	—	—	Medical; lived
Welch and Prather[31] 1949	48	F	Yes	L	No	—	*E. coli*	440	40	3 da.	3 da.	Fever, shock	Nephrectomy; died
Stokes[30] 1949	55	F	Yes	R	Yes	—	*Aerobacter a.*	Elev.	116	3 da.	12 da.	Fever, flank pain	I&D; died
Levy and Schwinger[18] 1953	65	F	Yes	R	No	No	*E. coli*	213	215	3 da.	1 da.	Fever, flank pain	Medical; died
Braman and Cross[6] 1956	60	F	Yes	L	No	No	*E. coli* *Aerobacter*	600	78	3 da.	20 da.	Stuporous, flank pain	I&D; lived
Olson[23] 1957	19	F	Yes	L	—	No	*E. coli*	—	—	1 wk.		Chills	I&D; nephrectomy; lived
	57	F	Yes	L	Yes	—	*E. coli*	—	—	8 mo.		Fever, chills	Nephrectomy; lived
McLelland[21] 1962	53	F	Yes	L	Yes	Yes	*E. coli*	—	—	—	—	Flank pain, shock	Lithotomy; died
Schultz and Klorfein[28] 1962	35	F	Yes	R	—	No	*E. coli* *Proteus*	Elev.	Elev.	1 mo.		Shock	Medical; died
Harrow and Sloane[12] 1963	71	F	Yes	L	No	No	*Proteus* *Aerobacter*	Elev.	Elev.	—	—	Diabetic acidosis	Nephrectomy; lived
Yu[32] 1966	53	M	Yes	R	No	No	*Coliforms*	322	192	2 da.	1 mo.	Fever, coma	I&D then nephrectomy; lived
Klein *et al.*[15] 1966	74	M	Yes	L	No	No	*Aerobacter a.*	630	35	2 wk.	3 da.	Chills, fever	Medical; died
	69	M	Yes	R	—	—	*E. coli*	472	96	3 da.	8 da.	Stupor, flank pain	Nephrectomy; died
Schainuck *et al.*[27] 1968	20	F	Yes	L	No	No	*E. coli*	340	10	2 da.	2 mo.	Abdominal pain	Nephrectomy; lived
Banks *et al.*[3] 1969	75	F	Yes	R	No	—	*E. coli*	—	—	2 da.	2 da.	Flank pain, fever, stupor	Medical; died
	53	F	Yes	L	—	—	*E. coli*	—	—	5 da.	7 da.	Fever, stupor	Medical; died
	74	M	Yes	R	No	—	—	—	—	3 da.	3 da.	Fever, stupor	Medical; died
	69	M	Yes	L	Yes	—	*E. coli*	—	—	3 da.	8 da.	Fever, obtunded	Nephrectomy; died
Langston and Pfister 1970	65	M	Yes	L	No	No	*Klebsiella Aerobacter a.*	630	78	7 da.	—	Chills, fever	Nephrectomy; died of late complications

BS = blood sugar; BUN = blood urea nitrogen; PTA = patient ambulatory; Hosp. = hospitalized; Elev. = elevated; I & D = incision and drainage.

Summary of Full Prescribing Information

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CRETORY UROGRAPHY AND IP INFUSION PYELOGRAPHY

cription: Hypaque sodium is sodium 3,5-diacetamido-2,4,6-triiodo-zoate, a radiopaque medium used to delineate internal structures. aque sodium 50 per cent contains 300 mg. iodine per ml. Contains ium disodium edetate 1:10,000 as stabilizer. Na_2CO_3, NaOH, and added to adjust pH between 6.5 and 7.5. Not for multiple dose use. uld be protected from strong light.

ning: Do not use Hypaque sodium for myelography. Injection of n a small amount into the subarachnoid space may produce convul-ıs and result in fatality.

ieral and Specific Precautions: Caution is advised in patients with a ory of bronchial asthma, other allergic manifestations, or of sensi-y to iodine; these conditions represent a special risk, though not an olute contraindication. To avoid or minimize possible allergic reac-s, premedication with antihistamines may be considered. **Benadryl® ınd of diphenhydramine hydrochloride) in the same syringe with aque may result in precipitation.** A test dose of contrast medium is entirely reliable. Severe reactions, including fatalities, have occurred ı a test dose as well as with the full diagnostic dose.

tion is advised in patients with severe cardiovascular disease. In ents with impaired renal function and cardiovascular disease, drip ısion pyelography imposes a sudden osmotic and sodium load which precipitate congestive heart failure. Contrast media injected into ries or veins can promote sickling of red cells in susceptible indi-ıals. In patients with reduced renal function, repeat of excretory or ograde pyelography should be delayed at least 48 hours to avoid porary suppression of urine. In preparation for excretory urography ıatients with multiple myeloma, partial dehydration is not recom-ıded, since this may predispose to the precipitation of myeloma ein in the kidney tubules. When thyroid function studies (PBI and ıour radioiodine uptake levels) are indicated, they should be per-ned *prior* to radiographic studies or several days afterwards, to id inaccurate results.

ieral and Specific Adverse Reactions: Adverse reactions, usually of ıinor nature, have occurred in 10-14 per cent of patients who have ɛived Hypaque intravenously. Reactions due to *faulty technique* ude hematomas and ecchymoses, following extravasation from the ı, and pyrogenic reactions. *Hemodynamic reactions* include vaso-tation with flushing, hypotension and, rarely, vein cramp or throm-hlebitis. Serious *cardiovascular reactions* include rare cases of car-: arrhythmias (e.g., ventricular fibrillation), shock, and cardiac ıst. Transient proteinuria may occur occasionally following the injec-of radiopaques and, rarely, oliguria and anuria have been reported ɔndary to a hypotensive reaction. *Allergic reactions* include asthmatic cks, nasal and conjunctival symptoms, cutaneous reactions such as ɛaria and, rarely, anaphylactic shock, sometimes with fatal out-ıe. Severe reactions may also be manifested by signs and symptoms ting to the *respiratory system* (dyspnea, cyanosis, pulmonary or ngeal edema), or to the *nervous system* (restlessness, confusion, or vulsions). *Other reactions* include nausea, vomiting, excessive sal-ion, anxiety, headache, and dizziness. Infrequently, "iodism" (salivary ıd swelling) from organic compounds appears two days after ex-ure and subsides by the sixth day. Reactions to drip infusion pyelog-ıy may not appear until some hours after the examination.

IGIOGRAPHY (PERIPHERAL ARTERIOGRAPHY ID VENOGRAPHY)

ɛautions *(See also General Precautions):* Extreme caution is advised onsidering peripheral arteriography in patients suspected of having ımboangiitis obliterans (Buerger's disease). Any procedure (even dling or insertion of a catheter) may induce a severe arterial and/or ous spasm. Caution is also advisable in patients with severe isch-a associated with ascending infection.

erse Reactions *(See also General Adverse Reactions):* In addition to ɜe listed under General Adverse Reactions, are those due to arterial ima during the procedure (i.e., needling, insertion of catheter, sub-injection, perforation, etc.) as well as to the hypertonicity or of the medium; also reported are transient arterial spasm, extra-ıtion, hemorrhage, hematoma with tamponade, injury to nerves dimal to artery, thrombosis (rare in venography, if vein is irrigated ɔwing injection), dissecting aneurysm, arteriovenous fistula, (e.g., ı accidental perforation of femoral artery and vein during needling), ısient leg pain from contraction of calf muscles in femoral arteriog-ıy; transient hypotension after intraarterial (brachial) injection, and ɔhial plexus injury with axillary artery injections.

REBRAL ANGIOGRAPHY

ɜmuch as cerebral angiography is a highly specialized procedure re-ing the use of special techniques, it is recommended that Hypaque um (brand of sodium diatrizoate) be used for this purpose only by ɜons skilled and experienced in carrying out the procedure.

traindication: Carotid angiography should be avoided during the ɡressive period of a stroke because of the increased risk of cerebral plications.

ɛautions *(See also General Precautions):* Select patients for this pro-ıre with care. Use the 50 per cent solution with caution in extreme lity (but not old age *per se*), advanced arteriosclerosis, severe hy-ension, and cardiac decompensation.

diagnostic value of the procedure, according to many authorities, n employed early enough in locating lesions amenable to surgery, outweigh the added risk to patients who have recently experienced b embolism or thrombosis (stroke syndrome). A small number of postangiographic fatalities, including progressive thrombosis, have occurred in which the procedure did not appear to play a role. Patients with severe cerebrovascular disease may be examined primarily by indirect methods of angiography.

Care should be exercised to avoid contaminating catheters, syringes, needles, and contrast media with glove powder or cotton fibers.

Angiography is hazardous in subarachnoid hemorrhage. In migraine, the procedure can be hazardous because of ischemic complications, particularly if performed during or soon after an attack.

Adverse Reactions *(See also General Adverse Reactions):* The major sources of complications are faulty technique, occlusive atherosclerotic vascular disease, repeated injections of the contrast media, and higher doses than those recommended.

Untoward reactions are mostly mild and transient, although permanent visual field defects and deaths have been reported.

Vascular reactions: flushing, vessel spasm, thrombophlebitis, and cutaneous petechiae.

Neurologic complications: transient cerebral blindness, neuromuscular disorders, convulsions, coma, hemiparesis, unilateral dysesthesias, visual field defects, aphasia, and respiratory difficulties.

AORTOGRAPHY

Precautions *(See also General Precautions):* Special caution is advised to avoid inadvertent intrathecal injection when using the translumbar technique. The incidence and severity of reactions or complications that may occur are influenced by: the care and experience with which the procedure is performed; the amount and type of medium used; the age and condition of the patient; and the premedication and anesthesia used. Since aortography is not without some danger, it should be performed only by those experienced in the technique. Wherever possible, repeated injections of the solution during a single study should be avoided.

Adverse Reactions *(See also General Adverse Reactions):* Renal damage and shutdown; neurologic complications such as transverse myelitis or paraplegia; cardiovascular complications such as shock, cardiac arrest or failure, coronary occlusion, arterial thrombosis, embolism, and perforation of vessels; extravasation or hemorrhage from the puncture site or retroperitoneal catheter perforation; necrosis of the intestinal wall; acute pancreatitis; deaths; diffuse cutaneous petechiae; subintimal injection and aortic dissection.

INTRAOSSEOUS VENOGRAPHY

See General Precautions and Adverse Reactions. A general anesthetic is sometimes necessary since the method is painful. Occasionally, extravasation of the contrast medium from the needle into the soft tissue may occur.

DIRECT CHOLANGIOGRAPHY

Precaution *(See also General Precautions):* This procedure should be used with caution in the presence of acute pancreatitis, injecting no more than 5 to 10 ml. without undue pressure.

Adverse Reactions *(See also General Adverse Reactions):* These may be attributed to injection pressure or excessive volume of the medium, resulting in overdistention. Such pressure may produce a sensation of epigastric fullness, followed by moderate pain in the back or right upper abdominal quadrant, which will subside when injection is stopped.

Some of the medium may enter the pancreatic duct and cause a transient serum amylase elevation 6 to 18 hours later, without apparent ill effects. Pancholangitis resulting in liver abscess has been reported. Occasionally, nausea, vomiting, fever, and tachycardia have been observed.

HYSTEROSALPINGOGRAPHY

Contraindications: Menstruation, active or imminent; infection in any part of the genital tract, including the external genitalia; pregnancy, known or suspected. Use not advised for six months after end of pregnancy, or 30 days after conization or curettement.

Precaution *(See also General Precautions):* Caution should be exercised in patients with known or suspected carcinoma to avoid possible spread of the lesion by the procedure.

SPLENOPORTOGRAPHY

Precautions *(See also General Precautions):* Procedure should be performed with caution on patients with blood dyscrasias, a tendency to severe bleeding, or a spleen recently become tender and palpable.

Adverse Reactions *(See also General Adverse Reactions):* Persistent hemorrhage with hemoperitoneum has been reported, and, in some instances, hemorrhage has required splenectomy.

How Supplied: Ampuls of 30 ml., boxes of 10 and 25. Boxes of 25—1 ml. ampuls for testing sensitivity are available upon request. Vials of 20 and 30 ml., rubber stoppered, boxes of 10 and 25. Vials of 50 ml., rubber stoppered, boxes of 10. Each vial contains sufficient excess to permit withdrawal of 1 ml. for testing sensitivity.

If the solution is chilled, crystals may form but will readily dissolve if the ampul or vial is placed in moderately hot water before use. Cool to body temperature before injecting.

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TABLE II

ROENTGENOGRAPHIC AND PATHOLOGIC FINDINGS

Authors	Roentgenographic Findings	Pathologic Findings
Kelly and MacCallum[14]		Pus and gas
Randall[26]	Gas in the renal parenchyma	
Mathe and De la Pena[20]	Gas was demonstrated in the kidney	Incomplete double collecting system; upper pole of kidney closed off by ureteral stricture and infected with colon bacillus
Nogueira[22]	—	—
Olsson[24]	IVP—no function; gas in the kidney capsule, parenchyma and pelvis	Abscesses extending into the cortex, chronic pyelonephritis, papillary necrosis
Jensen[13]	—	—
Alexander[1]	Day 2—IVP: no function; air pyelogram with multiple air pockets in cortex; Day 4—entire kidney surrounded by gas; retrograde pyelography resulted in extravasation from pelvis	Gas within Gerota's fascia, diffuse extensive renal necrosis, chronic pyelonephritis
Gillies and Flocks[10]	3 Weeks—IVP: functioned; small bubbles of gas distributed radially within the kidney; next day gas in perirenal tissue	
Harrison and Bailey[11]	IVP—no function; gas bubbles in the kidney and perirenal area	Little renal tissue, multiple abscesses and intrarenal vascular thromboses
Plaggemeyer *et al.*[25]		
Welch and Prather[31]	Day 3—retrograde pyelogram showed entire left kidney outlined by gas with interstitial backflow of contrast medium	Gas distending capsule, diffuse necrosis with little renal tissue
Stokes[30]	Small intrarenal air abscess; Hours 16—later air throughout retroperitoneum; retrograde extravasation	Entire kidney destroyed
Levy and Schwinger[18]	Day 1—little gas in right upper quadrant; IVP: no function; Hour 16—kidney and capsule outlined; Day 2—retroperitoneal gas	Extensive necrosis, large upper pole abscess extending retroperitoneally, upper pole infarct
Braman and Cross[6]	Day 3—IVP: no function; gas in kidney and retroperitoneum, increasing to Day 10 when retrograde pyelography resulted in extravasation	Incision and drainage: rush of air from capsule, crepitant kidney

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RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 110 SEPTEMBER, 1970 NO. 1

ANGIOGRAPHIC FEATURES OF BRAIN STEM TUMORS AND DIFFERENTIAL DIAGNOSIS FROM FOURTH VENTRICLE TUMORS*

By YUN PENG HUANG, M.D., *and* BERNARD S. WOLF, M.D.
NEW YORK, NEW YORK

ANGIOGRAPHIC criteria for the diagnosis of tumors of the fourth ventricle have been presented in a previous publication.[19] This paper records observations of angiographic features of tumors of the brain stem. Since both brain stem and fourth ventricle tumors are located in the midline, it is not surprising that they share several angiographic features. In the majority of cases, however, there are sufficient differences to permit a correct diagnosis. This distinction is of importance, since operative intervention in brain stem tumors is neither feasible nor advisable but is often palliative and may be curative in fourth ventricle tumors.

The usefulness of angiography in posterior fossa lesions has increased recently with utilization of bilateral retrograde brachial angiography, catheter, stereoscopic and subtraction techniques. As a result, posterior fossa angiography is used more frequently as the first neuroradiologic special procedure. A limited, safer air study, tailored to a specific problem, may then be done if necessary.

This report is based on an analysis of 23 cases of brain stem space-occupying lesions in which satisfactory visualization of posterior fossa vessels was achieved and in which the diagnosis was confirmed by subsequent encephalography, ventriculography or autopsy.

PATHOLOGIC FEATURES

The most common cause of enlargement of the brain stem is neoplasm, usually an astrocytoma, less frequently a glioblastoma multiforme, oligodendroglioma or metastasis. By the time the patient appears for examination, a brain stem glioma has usually involved most of the pons with variable extensions into the cerebellum, midbrain and sub- and hypothalamic regions. Marked downward extension into the medulla oblongata is usually not seen until late, although occasionally a tumor may originate in the medulla oblongata.[24] In-

* Presented at the Seventh Annual Meeting of the American Society of Neuroradiology, Cleveland, Ohio, April 18, 1969.
From the Department of Radiology, the Mount Sinai Hospital and the Mount Sinai School of Medicine, New York, New York.
Partly supported by a research grant from the National Heart Institute, National Institute of Health, U. S. Public Health Service 5 R01 HE09913-04.

TABLE II (*Continued*)

Authors	Roentgenographic Findings	Pathologic Findings
Olson[23]	1. IVP: no function; numerous radial streaks of gas through the parenchyma and in the capsule	Incision and drainage: multiple abscesses and gas
	2. Retrograde pyelogram consistent with pyelonephritis	Chronic pyelonephritis and necrotizing papillitis
McLelland[21]	IVP: function; progressive accumulation of intrarenal gas	Pyelonephritis and pyelitis with ureteritis
Schultz and Klorfein[28]	IVP: no function; kidney replaced by gas; retrograde pyelogram: blunt calyces	
Harrow and Sloane[12]	IVP: no function; mottled gas in interstitium and perirenal areas confined by Gerota's fascia; retrograde pyelography resulted in intrarenal extravasation	Severe acute and chronic granulomatous reaction
Yu[32]	IVP: no function; radiating lines of gas in kidney; outline of Gerota's fascia; Day 2—gas retroperitoneally and into thorax	Entire kidney replaced by inflammatory tissue
Klein *et al.*[15]	1. IVP: no function; stippled gas within kidney; retrograde pyelography, extravasation	Friable gas filled cysts, ischemic infarction
	2. Mottled gas in kidney and smooth gas in fascia	—
Schainuck *et al.*[27]	Day 1—mottled gas in kidney; Day 2—IVP: no function; Day 10—gas dissecting into Gerota's fascia; Day 34—arteriograms and tomograms confirmed multiple abscesses	Multiple gas filled abscesses, several perforating the capsule; thrombosis of small intrarenal arteries
Banks *et al.*[3]	1. Day 1—mottled intrarenal gas and gas within Gerota's fascia outlining upper pole	Acute and chronic pyelonephritis, abscess in upper pole
	2. Day 1—mottled gas throughout kidney and within Gerota's fascia, retrograde pyelography, extravasation	Septic infarcts and abscesses, pyelonephritis and papillary necrosis
	3. Day 1—mottled gas throughout kidney and within Gerota's fascia, retrograde pyelography, extravasation	Massive septic infarction of kidney, thrombosis of the renal artery
	4. Day 1—mottled gas throughout kidney and within Gerota's fascia, retrograde pyelography normal	Acute and chronic pyelonephritis, necrotizing papillitis and infarcted upper pole
Langston and Pfister	Day 2—IVP and nephrotomograms: no function; mottled gas throughout parenchyma and perirenal space contained by Gerota's fascia	Kidney almost totally destroyed by confluent abscesses extending into the perinephric space and perirenal fat and down the ureter; no ureteral obstruction

IVP = intravenous pyelogram.

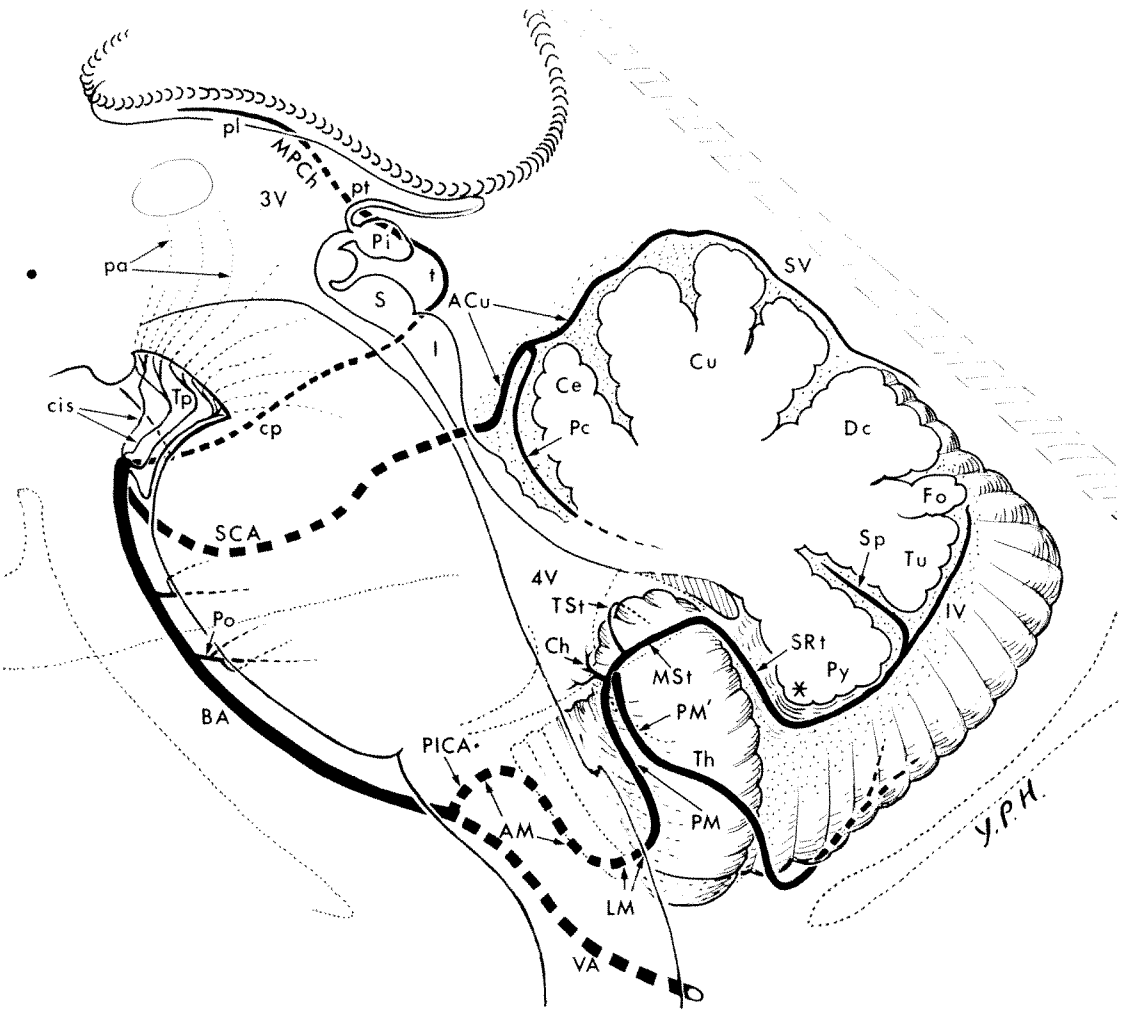


FIG. 1. Diagrammatic representation of "mid-sagittal section" showing the normal course of diagnostically important posterior fossa arteries. The anterior portion of the inferior vermis has been removed to expose the medial aspect of the tonsil. Covered portions of arteries and parenchymal twigs are shown in interrupted fashion.

Labeled structures include the anterior culminate segment (ACu), the anterior medullary segment (AM), basilar artery (BA), central lobule (Ce), choroidal artery (Ch), cisternal segment (cis), circumpeduncular segment (cp), culmen (Cu), declive (Dc), inferior colliculus (I), vermian segment (IV) of the posterior inferior cerebellar artery, the folium (Fo), lateral medullary segment (LM), medial posterior choroidal artery (MPCh), parenchymal segment (pa), precentral cerebellar artery (Pc), pineal gland (Pi), posterior inferior cerebellar artery (PICA), plexal segment (pl), posterior medullary segments (PM, PM') of the posterior inferior cerebellar artery and its tonsillo-hemispheric branch, pontine arteries (Po), pretectal segment (pt), pyramid (Py), superior colliculus (S), superior cerebellar artery (SCA), suprapyramidal branch (SP), superior retrotonsillar segment (SRt), medial supratonsillar segment (MSt), and vermian segment (SV) of the superior cerebellar artery, tectal segment (t), tonsillo-hemispheric branch (Th), transverse supratonsillar branch (TSt), thalamo-perforating arteries (Tp), tuber (Tu), vertebral artery (VA). The copula pyramidis (*), third and fourth ventricles (3V, 4V) are also labeled.

The basilar artery may be located anywhere within the pontine cistern, that is, not intimately applied to the brain stem. Small pontine branches (Po), however, form a network directly on the surface of the pons. The thalamo-perforating arteries (Tp) arise in the deep portion of the interpeduncular cistern from the posterior cerebral arteries and assist in outlining the configuration of this cistern. They may be divided into cisternal (cis) and parenchymal portions (pa).[10] The prepontine and lateral pontine segments of the superior cerebellar artery (SCA) usually show an arcuate course convex inferiorly. The origin of the pre-

cent of those cases where the information was available.

The treatment of this disease prior to the era of effective antibiotics was surgical. In the 1950s the frequency of unobstructed renal emphysema was noted and several authors recommended medical therapy.[28,30] More recently, however, the need for surgical intervention has been stressed by Banks *et al.*[3] in their report of 4 cases.

The results of medical or surgical treatment of this illness are poor, with an overall mortality of 55 per cent and 66 per cent, respectively, of the cases reported after 1940. The results are slightly better in the group treated with both nephrectomy or incision and drainage and intensive medical therapy, in which after 1940 the survival was 50 per cent. As the pathologic specimens, when reported, uniformly demonstrated a virtually destroyed kidney reduced to a mass of confluent abscesses, the rationale for surgery is seen. One would imagine that there would be a need for delay to treat uncontrolled diabetes and to control the infection, but in fact the majority of patients were not in severe acidosis, nor is there any evidence from the limited data that antibiotic control can be achieved.

Why the infection gets out of control is not yet understood. Possibly the elevated blood sugar plays some role, as may obstruction when present. The increasing incidence of drug-resistant *coliforms* may be a significant factor. Four authors[3,11,18,27] noted focal renal infarction in the destroyed kidneys, one of which had a thrombosed renal artery. We did not find this in our case. The one case in which aortography was performed showed no major vessel obstruction.[27]

ROENTGENOGRAPHIC FINDINGS

Gas in the urinary tract may be due to previous instrumentation or surgery, fistulae between the genitourinary tract and the gastrointestinal or lower genital tracts, or to relatively benign *coliform* infections limited to the bladder. However, since the spontaneous generation of gas within the renal parenchyma implies rapidly spreading infection and often leads to a fatal gram-negative septicemia, it is important to be aware of the early roentgenologic signs. These may be very difficult to detect because of frequently associated intestinal ileus.

In 7 of the cases reviewed,[1,10,15,18,23,27,30] the earliest sign was a diffuse mottling of renal parenchyma in which the gas bubbles were noted by 2 authors[10,23] to be arranged radially as though along the pyramids. This radial distribution was later noticed in more advanced cases in which the gas had extended to Gerota's fascia.[12,23,32] In the remainder of the series the distribution was not commented upon or the kidney was almost totally involved at the time of the initial roentgenologic examination.

Review of the roentgenograms published in the literature (Table II) suggests that the picture of a bubbly renal parenchyma surrounded by a crescent of gas within Gerota's fascia, which was noted in our patient, is typical of the advanced stage of renal necrosis.[1,3,12,15,18,23,24,28,32] In several instances further extension was noted when Gerota's fascia was eroded through and the gas was seen roentgenographically to extend throughout the retroperitoneum[18,30,31] and even into the posterior thoracic wall.[32] The left kidney was involved more often than the right. As can be seen from Table II, the pathologic findings in our patient are typical.

When the infection has progressed to an end stage of confluent gas-filled abscesses, the pathologists have given us a dramatic correlation of the roentgenologic findings: The kidney was purple and gray and it gave the impression of lung tissue.[1] At the time of incision there was a rush of air, revealing a crepitant kidney on a friable pedicle.[6]

CONCLUSION

The question arises as to what procedures should be undertaken to further evaluate

volvement of the brain stem is usually symmetric. Extension into adjacent structures is, however, not infrequently unilateral. As well shown by Russell and Rubinstein,[24] Dandy,[3] and Courville,[2] there often is "nodular hypertrophy" of the surface of the enlarged pons with tumor masses fungating forward in between the basilar artery and its major branches. Under these circumstances, the surface of the pons is "deeply grooved" by the basilar artery and the proximal portions of its major branches. As lobulated masses bulge from the anterior surface of the enlarged pons, they encroach on the pontine cistern. After reaching the clivus, further growth displaces the enlarged pons backward carrying the basilar artery with it. Paradoxical displacements of this type simulating extra-axial masses may also be observed in relation to the midbrain or medulla, although less frequently.

With development of brain stem tumor, the brain stem becomes enlarged longitudinally, transversely and anteroposteriorly. The floor of the fourth ventricle is displaced backward, flattened from in front, widened transversely and elongated longitudinally. The body of the cerebellar tonsil is compressed and displaced backward. When the bulk of the lesion is located in the pons, the upper pole of the cerebellar tonsil is more posteriorly located than its inferior pole. When the tumor originates in the medulla, the reverse may occur. Extension of the tumor into the brachium pontis and the restiform body produces marked increase in the size of these structures with downward arcuate displacement of the lateral recess of the fourth ventricle and the adjacent posterolateral fissure. With further extension into the region of the dentate nucleus, the lateral wall of the fourth ventricle may be displaced medially. When the cerebellar tonsil is involved by tumor, the anteroposterior diameter of the cerebellar tonsil is not decreased but remains normal or may be increased. The degree of internal hydrocephalus in brain stem tumors is not marked until late.

ANGIOGRAPHIC FEATURES OF BRAIN STEM TUMORS

There have been several reports[4,23] describing changes in the course of the posterior inferior cerebellar artery in brain stem tumors. These changes are of great utility, particularly when displacements of specific segments of this vessel are analyzed in relation to the anatomic structures. Many other vessels, arteries and veins, show changes as well. For ease of description, displacements in the arterial and venous phases will be considered separately.

---

central cerebellar artery (Pc) is variable, but often demonstrates a recurrent course in order to enter the precentral cerebellar fissure. The fissural and parenchymal portions of this vessel are located behind and parallel to the brachium conjunctivum and the upper portion of the roof of the fourth ventricle. The vermian branch (SV) of the superior cerebellar artery (SCA) outlines the apex and tentorial aspect of the superior vermis.

The anterior medullary segment (AM) and the lateral medullary segment (LM) of the posterior inferior cerebellar artery (PICA) vary in their course depending on the site of origin of this vessel. However, the posterior medullary segment (PM) of the artery rather consistently runs adjacent to and behind the floor of the lower portion of the fourth ventricle (See also A_2 in Fig. 3). The "choroidal point" or junction between the posterior medullary (PM) and the supratonsillar segments (St) is frequently at the site of origin of a major choroidal artery (Ch) of the fourth ventricle. The exact location of the supratonsillar segment in relationship to the superior pole of the tonsil is variable. The superior retrotonsillar segment (SRt) outlines the upper half of the posterior margin of the cerebellar tonsil. The asterisk (*) indicates the copula pyramidis around which the vermian segment (IV) of the posterior inferior cerebellar artery runs. The origin and course of the tonsillo-hemispheric branch is quite variable. In this diagram, it is shown running a short distance behind the posterior medullary segment (PM') of the posterior inferior cerebellar artery but still in the cerebello-medullary fissure on the medullary aspect of the tonsil.

the possibility of renal emphysema when the diagnosis is uncertain. The frequent rapid progression of the early roentgenographic signs may obviate further evaluation unless it is undertaken immediately.

Probably infusion nephrotomography is the most useful method, since it will eliminate overlying intestinal gas patterns. The involved kidney is unlikely to function (only 2 of 13 in which an intravenous pyelogram was obtained, opacified), but as a nephrectomy will almost certainly be considered, evaluation of the contralateral kidney is needed.

Retrograde pyelography has frequently been done both to rule out obstruction and to aid in delineating the renal parenchyma. In 7 out of 11 cases this resulted in intrarenal or extrarenal extravasation of the contrast medium and should be undertaken with caution.

SUMMARY

Review of the previously reported 27 cases and our own case of renal emphysema emphasizes that this is a roentgenologic diagnosis of a medical-surgical emergency which at the present time still carries a 55 per cent mortality.

Typically, the patient is a diabetic female past middle age, in good metabolic control, sometimes with a previous history of *coliform* urinary tract infections. The illness is acute, usually less than 3 days in duration at the time of diagnosis, and the patient frequently presents with gram-negative sepsis.

The earliest roentgenographic signs are a diffuse mottling of the renal parenchyma by hydrogen and carbon dioxide filled abscesses which are often radially distributed. As the infection spreads through the cortex, the gas extends beyond the renal capsule and is contained by Gerota's fascia, producing a crescentic margin or complete outlining of the kidney. Occasionally the infection and gas spread from the perirenal fascia into the retroperitoneal space.

The best method for evaluating the uncertain case seems to be nephrotomography which should be performed as soon as possible so that immediate surgical and medical treatment may be started.

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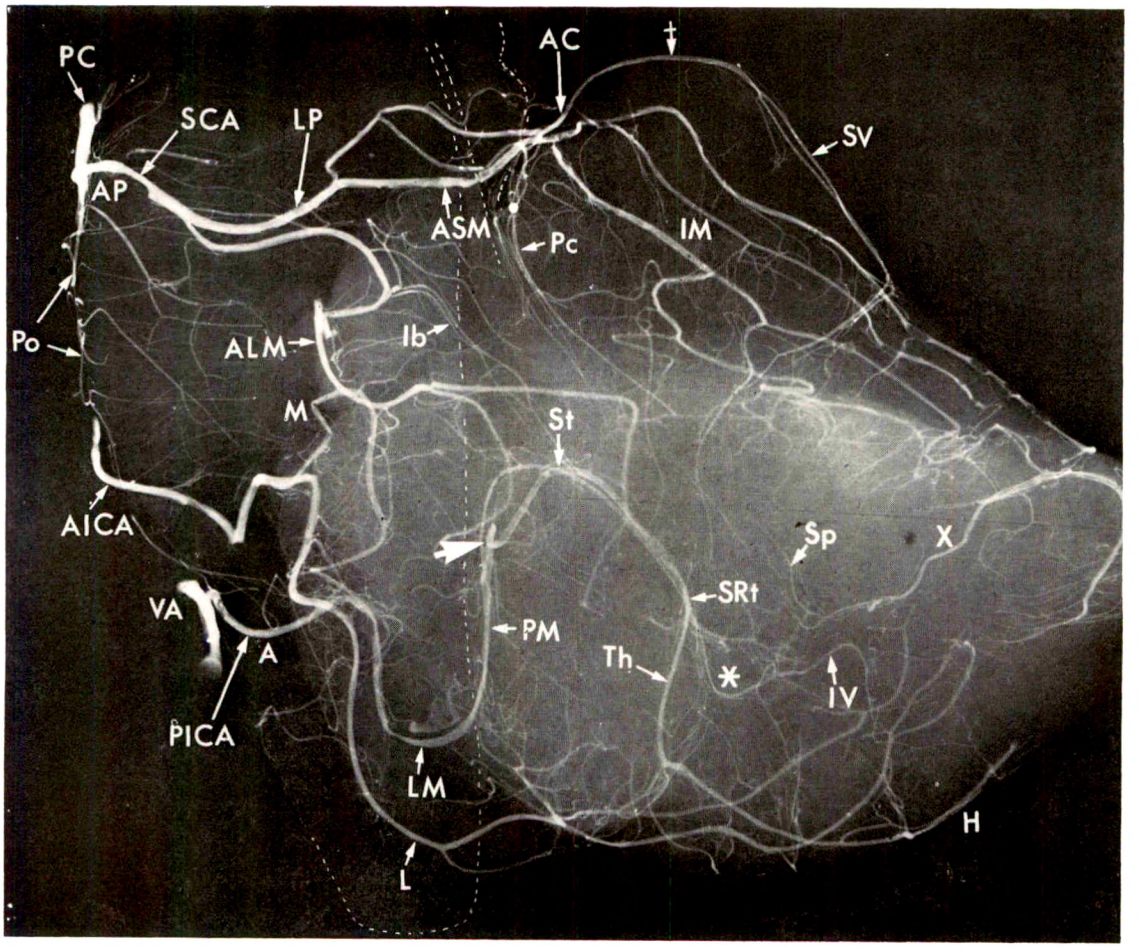


FIG. 2. Roentgenogram of an injected specimen showing the course of the arteries of the posterior fossa in the lateral view. The colliculi, the medulla, aqueduct and floor of the fourth ventricle are retouched.

The labeled structures include the anterior culminate segment (AC), anterior inferior cerebellar artery (AICA), the anterior lateral marginal branch (ALM), anterior medullary segment (A), anterior pontine or prepontine segment (AP), anterior superior marginal segment (ASM), hemispheric branch (H), interbrachial branch (Ib), intermediate segment (IM), vermian segment (IV) of the posterior inferior cerebellar artery, lateral branch (L) of the anterior inferior cerebellar artery, lateral medullary segment (LM), lateral pontine segment (LP), medial branch (M) of the anterior inferior cerebellar artery, precentral cerebellar branch (Pc), the proximal portion of the posterior cerebral artery (PC), posterior inferior cerebellar artery (PICA), pontine branches (Po), posterior medullary segment (PM), superior cerebellar artery (SCA), suprapyramidal branch (Sp), superior retrotonsillar segment (SRt), supratonsillar segment (St), vermian segment (SV) of the superior cerebellar artery, tonsillo-hemispheric branch (Th), and the vertebral artery (VA). The large arrow (↑) indicates the choroidal point at the distal end of the posterior medullary segment (PM). The asterisk (*) indicates the location of the copula pyramidis. The apex of the cerebellum (‡) is also indicated.

The precentral cerebellar branch (Pc) of the superior cerebellar artery outlines the roof of the upper part of the fourth ventricle. The white dot in the course of the precentral cerebellar artery indicates the approximate point at which the artery enters the precentral cerebellar fissure. The posterior medullary segment (PM) of the posterior inferior cerebellar artery (PICA) corresponds to the posterior aspect of the lower brain stem. The distance between the superior retrotonsillar segment (SRt) and the posterior medullary segment (PM) indicates the height of the fourth ventricle and is an indication of the anteroposterior diameter of the body of the cerebellar tonsil. In this specimen, the suprapyramidal branch (Sp) originates from the vermian segment of the opposite side (X).

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TOTAL HIP REPLACEMENT*

ROENTGEN APPEARANCE OF CURRENT DEVICES

By FRANKLIN L. ANGELL, M.D., *and* FREDERICK B. WATTS, JR., M.D.
BOSTON, MASSACHUSETTS

MULTIPLE devices are now used to totally replace the hip joint. The expansion of knowledge in the fields of biomechanics and metallurgy, coupled with the development of plastics and "cold curing" acrylic bonding material has resulted in the construction of appliances that are biologically tolerated and more structurally and functionally sound than those previously used.[2,3,6,7]

Characteristic postoperative changes are associated with the insertion of each device, and lack of familiarity with the appearance of the devices employing methylmethachorlate as a bonding agent may make interpretation of the postoperative changes difficult. The need for the radiologist to be aware of the roentgen appearance of the currently used devices is apparent.

The surgical indications for the procedure and the factors influencing the choice of a particular prosthesis are beyond the scope of this report. Such information is available elsewhere.

The McKee-Farrar,[8,9] Charnley-Mueller,[4] and Ring[6,10] prostheses as well as the vitallium mold[1,5] are used currently in total hip replacement at the Massachusetts General Hospital. In special circumstances, individually designed prostheses have been constructed for those patients with extraordinary problems. A description of these prostheses and of their characteristic roentgen appearance is presented.

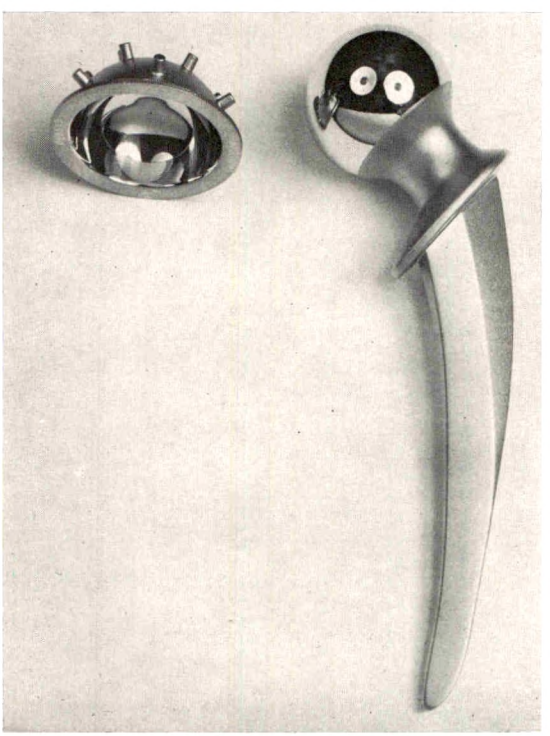


FIG. 1. McKee-Farrar prosthesis. Matched cup and modified Thompson prosthesis.

THE MCKEE-FARRAR PROSTHESIS

(Metal Head to Metal Cup)

Matched cup and modified Thompson prosthesis (Fig. 1).

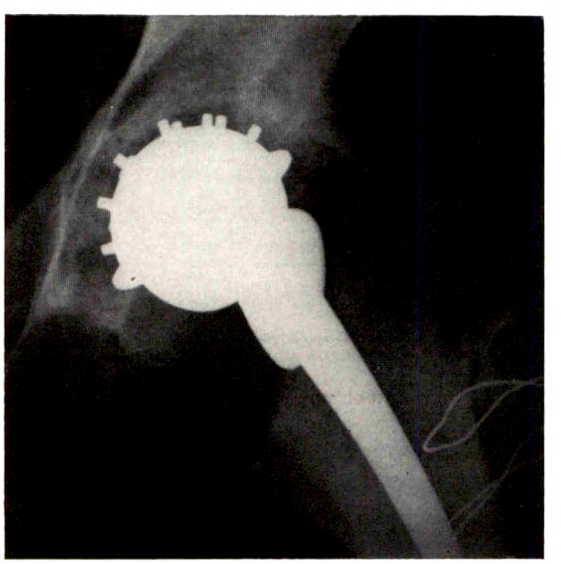


FIG. 2. Roentgenogram of the hip showing the McKee-Farrar prosthesis in place.

* From the Departments of Radiology, Harvard Medical School and Massachusetts General Hospital, Boston, Massachusetts.



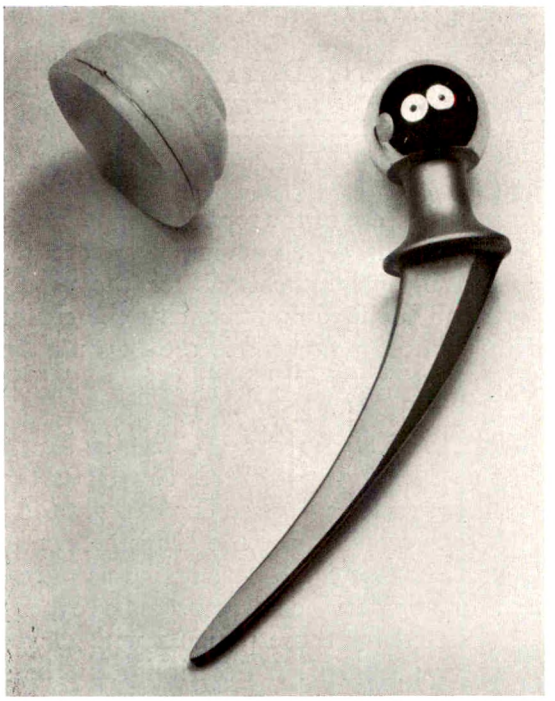


Fig. 3. Charnley-Mueller prosthesis. Teflon cup is matched to the modified Thompson prosthesis. Note the wire around the perimeter of the cup.

Pelvic Component. The cup is made with many small studs extending at right angles from a curved surface. This design facilitates firm fixation at the acetabular site.

Femoral Component. A modified Thompson prosthesis is matched to the acetabular cup.

The remnants of articular cartilage are removed from the acetabulum leaving the walls rough. Soft tissue is removed from the fovea. A cavity is made in the ischium. This is directed towards the ischial spine. Multiple holes are drilled into the superior and superomedial aspect of the acetabulum. Penetration of the inner cortex of the ilium frequently occurs. Additional holes are drilled wherever needed. The outer cortex of the ilium above the acetabulum is debrided and 2 screws are inserted parallel to the roof of the acetabulum. They penetrate both the outer and inner cortex of the ilium. The acrylic cement binds the acetabular cup and some of the mix flows above the ilium where it comes in contact with the 2 screws. This produces additional fixation of the acetabular prosthesis. Then after curetting the femoral neck and proximal shaft, the femoral component is inserted into the medullary cavity. It is also held in place by plastic cement.

A roentgenogram of the hip with McKee-Farrar prosthesis in place shows the deepened acetabulum and the drilling sites in the ischium (Fig. 2).

THE CHARNLEY-MUELLER PROSTHESIS

(Metal Head to Teflon Cup)

The teflon cup is matched to the modified Thompson prosthesis. Note the wire around the perimeter of the cup (Fig. 3).

Pelvic Component. A high density polyethylene cup containing concentric circular grooves is placed around its outer surface. These grooves are parallel to the rim. A radiopaque wire encircles the cup near the inner margin of the rim.

Femoral Component. A modified Thompson prosthesis is matched individually to each polyethylene acetabular cup.

The acetabulum is deeply reamed with penetration of the ischial cortex. Multiple cavities are created in the ilium. These serve as binding sites for the acrylic cement and anchor the cup to the pelvis. The cup is directed slightly laterally and anteriorly in the normal plane of the acetabulum. The femoral component is inserted into the femur. The greater trochanter is transfixed onto the proximal femoral shaft in an attempt to preserve muscular stability at the hip.

Roentgenograms of the hip with Charnley-Mueller prosthesis in place are demonstrated in Figure 4, *A–C*.

THE RING PROSTHESIS

(Metal Head to Metal Cup)

The metal cup is matched to a modified Moore prosthesis (Fig. 5).

Pelvic Component. This consists of a cup with 4 grooves on its outer edge and a stem.

Femoral Component. A modified Moore

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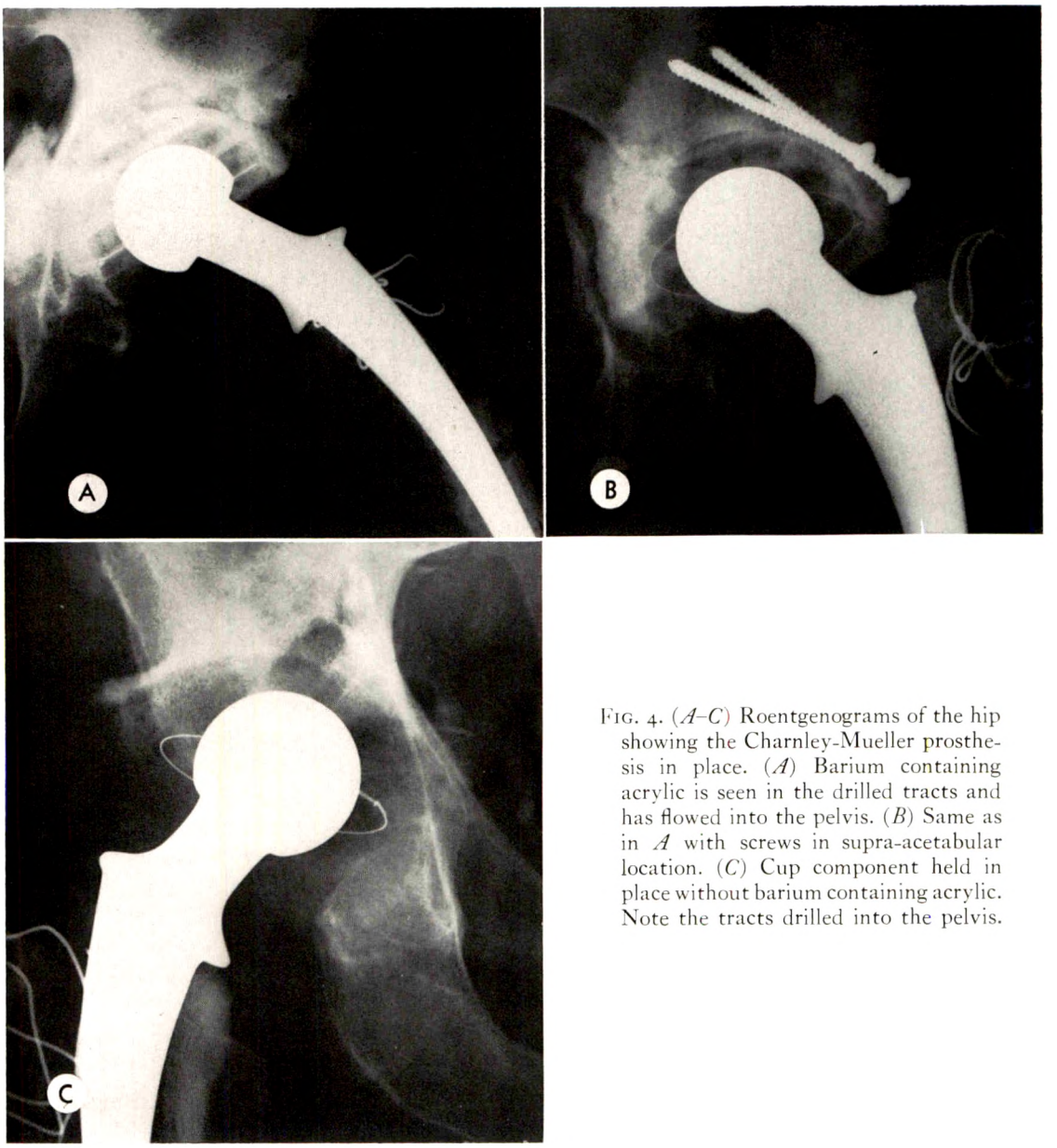


Fig. 4. (*A–C*) Roentgenograms of the hip showing the Charnley-Mueller prosthesis in place. (*A*) Barium containing acrylic is seen in the drilled tracts and has flowed into the pelvis. (*B*) Same as in *A* with screws in supra-acetabular location. (*C*) Cup component held in place without barium containing acrylic. Note the tracts drilled into the pelvis.

prosthesis which is matched to the acetabular cup constitutes the femoral component.

The acetabular prosthesis is introduced into the ilium. The stem is directed through the bar of bone which extends from the upper margin of the acetabulum to the sacroiliac joint. The axis of the coupled acetabular and femoral components is anterior and slightly lateral.

A roentgenogram demonstrating the Ring prosthesis in place is shown in Figure 6. The stem is directed towards the sacrum just above the sacral notch.

VITALLIUM MOLD
(Metal to Tissue)

A vitallium mold is illustrated in Figure 7.

Pelvic and Femoral Components. The outer surface of the mold is convex and the inner surface is concave.

The mold is placed as deeply as possible

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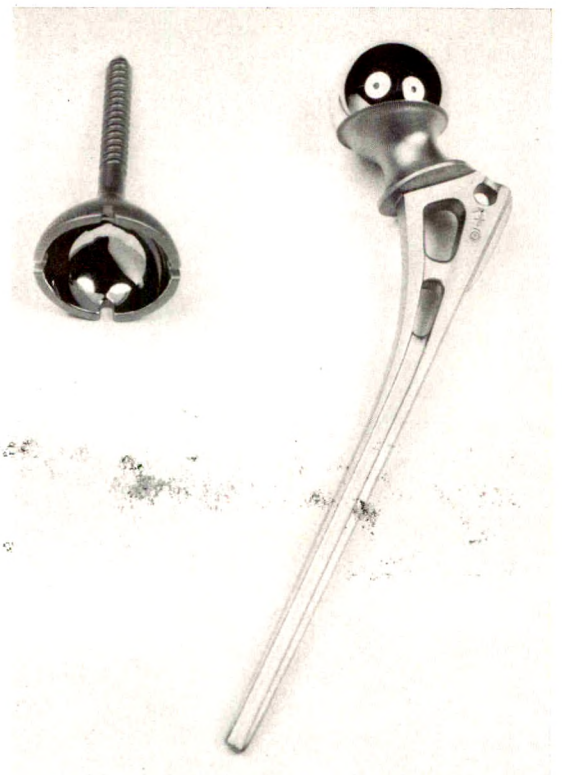


Fig. 5. Ring prosthesis. The metal cup is matched to a modified Moore prosthesis.

into the acetabular cavity. The margins of the cup project beyond the acetabular rim. The reshaped femoral head, neck, or shaft is directed into the mold. This produces 4 surfaces that glide over one another. The mold is not otherwise fixed and stability at the hip is produced by rearrangement of the muscle attachments.

A roentgenogram of a vitallium mold seated deep in the acetabulum is shown in Figure 8. The attachments on the trochanter have been revised.

SPECIAL DEVICES

Appearance is determined by the functional requirements of the prosthesis and its manner of attachment. No preinsertion photographs were available.

A hemipelvectomy prosthesis, devised by Thomas L. Delorme, M.D., was inserted following removal of an invasive chondrosarcoma of the pubis (Fig. 9).

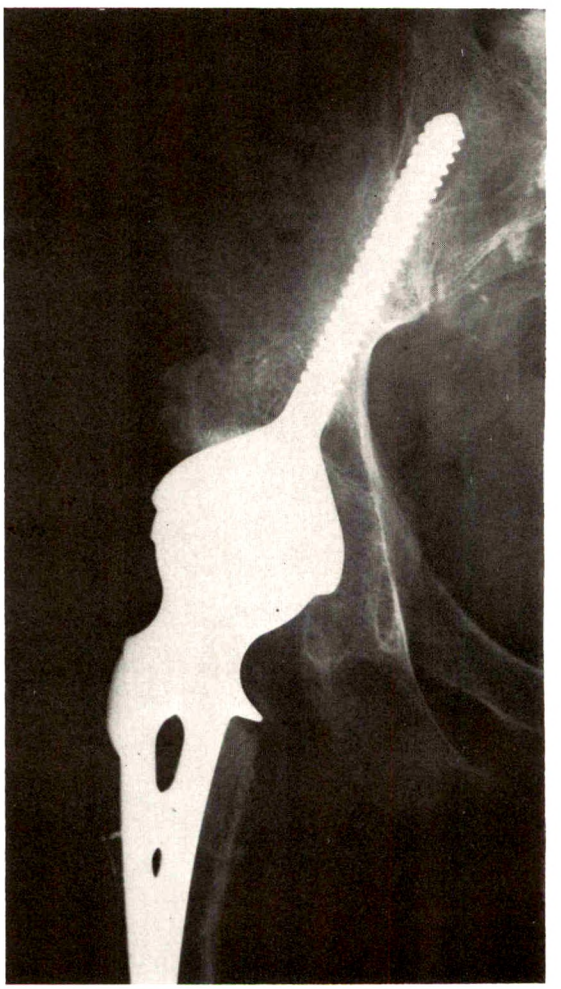


Fig. 6. Roentgenogram of the hip showing the Ring prosthesis in place.

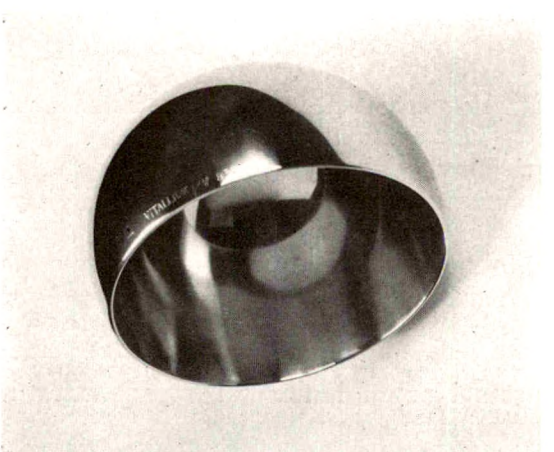


Fig. 7. Vitallium mold.

In any particular case, the findings in one or the other phase may be more striking but all features must be integrated into a consistent pattern.

A. ARTERIOGRAPHIC FEATURES

Anterior displacements of the basilar artery and the distal portion of the vertebral arteries are well known features of brain stem tumors. However, these changes are not specific since any mass lesion posterior to these vessels may have the same effect. Moreover, as pointed out above, the basilar artery is often not displaced as far forward by a brain stem tumor as might be anticipated. It may in fact be displaced posteriorly as seen with prepontine space-occupying lesions. It is therefore necessary to give maximum attention to other arteries—their segmental divisions as well as branches (Fig. 1; and 2). These include: (1) the various segments of the superior cerebellar arteries[5,17,20,22] (the prepontine, lateral pontine, anterior superior marginal or juxtategmental, anterior culminate and vermian segments); (2) the branches of the superior cerebellar arteries (the precentral cerebellar, anterior lateral marginal, intermediate and superior vermian branches); (3) the segments of the posterior inferior cerebellar arteries[9,12,19,30] (the anterior medullary, lateral medullary, supratonsillar, superior retrotonsillar and vermian segments); (4) the branches of the posterior inferior cerebellar arteries (the tonsillo-hemispheric and suprapyramidal branches); (5) the transversely running pontine arteries;[6] (6) the anterior inferior cerebellar arteries;[1,27] (7) the posterior cerebral arteries; (8) the thalamo-perforating arteries;[10,28] and (9) the medial and lateral posterior choroidal arteries.[7]

When the upper brain stem is grossly involved (Fig. 3; 4; 5; 6; and 7) the *superior cerebellar artery*[17] is stretched, rounded and displaced outward. The prepontine segment of the artery is displaced forward. The usual downward dipping of the artery as it extends posteriorly around the brain stem is diminished, lost, or occasionally reversed. The anterior culminate segment of this artery and, to a lesser degree, the superior vermian segment are displaced backward. The latter segment comes closer to the straight sinus, and the angle formed by these segments of the artery over the apex of the cerebellum becomes more acute. The *precentral cerebellar artery*,[17] although more variable than the precentral cerebellar vein,[13] is particularly helpful since it runs between the brain stem and the cerebellum. Usually, this vessel arises either from the anterior superior marginal segment or anterior culminate segment of the superior cerebellar artery and runs backward or forward to enter the fissure between the central lobule and lingula, *i.e.*, the precentral cerebellar fissure. In the depth of this fissure, on either side of the vermis, it penetrates the parenchyma of the cerebellum to run with the brachium conjunctivum (Fig. 2). The artery may be said to have 3 segments: cisternal, fissural and parenchymal segments. With an extensive brain stem tumor, the fissural and parenchymal segments of the precentral cerebellar artery are displaced backward but maintain a smooth oblique course roughly parallel to the clivus (Fig. 4; 5*A*; and 6*A*). The distance between this artery and the basilar artery—the width of the brain stem—is increased. This distance corresponds to the previously described measurement between the colliculo-central point[13] of the precentral cerebellar vein and the anterior aspect of the pons (outlined by the basilar artery or pontine vein), a mean of 3.2 cm. and a range of 2.9 to 3.6 cm. The distance between the colliculo-central point or the most anterior point of the precentral cerebellar artery and the clivus has a mean value of 3.9 cm. and a range of 3.6 to 4.3 cm. As a result of posterior displacement of the precentral cerebellar artery, the distance between this artery and the vermian segment of the superior cerebellar artery is diminished (Fig. 5*A*; and 8*A*).

With gross involvement of the lower brain stem, displacement of various segments of the *posterior inferior cerebellar ar-*

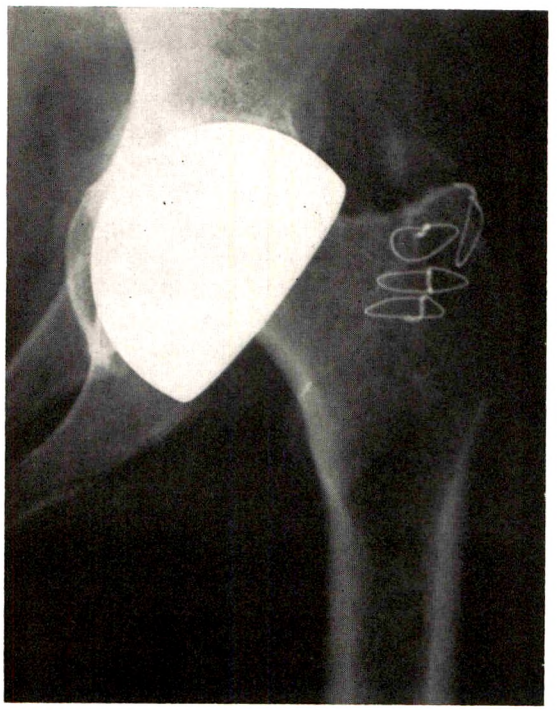


Fig. 8. Roentgenogram of the hip showing the vitallium mold seated deep in the acetabulum.

Pelvic Component. This specially designed replacement prosthesis was constructed for the left ilium and pubis which is attached to the sacrum, contralateral ilium and pubis. The acetabular cup is incorporated in the prosthetic architecture.

Femoral Component. A modified Moore prosthesis with a long intramedullary stem extending into the femoral shaft above the supracondylar portion formed the femoral component.

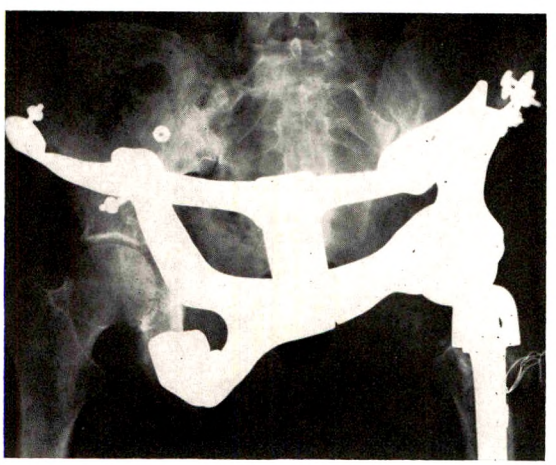


Fig. 9. Hemipelvectomy prosthesis, devised by Thomas L. Delorme, M.D.

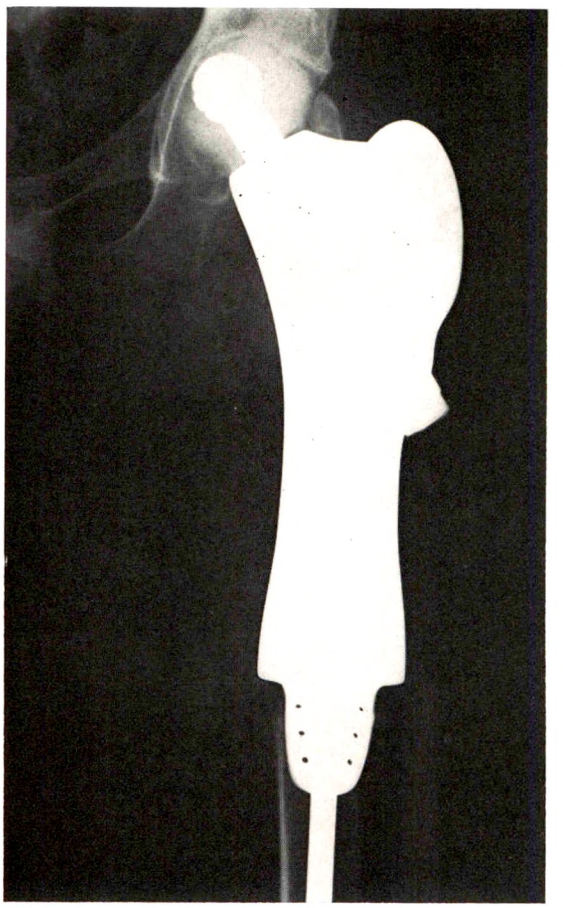


Fig. 10. Proximal femoral (replacement) prosthesis, devised by Robert J. Boyd, M.D.

A proximal femoral (replacement) prosthesis, devised by Robert J. Boyd, M.D., inserted following removal of a giant cell tumor of the femoral neck is shown in Figure 10.

SUMMARY

Total prosthetic replacement of the hip is becoming a frequently performed orthopedic procedure. The radiologist should be familiar with the appearance of various devices as well as the methods of insertion and fixation of the pelvic and femoral components, as these determine the postoperative roentgen appearance of the hip.

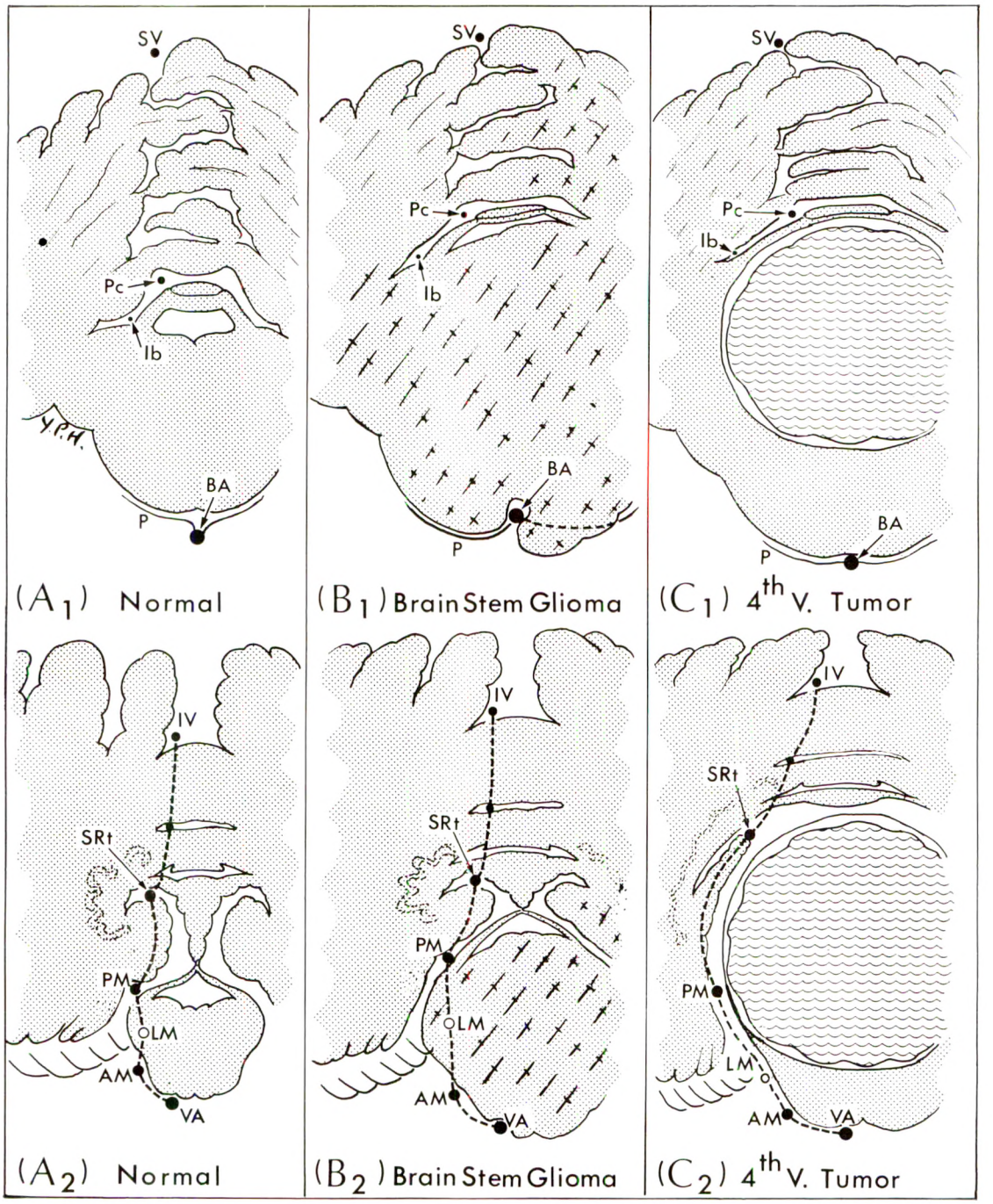


FIG. 3. Diagrammatic horizontal sections of cerebellum and brain stem showing the anatomic relationships of pertinent arteries in normal cases, in brain stem tumors, and in fourth ventricle tumors. Portions of the arteries not in the plane of section are dashed.

The labeled structures include the anterior medullary segment (AM), basilar artery (BA), interbrachial artery (Ib), vermian branch (IV) of the posterior inferior cerebellar artery, lateral medullary segment (LM), pontine artery (P), precentral cerebellar artery (Pc), posterior medullary segment (PM), superior retrotonsillar segment (SRt), vermian branch (SV) of the superior cerebellar artery and vertebral artery (VA).

The more recently developed prostheses and their roentgen appearances are presented.

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tery[19] is marked. The portions of this vessel in front of the brain stem are displaced forward. The posterior medullary segment, located between the posterior aspect of the medulla and the anterior aspect of the body of the tonsil, is markedly displaced backward. The intervening portions will be stretched, elongated and lose their normal tortuosity (Fig. 5*A*; and 8*A*). Since there is considerable variation in the course of the anterior and lateral medullary segments of the posterior inferior cerebellar artery, it is difficult to be certain of abnormalities of these segments. This is especially true in the Towne projection (Fig. 8*B*; and 9*B*). On the other hand, the location of the posterior medullary segment, particularly its distal part, is more consistent and posterior displacement of this segment is more reliable and of greater diagnostic importance.[4,23] Displacement of this segment is usually most marked near its junction with the supratonsillar segment, *i.e.*, the choroidal point (Fig. 5*A*; 8*A*; and 9*A*). In 90 per cent of normal cases, the foot of the perpendicular, dropped from the choroidal point to a line joining the anterior margin of the foramen magnum and the torcular (FT line), will be located from 1 mm. anterior to 3 mm. posterior to the point dividing the anterior one-third from the posterior two-thirds of the FT line.[19] Gonsette *et al.*[8] concluded that the posterior border of the medulla oblongata is located at the point dividing the anterior one-fourth and the posterior three-fourths of the line connecting the anterior margin of the foramen magnum and the torcular. The posterior medullary segment of the posterior inferior cerebellar artery is located behind this point

(A_1), (B_1) and (C_1) are sections through the upper pons; (A_2), (B_2) and (C_2) through the upper medulla oblongata.

(A_1) *Normal.* Note particularly the relationship of the precentral cerebellar artery (Pc) and the interbrachial artery (Ib) to the fourth ventricle. The vermian branch (SV) of the superior cerebellar artery outlines the culmen.

(A_2) *Normal.* The lateral medullary segment (LM) of the posterior inferior cerebellar artery is usually located considerably below the level of this section. The anteroposterior diameter of the body of the cerebellar tonsil is indicated by the distance between the posterior medullary segment (PM) and the superior retrotonsillar segment (SRt), bridged by the supratonsillar segment (not labeled) running over the superior pole.

(B_1) *Brain stem glioma.* The tumor is indicated by crossed lines. Note the deep groove on the anterior aspect of the pons produced by the basilar artery (BA). On the reader's right, the pontine artery produces a deep groove in a protruding tumor nodule, whereas on the left it is displaced forward with the expanded brain stem. The precentral cerebellar artery (Pc) is displaced markedly backward. The superior vermis is compressed as indicated by the decreased distance between the precentral cerebellar artery and the vermian segment (SV) of the superior cerebellar artery.

(B_2) *Brain stem glioma.* Since the lower part of the medulla oblongata is usually less severely involved, lateral displacement of the lateral medullary segment is often difficult to detect. In addition, normal variations in the course of this segment within the lateral medullary cistern are marked. The posterior medullary segment (PM) and, to a lesser degree, the superior retrotonsillar (SRt) and the vermian segments (IV) of the posterior inferior cerebellar artery are displaced posteriorly. The tonsil is compressed from in front. The proximal portion of the posterior inferior cerebellar artery made up by the anterior and lateral medullary segments is stretched.

(C_1) *Fourth ventricle tumor.* The basilar artery (BA) and pontine arteries (P) are displaced forward. Changes in the precentral cerebellar and interbrachial arteries are similar to those seen with brain stem tumors, although more marked lateral displacement of these vessels is often seen in fourth ventricle tumors.

(C_2) *Fourth ventricle tumor.* The lateral medullary (LM), posterior medullary (PM), the supratonsillar (not labeled), and superior retrotonsillar segments (SRt) are markedly stretched and elongated in the anteroposterior direction. These segments form a smooth continuous curve. The posterior medullary segment is shown as displaced slightly forward. However, if the original location of this segment was more posterior on the medial aspect of the tonsil, moderate posterior displacement may be present. The tonsils are widely separated and compressed from their medial aspects.

DOUBLE-CONTRAST ARTHROGRAPHY IN THE DIAGNOSIS OF PIGMENTED VILLONODULAR SYNOVITIS OF THE KNEE*

By LT. COLONEL RICHARD D. WOLFE, USAF, MC,† *and* MAJOR VINCENT J. GIULIANO, USA, MC‡
LACKLAND AFB, TEXAS

PIGMENTED villonodular synovitis is not a new disease, as it was first described by Simon in 1865, but even today, more than 100 years later, it is infrequently correctly diagnosed preoperatively, especially when it involves the knee. With the introduction of opaque contrast and then double-contrast knee arthrography, it would seem that more of these cases would be identified prior to surgery, but we were only able to find a total of 12 cases in which knee arthrography was used and was helpful in establishing the diagnosis. Seven of these used the double-contrast method, with 5 cases published by Bessler and Ruttiman[2] and 1 by Czekala,[10] while Butt and McIntyre[7] reported the only case in the English language literature. Others, each reporting single cases utilizing only positive contrast material, were Aye *et al.*,[1] Rein *et al.*[22] and Lindblom.[19] Brashear[5] mentions 1 case in which the diagnosis was suggested by pneumoarthrography, while Kelikian and Lewis[17] presented an equivocal case studied also with pneumoarthrography.

A recent case in which the preoperative diagnosis of pigmented villonodular synovitis of the knee was made with double-contrast arthrography motivated us to report the 2 cases we have in the hope of stimulating interest in its use in the diagnosis of synovial tumors.

REPORT OF CASES

CASE 1. A 25 year old white male was transferred to Wilford Hall USAF Medical Center after initial care at Schilling AFB, Kansas. He had been well until 4 months prior to admission when he noted swelling of his right knee. No other joints were involved and there was no history of trauma. The condition was diagnosed as probable strain, and rest and heat resulted in partial relief of the swelling. This recurred and when it became quite distended there was some pain with motion. A mass was easily palpable but was thought to be a lipoma. At surgery, it was thought to represent a muscle mass but the inadequate biopsy revealed only normal muscle and he was referred to our facility.

On admission there was a recent but well-healed lateral incision with diffuse swelling of the entire joint, most pronounced in the area of the suprapatellar bursa and laterally. Flexion was limited to 70° by pain. The knee was otherwise stable and no local heat or erythema was noted. All laboratory studies, including sedimentation rate × 3, were normal. Plain roentgenograms of the knee revealed a normal bony architecture without osteoporosis and with the joint space maintained. A soft tissue mass was seen in the area of the suprapatellar bursa.

Double-contrast arthrography was performed, the details of which are not in the record, and these representative roentgenograms were obtained (Fig. 1, *A–D*). Aspiration at the time of arthrography revealed a frankly bloody effusion which showed no growth on culture. Although the roentgenograms are felt to be diagnostic of pigmented villonodular synovitis, this was not fully appreciated preoperatively for it was thought that this most likely represented a rhabdomyosarcoma with invasion of the synovium.

At surgery an extremely large, fungating mass occupied the entire knee joint and extended into the thigh laterally. A total synovectomy was accomplished and this was followed by radiation therapy with 1,600 rads over a 2 week period. Pathologic examination

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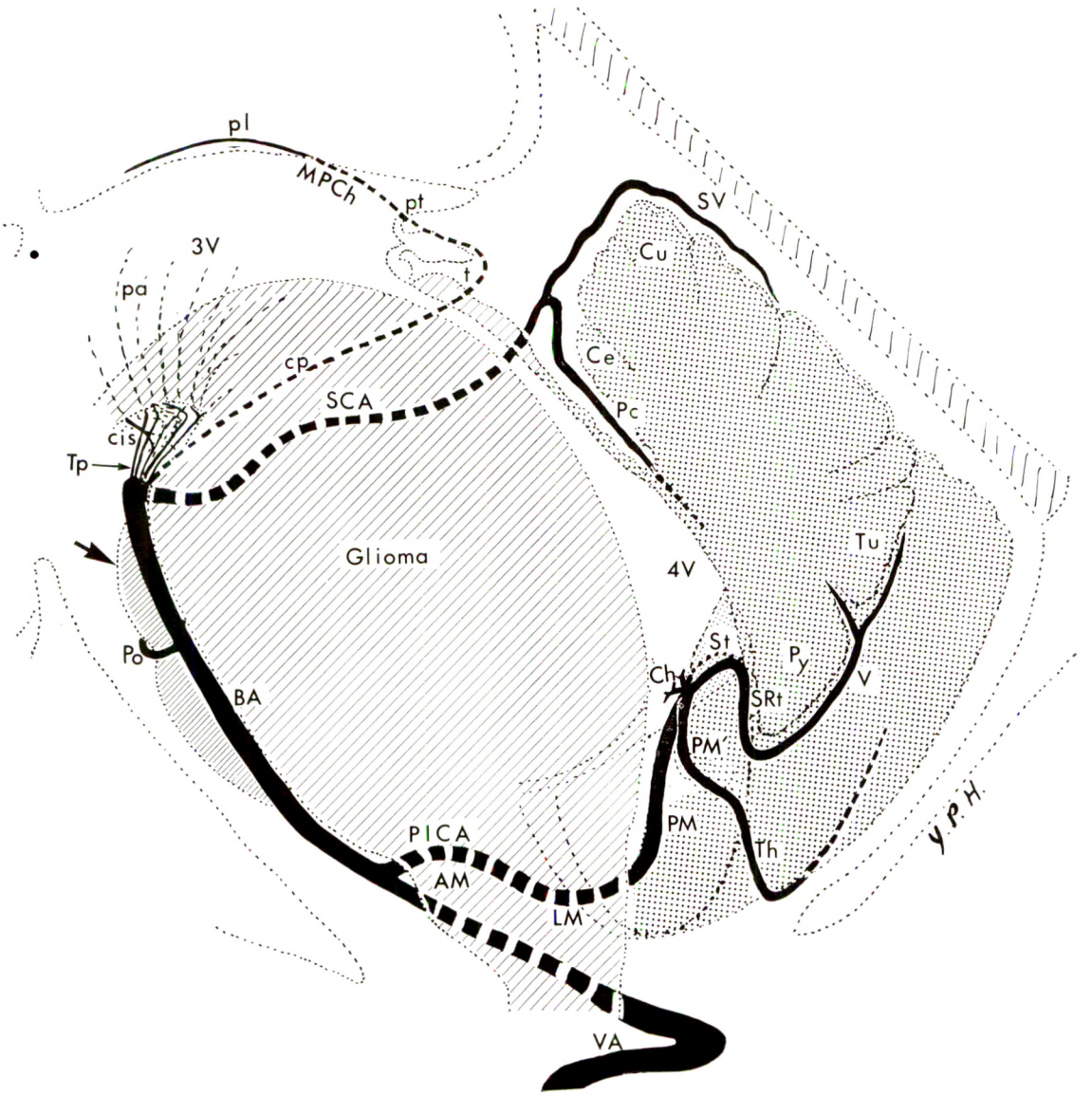


FIG. 4. Diagrammatic representation of "mid-sagittal section" showing arteriographic changes in brain stem tumors. An extensive tumor, from medulla to subthalamic region, is illustrated.

Labeled are the anterior medullary segment (AM), basilar artery (BA), central lobule (Ce), the choroidal branches (Ch), cisternal segment (cis), circumpeduncular segment (cp), culmen (Cu), lateral medullary segment (LM), medial posterior choroidal artery (MPCh), parenchymal segment (pa), precentral cerebellar branch (Pc), posterior inferior cerebellar artery (PICA), plexal segment (pl), posterior medullary segment (PM) of the posterior inferior cerebellar artery, posterior medullary segment (PM′) of the tonsillo-hemispheric branch, pontine artery (Po), pretectal segment (t), pyramid (Py), superior cerebellar artery (SCA), superior retrotonsillar segment (SRt), supratonsillar segment (St), vermian segment (SV) of the superior cerebellar artery, tectal segment (t), tonsillo-hemispheric branch (Th), thalamo-perforating arteries (Tp), tuber (Tu), and vertebral artery (VA). The third and fourth ventricles (3V, 4V) are also labeled.

The vertebral (VA) and basilar arteries (BA) have an elongated, stretched appearance with effacement of the usual posterior convexity of the vertebral artery near its junction with the basilar artery. The basilar artery is displaced forward. Lobulated masses (unlabeled arrow) protrude from the anterior surface of the enlarged pons and extend further forward than the basilar artery. Angiographically, this is indicated

revealed 115 grams of tissue, the largest piece being 15×8×1.5 cm. with a shiny brown surface. The histologic appearance was quite typical of pigmented villonodular synovitis. The postoperative recovery was slow and at discharge 4 months later he had a fibrous ankylosis of the knee with weakness and loss of motion. Further follow-up has not been possible.

CASE II. A 23 year old white female had noticed recurrent episodes of swelling and stiffness of the right knee for approximately 1 year. She was in otherwise good health with no other joints involved. She recalled having fallen on her knees several times while ice skating prior to the onset of symptoms.

On admission to Brooke Army General Hospital the only positive physical findings were limited to the right knee, which was slightly warm and diffusely tender, but only mildly so. There was a large suprapatellar effusion, but no definite mass could be palpated, even after aspiration of a large amount of blood-tinged synovial fluid.

Laboratory studies were all normal including the sedimentation rate, the slide latex test for rheumatoid factor and the fluorescent antinuclear antibody test. Plain roentgenograms of the knees were negative except for an effusion.

A double-contrast arthrogram was obtained utilizing 10 cc. of 75 per cent hypaque and 20 cc. of room air. The roentgenograms were diagnostic of a diffuse pigmented villonodular synovitis (Fig. 2, *A–D*).

At surgery a boggy synovial mass was identified in the suprapatellar region and a total synovectomy was accomplished through medial parapatellar and lateral posterior incisions. The mass was attached to the patella but not to the collateral ligaments. Pathologic examination revealed villous proliferation and hemosiderin-laden macrophages, considered typical of pigmented villonodular synovitis. No postoperative radiation therapy was given.

DISCUSSION

In 1941 Jaffe *et al.*[14] reviewed the confusing literature and their own cases and recognized common gross and histopathologic characteristics which allowed them to group under a common name all of these processes which previously were reported under various names. They designated the condition as "pigmented villonodular synovitis, bursitis or tenosynovitis" and this has remained as the accepted term.

The condition may be nodular or diffuse. If one excludes the nodular and quite characteristic phalangeal lesions, the vast majority are located in the knee and most of these are of the diffuse type. The less common nodular type of the knee is rarely if ever diagnosed prior to surgery, but instead presents as an internal derangement and frequently has episodes of locking. There are no reports of the use of arthrography in this localized type and it is doubtful if it would be helpful other than in a negative manner. If no meniscal injuries were demonstrated then one would think of other possibilities for the etiology of the patient's symptoms.

Gehweiler and Wilson[12] collected from the literature 300 cases of diffuse pigmented villonodular synovitis. The knee was by far the most common location and was then followed by the hip, ankle, tarsus, carpus, elbow and shoulder, in that order. They reported only the third and fourth cases of biopsy proven biarticular involvement with one of their cases having both knees involved and the other the right knee and right hip. Greenfield and Wallace[13] in 1950 presented a case with both knees, and Byers *et al.*[8] in 1968 reported 1 case with both ankles involved. Kelikian and Lewis[17] in 1949 reported 3 siblings with biarticular involvement but there was no proof and these do not appear to represent this entity. Friedman and Schwartz[11] reported a case with both knees affected but the involvement of the second knee occurred 10 years after the first and there was no biopsy of the second knee.

Lewis[18] in 1947 described the typical patient with the diffuse process as a young adult with monarticular involvement and with an excessive amount of synovitis which may be smooth in outline and homogeneous in density but is especially diagnostic when it is nodular. He emphasized joint space symmetry with no indication of cartilage abnormality, normal appearing bones without osteoporosis and no

by about 5 to 7 mm. since the vessel is more closely applied to the tonsil than to the medulla. When the neoplasm does not involve the tonsil, the normal arch of the supratonsillar segment of the posterior inferior cerebellar artery over the superior pole of the tonsil is shortened and tilted posteriorly (Fig. 5*A*). The superior retrotonsillar segment is displaced posteriorly to a lesser degree than the posterior medullary segment, indicating compression of the tonsil in an anteroposterior direction. When the tonsil is involved by extension of the neoplasm, however, the anteroposterior diameter of the tonsil may be increased. This is indicated by an increased distance between the posterior medullary and superior retrotonsillar segments and a widening of the supratonsillar arch (Fig. 9*A*). When this distance is normal, it is also likely that the tumor has spread into the tonsil since, in the absence of involvement, the distance should be diminished.

Because of posterior displacement of the superior retrotonsillar segment, the curved course of this vessel as it runs below the copula pyramidis to join the vermian segment becomes more acute (Fig. 5*A*; and 9*A*). When the neoplasm extends into the inferior vermis, however, the downward curve below the copula pyramidis may be widened and the vermian segment of the artery markedly arched backward and downward (Fig. 8*A*).

The tonsillo-hemispheric branch of the posterior inferior cerebellar artery is difficult to evaluate because of marked normal variations in its origin and course. Frequently, however, there is an identifiable portion of this vessel which runs on the posterior medullary aspect of the cerebellar tonsil behind the medulla oblongata. It may therefore also be referred to as a posterior medullary segment, but of the tonsillo-hemispheric branch.[19] This segment will also be posteriorly displaced (Fig. 8*A*; and 9*A*). In other instances, a portion of this vessel runs over the superior pole of the cerebellar tonsil and will therefore be displaced in a fashion similar to the corresponding segments of the posterior inferior cerebellar artery itself.

The *anterior inferior cerebellar artery* which runs in the lower part of the pontine and ponto-cerebellar cisterns is stretched laterally and displaced anteriorly. This is, however, difficult to evaluate roentgenologically because of the variable origin and course of this vessel (Fig. 8*B*). Modification of its appearance may, in addition, be concealed in the Towne projection. The anteroposterior reversed Water's view, combined with subtraction, may show changes in the course of this vessel more clearly.[27] In some cases, displacements of the *posterior cerebral artery*—elevation, lateral displacement, and widening of its curved course around the mesencephalon (Fig.

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by the fact that pontine branches (Po) extend farther forward than the basilar artery. The cisternal (cis) and parenchymal portions (pa) of the thalamo-perforating arteries (Tp) are displaced forward and upward. Short transverse portions of these arteries at their origins are lost due to elevation and effacement of the posterior inferior corner of the interpeduncular fossa. The lateral pontine portion of the superior cerebellar artery is stretched with a reversal of its normal inferior convexity. The precentral cerebellar artery (Pc) is displaced backward and straightened so that it appears to be roughly parallel to the clivus. The superior cerebellar artery (SCA) as it crosses the apex of the cerebellum is more pointed and the vermian branch (SV) is closer to the straight sinus. The anterior, lateral and posterior medullary segments (AM, LM, PM) of the posterior inferior cerebellar artery (PICA) are stretched and less tortuous than usual. The posterior medullary segment (PM) is displaced markedly posteriorly, particularly in its upper portion. The upper part of the cerebellar tonsil is flattened and tilted backward as indicated by shortening of the supratonsillar segment (St) and diminution in the distance between the posterior medullary (PM) and the superior retrotonsillar segments (SRt). The copular angle formed by the superior retrotonsillar (SRt) and vermian segments (V) of the posterior inferior cerebellar artery is more acute than normal. The vermian segment (V) is displaced slightly backward.

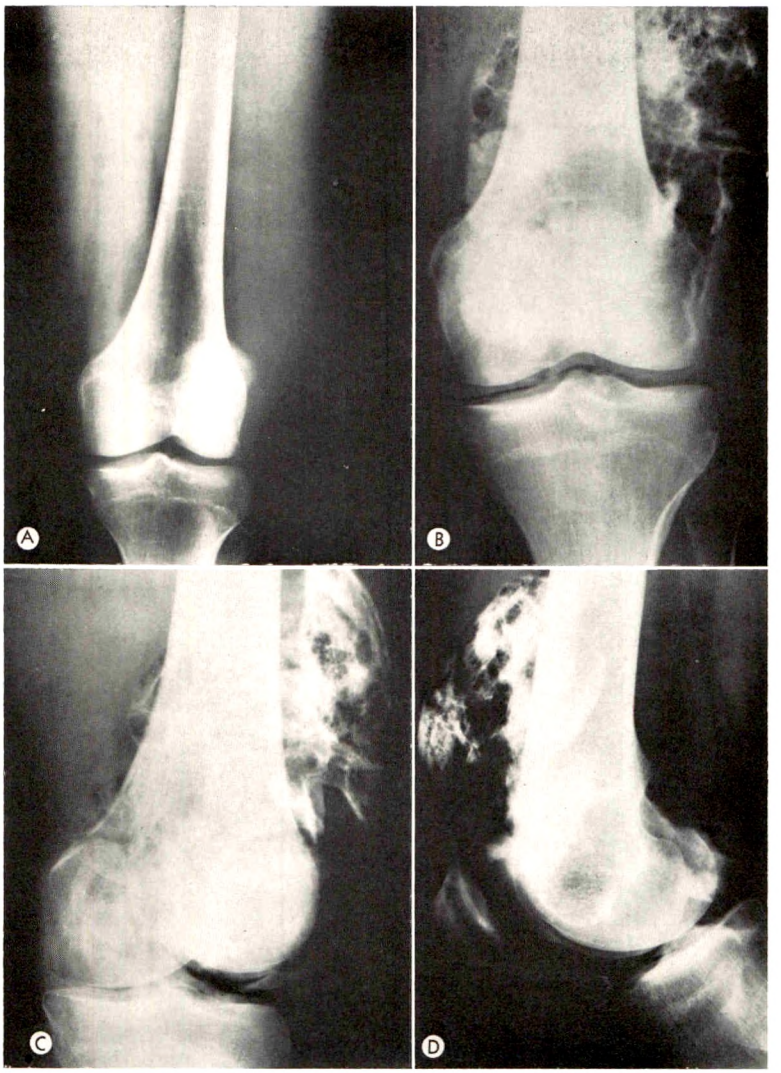


Fig. 1. Case 1. Diffuse pigmented villonodular synovitis of right knee. (*A*) Soft tissue swelling of suprapatellar area laterally. (*B*) Anteroposterior view. (*C*) External rotation view. (*D*) Lateral view. Enlarged suprapatellar bursa almost completely filled with mass composed of recesses and villous proliferations.

thigh or leg atrophy. When one adds a lack of calcification in the mass and a chronicity of symptoms to this, one has criteria which still apply to the vast majority of these patients.

McMaster[21] in 1960 reported 6 cases with bone changes. Three of these were in the hip, where previous cases had been reported by Breimer and Freiberger,[6] while his 3 cases in the knee were the first reported with bone changes in that joint. Since that time a few more cases have shown relatively nonspecific changes, although, unlike the hip, involvement of the knee remains the exception rather than the rule. There has been much discussion as to how the bone becomes involved but that is beyond the scope of this paper.[6,9,21,24] Neither of our patients demonstrated roentgenographic evidence of bone abnormality.

ETIOLOGY

The etiology of this process remains obscure, although most agree with Jaffe that it represents a benign inflammatory reaction to some as yet unidentified agent.

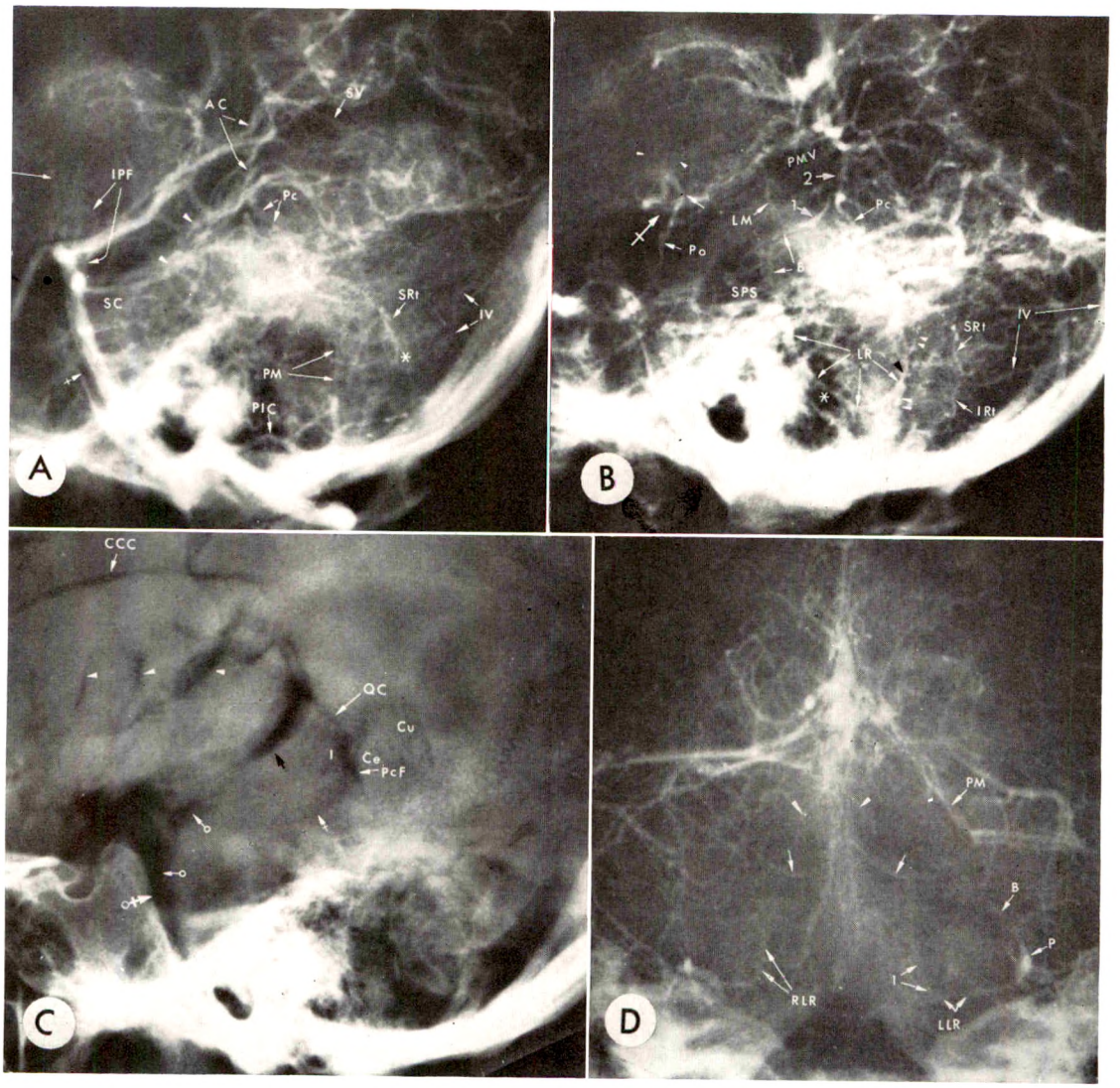


FIG. 5. Large brain stem tumor with extension through brachium pontis and restiform body into the cerebellar hemisphere and involvement of the mid-brain

(*A*) Left brachial angiogram, arterial phase, lateral view. The basilar artery is stretched and closely applied to the clivus (‡). The thalamo-perforating arteries (arrow, left upper margin) are straightened and crowded forward. The interpeduncular fossa (IPF) is elevated and effaced from below and behind. The posterior part of the posterior communicating artery is elevated (black arrow). The juxtategmental portion (unlabeled arrowheads) of the superior cerebellar artery is not remarkable. The anterior culminate segment (AC) is displaced backward. An acute angle is formed between this segment and the vermian segment (SV). The fissural portion of the precentral cerebellar artery (Pc) is displaced backward. The distance between the basilar artery and the precentral artery is increased, while the distance between the precentral artery and the vermian segment (SV) is diminished. The proximal portion of the posterior inferior cerebellar artery (PIC) is stretched. The posterior medullary segment (PM) is markedly displaced backward, corresponding to similar displacement of the floor of the fourth ventricle. The distance between this segment and the superior retrotonsillar segment (SRt) is diminished, indicating decreased height of the fourth ventricle. The copula pyramidis (*) and the vermian segment (IV) of the posterior inferior cerebellar artery are crowded backward.

(*B*) Venous phase, lateral view. The pontine segment (Po) of the anterior ponto-mesencephalic vein is displaced forward and upward. The region of the superior foramen caecum (↑) is elevated and the interpeduncular fossa (‡) is not only narrowed, but tilted and displaced forward. The thalamo-perforating

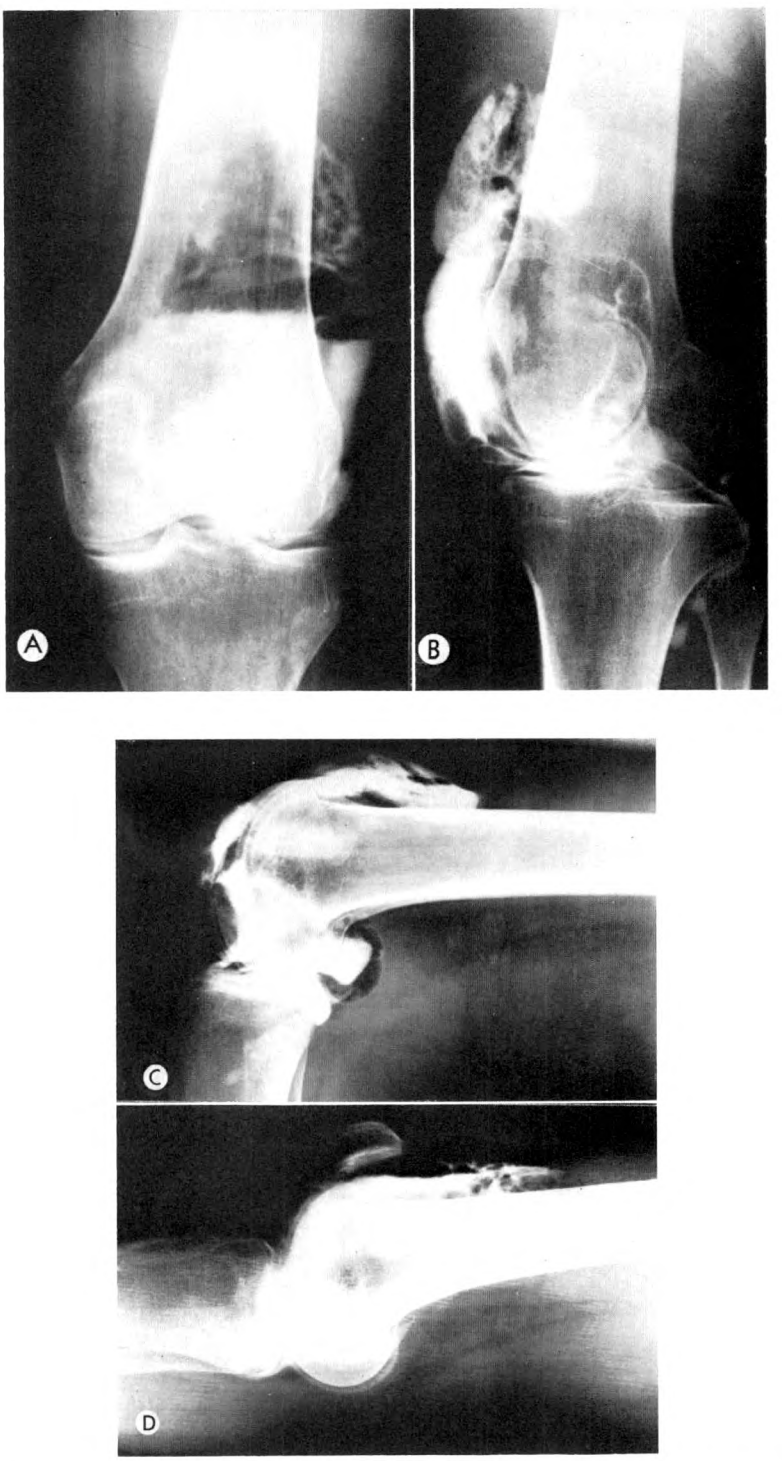


FIG. 2. Case II. Diffuse pigmented villonodular synovitis of right knee. (*A*) Upright anteroposterior view. (*B*) Internal rotation view. (*C*) Lateral view. (*D*) Cross-table lateral view. Moderate enlargement of suprapatellar bursa with filling defects coated with positive contrast material. Note value of upright and cross-table lateral views to differentiate from bubbles or frothy fluid.

7*A*)—may be sufficiently marked to be of diagnostic significance. It is curious that occasionally, despite upward displacement, the posterior cerebral and superior cerebellar arteries may be displaced medially instead of laterally due to nodular outgrowths of pseudo-extra-axial type (Fig. 9, *A* and *B*). In brain stem tumors, medial displacement of posterior cerebral and superior cerebellar arteries due to downward herniation of the hippocampal gyri is rare unless the primary involvement is in the aqueduct or periaqueductal region.

The *thalamo-perforating arteries* are stretched, displaced and arched forward. This is associated with elevation of the floor of the posterior portion of the third ventricle. With greater involvement on one side, the thalamo-perforating arteries may be displaced contralaterally (Fig. 7).

With extensive spread into the thalamus, the *lateral posterior choroidal artery* may show widening of its posterior arch and elevation of its suprathalamic portion as seen with primary thalamic tumors. Changes in the *medial posterior choroidal artery* are of special interest because of its close relationships to the tectal region. This artery (Fig. 1) usually originates from the proximal portion of the posterior cerebral artery and courses around the brain stem parallel and medial to the posterior cerebral artery (circumpeduncular segment). It then takes a curved recurrent course (tectal segment) superiorly and medially adjacent to the mesencephalo-diencephalic sulcus around the tectum. The vessel continues forward, passes lateral to the pineal gland to cross over the habenular trigone and commissure (pretectal segment), and enters the cisterns of the vein of Galen and velum interpositum to supply the choroid plexus of the third ventricle (plexal segment). With upward bulging of the pons, the circumpedun-

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veins (two opposing arrowheads) can be faintly seen and are displaced anteriorly. The lateral mesencephalic vein (LM) is also elevated. This vein communicates inferiorly with brachial tributaries (B) of the petrosal vein which outline the elevated superior aspect of the brachium pontis. The anterior culminate segment (2) of the precentral cerebellar vein is displaced backward by enlarged tectum. The marginal tributary (1) of the precentral cerebellar vein is also displaced backward and somewhat downward by the widened mid-brain. The posterior mesencephalic vein (PMV) is elevated. The vein of the lateral recess of the fourth ventricle (LR) is stretched and shows a marked inferior arcuate convexity indicating enlargement of the brachium pontis and restiform body. The normal angular course of this vein at the cerebello-ponto-medullary angle (*) is lost. The medial tonsillar supratonsillar tributaries (double arrowheads) join the vein of lateral recess of the fourth ventricle at the posteriorly displaced tonsillar incisura (black arrowhead). Together with the superior and inferior retrotonsillar tributaries (SRt, IRt) of the inferior vermian vein (IV), they outline the posteriorly displaced cerebellar tonsil, flattened from in front.

(*C*) Pneumoencephalogram, lateral view. No air entered the ventricular system. The anterior margin ( ) of the brain stem has an irregular outline and is unusually close to the clivus ( ). The interpeduncular cistern is elevated and its depth obscured. The precentral cerebellar fissure (PcF), between the inferior colliculi (I) and lingula in front and the central lobule (Ce) behind, is displaced backward as is the narrowed quadrigeminal cistern (QC). The apex of the cerebellum formed by the culmen (Cu) is pointed and displaced backward. The cistern of the corpus callosum (CCC) and insular sulci (arrowheads) are also labeled. The distance between the wing of the ambient cistern (retropulvinar cistern, black arrow) and the wing of the precentral cerebellar cistern (infratentorial portion of ambient cistern,) is increased indicating elongation of the mid-brain.

(*D*) Venous phase, Towne view. The faintly seen peduncular segment (1) of the vein of the lateral recess on the left (LLR) is displaced medially. The distance between this segment and the arched brachial tributary (B) of the petrosal vein (P) is increased. The vein of the lateral recess of the fourth ventricle on the right side (RLR) is somewhat laterally displaced.

The peduncular portions ( ) of the posterior mesencephalic veins (PM) are seen on both sides and show an exaggerated sweep on the left. The brachial tributaries ( ) of the precentral cerebellar vein are minimally separated.

Many have incriminated bleeding into the joint, although only Dahlin (as cited by Chung and James[9] in a personal communication) has seen a case in a hemophiliac with findings closely simulating but not identical to that of pigmented villonodular synovitis. Many interesting experimental studies have been only partially successful in producing lesions somewhat resembling this entity.[20,25,28] Wright[27] in 1951 felt that this represented a neoplasm related to the ordinary malignant synovioma and he insisted that benign giant cell synovioma is a more appropriate designation. This was not widely accepted, although recently Jones *et al.*[16] again suggested this possible relationship. They based this conclusion on an article by Bliss and Reed[3] in which 4 cases of malignant giant cell tumor of tendon sheaths, 2 with metastases, were reported. There had been no previous cases suggesting malignancy or metastases and there have still been none of the diffuse type involving the larger joints. Bobechko and Kostuik[4] recently reported 3 children with hemangiomas of the synovium. They suggested that these 2 conditions may be related and recommended arteriography in all adults with this diagnosis. Suffice it to say that the true etiology of this condition has not been definitely established!

ARTHROGRAPHY AND ARTERIOGRAPHY

Careful arthrography should be almost uniformly successful in demonstrating the fungating synovial lesions present. Some authors, however, have not found it of value, although in most cases only air was used.[13,15,23] We favor the double-contrast method for all arthrographies but especially when a synovial lesion is suspected. These will demonstrate the tremendous increase in the size of the articular cavity, will show the joint effusion and will demonstrate the synovial villi projecting into the suprapatellar bursa and posterior knee pouches. The coating by the positive contrast material and the distention from the air render an easy diagnosis possible when proper roentgenograms are obtained. A cross-table lateral and an upright views are of utmost importance in demonstrating these synovial abnormalities.

During the actual injection of the positive contrast material in our second patient, she complained of pain and even cried, although she did not show a low threshold for pain when the needle was inserted. In several hundred previous studies done by one of the authors (RDW) this was never seen. Usually there is no pain but only slight discomfort from the mild distention. Even the burning encountered if some of the contrast material extravasates is never as severe as this was. It is probable that the large increase in surface area with its abnormal synovial lining is responsible for this irritative phenomenon and this may be a diagnostic point to watch in future arthrograms. Unfortunately there was no written comment regarding the arthrogram in the first patient.

Arteriography was performed by Rein *et al.*[22] but they concluded that the malignant type vascular changes seen rendered this of little diagnostic assistance. Its greatest value was in delineating the extent of the tumor.

TREATMENT

Total synovectomy with or without postoperative irradiation is generally accepted as the treatment of choice. Byers *et al.*[8] in a large series of their own cases and in reviewing the literature found an extremely poor result with extensive synovectomy of the knee with a recurrence rate of 46 per cent. A complete cure with total synovectomy of the knee was obtained in only 17 per cent. In 5 cases in which biopsy alone was done no further deterioration occurred. They treated 8 patients with radiation therapy and, although all showed regression of bone and soft tissue lesions, only 2 could be considered as having satisfactory results. Fortunately not all of the results in the literature are so dismal.

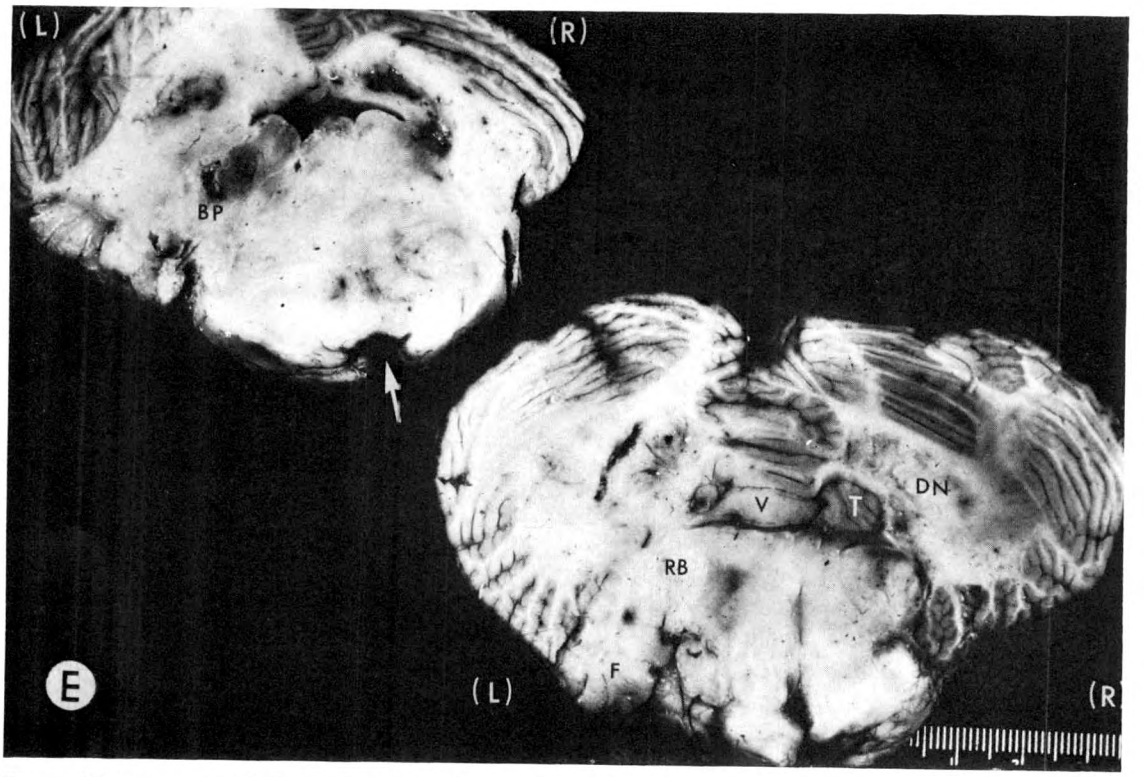


FIG. 5. (*E*) Same case. Horizontal sections of the mid-pons (upper section) and upper medulla (lower section). The brain stem is markedly enlarged by infiltrating astrocytoma, with resultant flattening and posterior displacement of the fourth ventricle. The deep groove on the anterior aspect of the pons is occupied by the basilar artery (arrow). The brachium pontis, restiform body, dentate nucleus (DN), medullary substance of the cerebellar hemisphere, flocculus (F) and inferior vermis (V) on the left side (L) are infiltrated by the tumor. The tonsil (T) on the right (R) is displaced backward and flattened from front. The tonsil on the left side is not clearly seen.

FIG. 6. Brain stem astrocytoma.

(*A*) Left brachial angiogram, arterial phase, lateral view. The upper part of the basilar artery (⇕) is straightened but is not unusually close to the clivus (‡). The clivus is convex anteriorly. The thalamoperforating arteries (single arrowhead) are stretched and displaced forward. The precentral cerebellar artery (row of black arrowheads) and the anterior culminate segment (white arrows) of the superior cerebellar artery are posteriorly displaced.

(*B*) Arterial phase, Towne view. The superior cerebellar arteries (arrowheads) are displaced outward and outline the circular shape of the enlarged upper brain stem.

(*C*) Venous phase, lateral view. The pontine veins on the anterior aspect of the pons (⇕) are unusually close to the clivus (‡). This is in contrast to the position of the basilar artery (Fig. 6*A*). The difference is due to nodular hypertrophy and deep grooving of the pons. The precentral cerebellar vein (Pc) is displaced markedly posteriorly. The distance between this vein and the straight sinus (SS) is diminished. The posterior portion of the posterior mesencephalic vein (PMV) appears elevated but this is difficult to judge because of normal variations of this vessel in this area. The internal cerebral vein (IC) is not remarkable.

(*D*) Pneumoencephalogram, lateral view, erect position. The floor (row of arrowheads) of the aqueduct and fourth ventricle (4V) is displaced markedly backward in arcuate fashion. The collicular plate, with quadrigeminal cistern (QC) behind it, is displaced backward and upward, flattened and elongated. The central lobule (Ce) and the culmen (Cu) are displaced backward. The superior vermis is pointed at its apex (‡). The superior cerebellar cistern (‡) is narrow. The lateral ventricle (LV) and the third ventricle (3V) are not enlarged.

SUMMARY

Two patients with a histologically proven diagnosis of pigmented villonodular synovitis of the knee are reported. This diagnosis should be considered and an arthrogram obtained when a young adult presents with chronic swelling and stiffness of a single joint with minimal if any pain and with bogginess of the synovium.

The condition is even further suspected when aspiration reveals a moderately large quantity of dark blood-containing effusion, and plain roentgenograms show a normal joint other than the effusion and occasionally some nodularity of the suprapatellar area.

When the double-contrast method is used, preoperative arthrography should result in a diagnosis, as was the case in the 2 patients reported.

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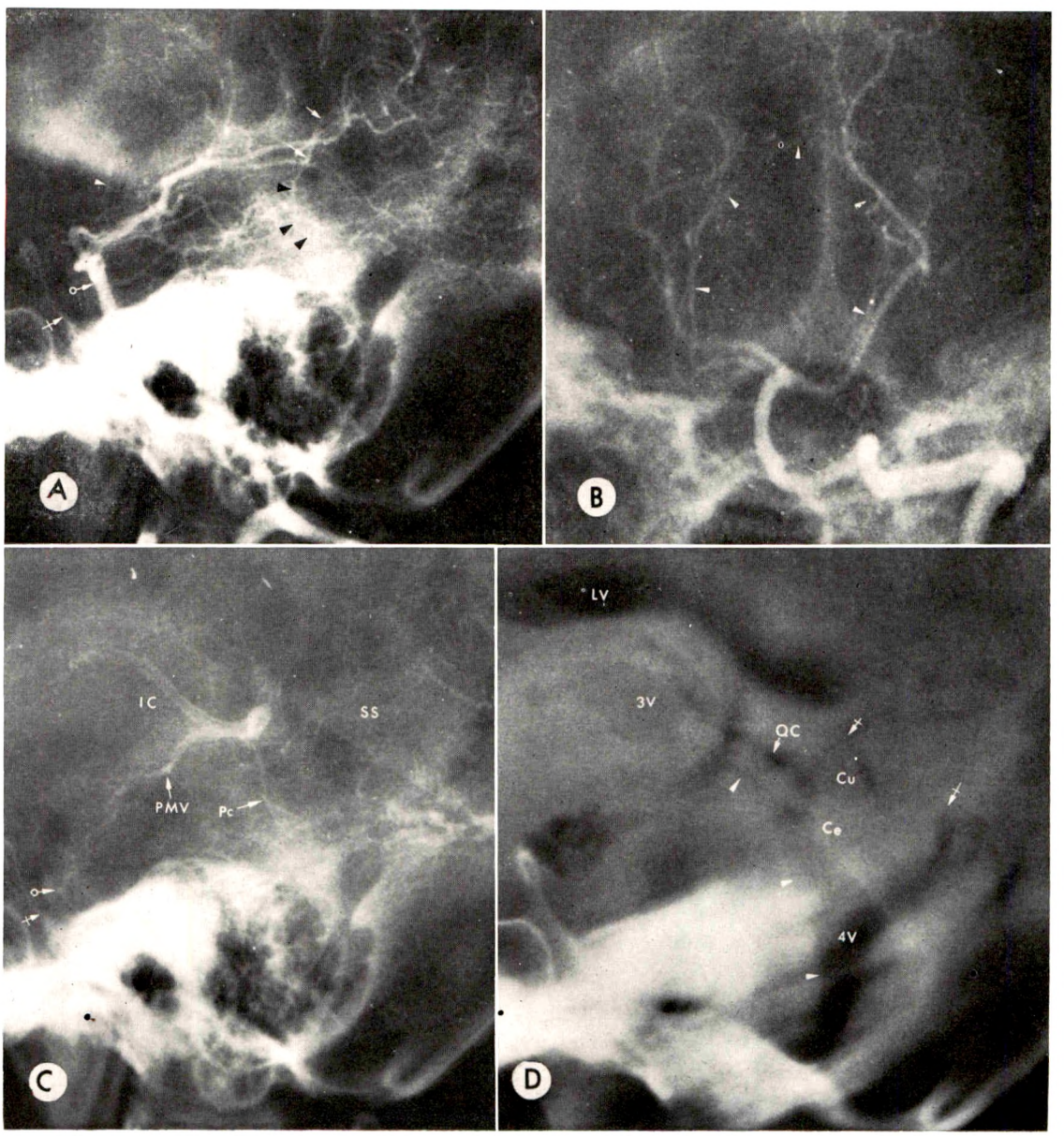
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cular segment is displaced upward and outward. As seen in the lateral projection, the distance between circumpeduncular and pretectal segments is decreased and the posterior arch (tectal segment) becomes more acute (Fig. 9*C*). With tumor extension into the quadrigeminal plate, the tectal segment is displaced laterally and outward and the pretectal segment elevated. With thalamic involvement, the plexal segment is elevated.

When the tumor extends into the medulla oblongata and upper cervical cord, *anterior spinal arteries*[25] are displaced forward while *posterior spinal arteries* are displaced backward.

B. VENOGRAPHIC FEATURES

Veins of importance in the diagnosis of brain stem tumors include: (1) the roof, mesencephalic and pontine segments of the anterior ponto-mesencephalic vein;[14,16] (2)



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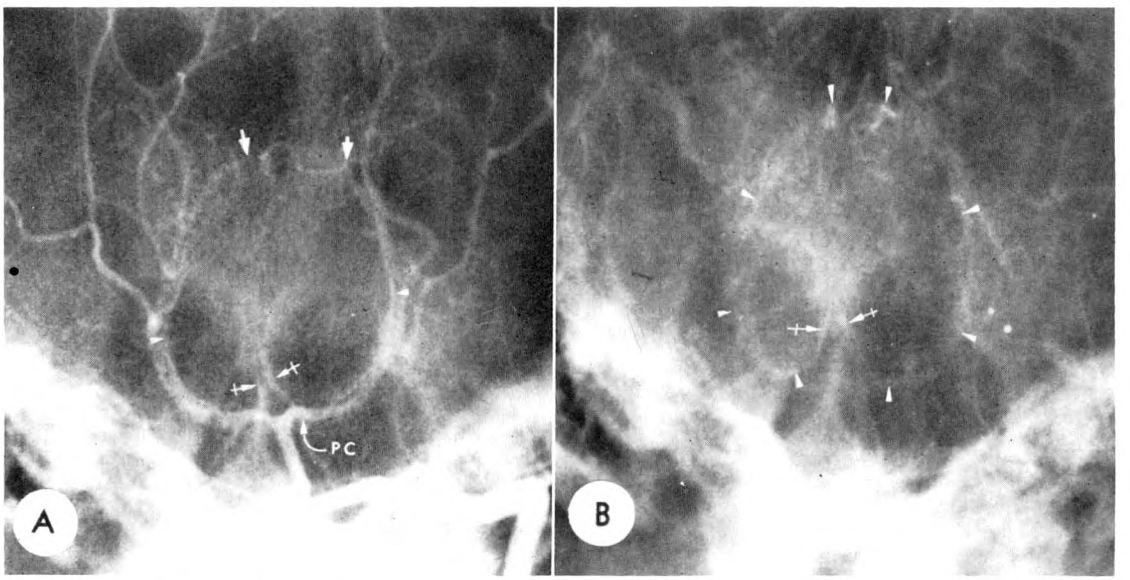


FIG. 7. Upper brain stem tumor.

(*A*) Left retrograde brachial angiogram, arterial phase, Towne view. Outward displacement (small arrowheads) of the posterior cerebral arteries (PC) indicates enlargement of the brain stem. This is more marked on the left side as evidenced by a shift of the thalamo-perforating arteries (↟) to the right (reader's left). The tectal portions of the medial posterior choroidal arteries (↑) are rounded and displaced backward.

(*B*) Early venous phase, Towne view. Faintly visualized posterior mesencephalic veins on both sides (arrowheads) outline the enlarged mid-brain with shift of the interpeduncular fossa to the right (↟).

the parenchymal, fissural, anterior culminate and terminal segments of the precentral cerebellar vein;[13] (3) the superior vermian vein[14] and its culminate tributaries; (4) the vein of the lateral recess of the fourth ventricle[15] and supratonsillar and medial tonsillar tributaries; (5) the lateral mesencephalic[14,29] and interbrachial veins;[14] and (6) the inferior vermian vein[18] and superior and inferior retrotonsillar tributaries (Fig. 10; 11; and 12).

Enlargement of the brain stem anteriorly is indicated by forward and upward displacement of the pontine segment of the anterior ponto-mesencephalic vein. With involvement of the upper brain stem, the deep portion of the interpeduncular cistern is elevated, narrowed from below and behind, and tilted anteriorly[26] (Fig. 6*D*; and 8*E*). These changes can be recognized by observing the course of the roof, mesencephalic and pontine segments of the anterior ponto-mesencephalic vein which outline the chiasmatic, interpeduncular and pontine cisterns (Fig. 5*B*; 8*C*; and 12). The parenchymal portions of the *thalamo-perforating veins*[16] are often displaced forward, tilted and arched anteriorly (Fig. 5*B*). This is due to upward herniation of the brain stem or tumor infiltration into the mesencephalon and subthalamic area.

The *precentral cerebellar vein* which accompanies the precentral cerebellar branch of the superior cerebellar artery is displaced backward, usually maximally at its colliculo-central point (Fig. 6*C*; 8*C*; and 12). The fissural segment of the vein becomes therefore roughly parallel to the clivus, and the colliculo-central angle between the fissural (first) and anterior culminate (second) segments becomes more obtuse. Posterior displacement of the precentral cerebellar vein is associated with backward crowding of the culminate tributaries of the *superior vermian vein*. The supraculminate tributary approaches the straight sinus (Fig. 5*B*; and 12).

With widening of the brain stem from

BONE CHANGES IN CHRONIC ADULT GAUCHER'S DISEASE*

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GAUCHER'S disease is an uncommon metabolic disturbance in which abnormal lipids accumulate in reticuloendothelial cells. It is a hereditary disease believed to be transmitted as an autosomal recessive in the majority of cases. The typical involved cells, called Gaucher's cells, are reticuloendothelial, and are large, 20 to 80 μ in diameter. The cells are not found in the peripheral blood without using special staining techniques. The systems principally involved are the osteoarticular system and reticuloendothelial system; lymph nodes, liver and spleen. In the infantile form there is neurologic and respiratory involvement.

The disease was originally described by Gaucher in 1882, when he considered the disease a primary splenic epithelioma. Current concepts of the disease have since evolved.

CLINICAL FEATURES

The majority of adult cases have been reported in Jews, but all other ethnic groups and races may be affected. There is equal sex incidence. The age of onset varies from infancy to old age, cases involved being reported from 1 week to 79 years.

There are 2 forms of the disease. An acute infantile form affects one third of patients. The patient usually deteriorates rapidly and death occurs usually within the first year and a half. Skeletal changes are rarely, if ever, seen.

The chronic form presents chiefly with splenic, and to a lesser extent hepatic, enlargement as well as involvement of the skeletal system. The majority of patients show symptoms in the first decade. Vague systemic symptoms, abdominal fullness, and weight loss, occur along with conjunctival and skin pigmentation, and bleeding tendency. Pancytopenia, anemia, thrombocytopenia or leukopenia, are found in varying combinations in the majority of cases. Silverstein and Kelly, in a recently published series of 38 patients, found that 75 per cent had osseous lesions evident roentgenographically.[8] Of these patients, only 35 per cent had symptoms referable to their bone lesions. Their findings also suggest that anemia and splenectomy may contribute to the osseous changes in Gaucher's disease; anemia by stimulating the activity of more marrow space for invasion by Gaucher's disease, and splenectomy by eliminating a large reservoir for Gaucher's disease. Episodes of bone crises may also occur, with acute pain, tenderness and elevated temperature, which simulate osteomyelitis. Open drainage at these sites of bone infarction creates the hazard of a persistent draining sinus.

Diagnosis is usually established by demonstration of Gaucher's cells in the spleen or marrow. The serum acid phosphatase values may be elevated. Other laboratory values are usually within normal limits.

ROENTGEN FINDINGS

The most frequent sites of skeletal involvement are the femora, spine, hips, shoulders, the tubular bones, and pelvis. The lower extremities are more often involved than the upper. The ribs, sternum, scapulae and mandible may also be involved, as well as any bone in the body. Early reports indicated that the skull is not involved and it is still claimed that the skull is not involved as an isolated lesion. The skull may, however, be involved in multiosseous disease.

The basic processes of bone involvement

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side to side, the 1st or peduncular segment of the vein of the *lateral recess of the fourth ventricle* is laterally displaced, stretched and elongated. However, a brain stem tumor frequently involves the brachium pontis and restiform body and extends into the cerebellar hemisphere. As a result, displacement of this segment of the vein of the lateral recess laterally may be minimal. In fact, as seen in the Towne projection, it may be displaced *medially* and arched towards the midline (Fig. 5*D*; and 8*D*). In the lateral projection, the portion of the vein of the lateral recess below the brachium pontis and restiform body becomes stretched and displaced inferiorly in arcuate fashion. The rather acute angle, normally present in this projection between the peduncular and flocculer segments of the vein at the cerebello-ponto-medullary angle, is effaced and arched downward (Fig. 5*B*; 8*C*; and 12). The supratonsillar tributaries of the vein of the lateral recess are crowded and displaced posteriorly—unless the tonsil itself is involved by the tumor (Fig. 5*B*). The medial tonsillar vein, which often joins the vein of lateral recess at or near the tonsillar incisura, will be displaced markedly backward since it is located immediately behind the medulla oblongata (Fig. 5*B*; and 8*C*). Not infrequently, there is a vein visible on the posterior aspect of the medulla oblongata, the *vein of the restiform body*.[16] This vessel is the upward continuation of the *median posterior spinal vein* and will be displaced posteriorly and laterally with medullary involvement (Fig. 8*C*; and 13*B*). Similarly the *anterior medullary vein*, an upward continuation of the *anterior spinal vein*, may be evident and displaced forward (Fig. 8*C*; and 13*B*). The spinal veins may show displacement similar to the spinal arteries.

The superior and inferior retrotonsillar tributaries of the *inferior vermian vein*, which outline the posterior aspect of the tonsil, are displaced backward (Fig. 5*B*; 8*C*; 9*D*; 12; and 13*B*). The copular angle formed by the inferior vermian vein and its superior retrotonsillar tributary becomes more acute than normal. Since the cerebellar tonsil is often outlined by various venous tributaries, it can frequently be recognized that its longitudinal axis is tilted either forward of backward, depending upon the location of the main bulk of the tumor (Fig. 5*B*; 8*C*; and 13*B*).

The *lateral mesencephalic vein* and the *interbrachial vein* are displaced laterally. Anteroposterior displacement of these two veins depends on the location of the main mass of the tumor, *i.e.*, whether in front or behind the corresponding sulci.

When the brachium pontis is involved by tumor, the brachial tributary of the petrosal vein is arched upward and outward and separated from the vein of the lateral recess (Fig. 5, *B* and *D*). With widening of the mesencephalon, the posterior mesencephalic vein[14] and, in more marked cases, the adjacent but more laterally located mesencephalic portion of the *basal vein of Rosenthal* are displaced outward and upward (Fig. 5*B*; 6*C*; 7*B*; and 9, *D* and *E*).

PARADOXICAL DISPLACEMENTS

When there are marked nodular protrusions from the surface of the enlarged pons, the nature of the pontine tumor is almost always astrocytoma. The basilar artery is not displaced forward in these cases but often maintains a normal distance from the clivus (Fig. 9, *A* and *C*). It may appear straightened and taut (Fig. 6*A*). In some cases, it may be markedly posteriorly displaced (Fig. 13*A*). There may be a considerable discrepancy in the distance between the clivus and the midline anterior ponto-mesencephalic vein or basilar artery and between the clivus and the more laterally located pontine vessels. Not infrequently, transverse pontine veins are visualized and are located much closer to the clivus than the basilar artery (Fig. 6, *A* and *C*). In others, there may be a tumor blush or abnormal tumor circulation located anterior to the anterior ponto-mesencephalic vein which may maintain a normal distance from the clivus (Fig. 9*D*).

are similar to other diseases in which there is proliferation of bone marrow or infiltration of bone marrow with abnormal cells. There may be a loss of bone density, simulating osteoporosis. In common with other similar diseases, the spine is often involved. Gaucher's disease should be considered in the differential diagnosis of loss of bone density in the spine of young adults, along with sickle cell anemia, Cushing's disease, and steroid therapy. Bone trabeculae are resorbed and the remaining trabeculae stand out in relief to give a picture of an altered or coarsened bony trabecular pattern. A coarse, foam-like appearance may at times be seen. Reparative processes may reinforce this picture and lead to bone sclerosis.[10]

Isolated patchy destructive areas can occur in a geographic or moth-eaten pattern, which give a picture very similar to osteolytic metastases (Fig. 1). The margination of these lesions can vary. Localized

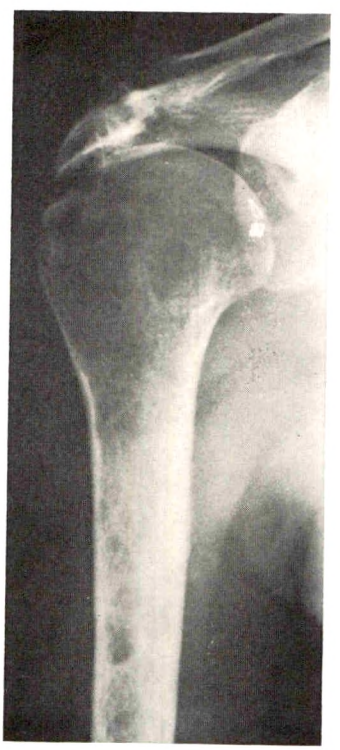


Fig. 1. Gaucher's disease, upper humerus; 77 year old female. Patchy, destructive osteolytic areas involving the humerus.

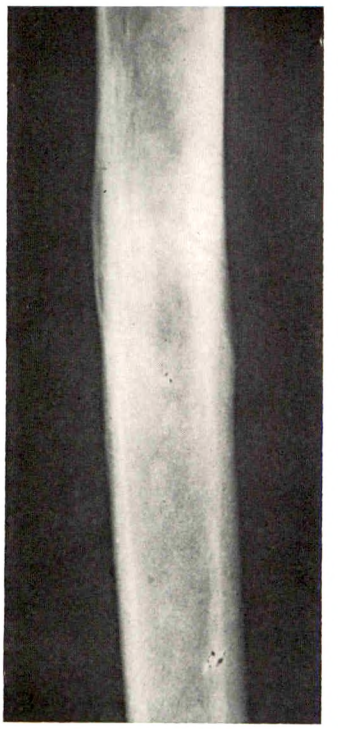


Fig. 2. Splitting of the cortex into several layers in the upper femoral shaft is noted, as well as cortical thickening and patchy sclerotic areas.

or patchy sclerotic areas can occur, some of which are due to bone infarction. Ischemic necrosis is one of the most common findings.

The lesions can progress to pathologic fracture, usually in the proximal femora, vertebrae, and ribs. These may occur in areas of normal appearing bone as well as in altered bone. There is ordinarily prompt callus formation and early union, although hip fractures may fail to unite. Narrowing of the joint space may occur, although cartilage is not primarily affected. The intervertebral spaces may be narrowed, but in our material, this was apparently due to herniation of the nucleus pulposus into a vertebral body. Bone infarctions may occur with a clinical and roentgenologic picture that would be very difficult to distinguish from osteomyelitis.

The cortex is involved in various ways. Destruction of the cortex may be seen, secondary to either infarction or involve-

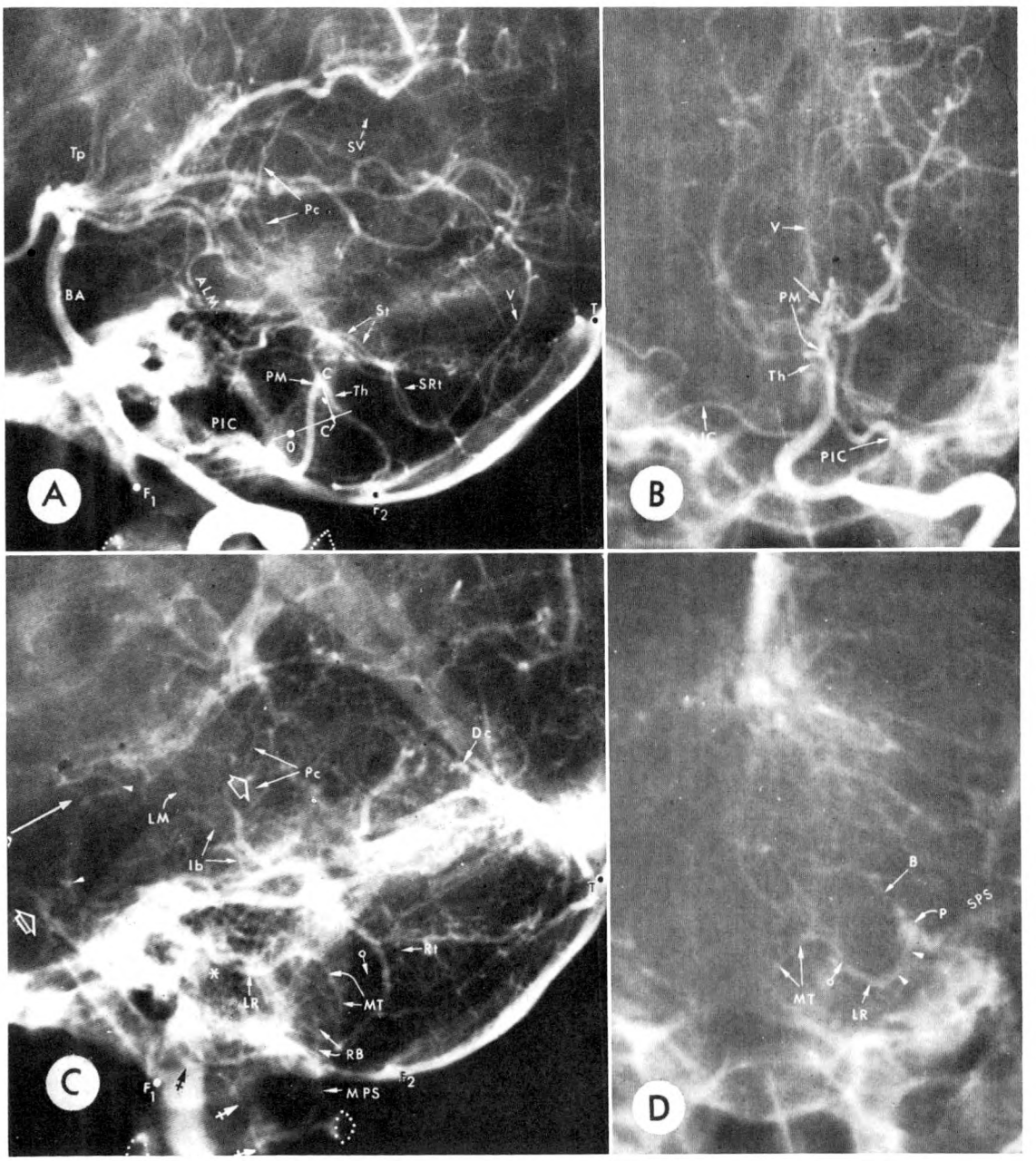


FIG. 8. Brain stem tumor with greatest involvement inferiorly.

(*A*) Left brachial angiogram, arterial phase, lateral view. The most striking findings are elongation of the proximal portion of the posterior inferior cerebellar artery (PIC) and posterior displacement of its posterior medullary segment (PM), especially in its upper portion. The posterior displacement of this segment may be confirmed by dropping a perpendicular from the choroidal point (C) to a line joining the anterior margin (F_1) of the foramen magnum to the torcular (T). The perpendicular should intercept (C′) this line (F_1T) on the average a fraction of a millimeter behind the junction of the anterior one-third with its posterior two-thirds (O). In this case, the distance (OC′) measures 8 mm. If this perpendicular line is continued further downward, it extends considerably behind the arch of the atlas. The superior pole of the tonsil is rather well outlined by multiple supratonsillar segments (St) and the superior retrotonsillar segment (SRt). The tonsil is not narrowed (probable tumor infiltration) but appears tilted so that its lower

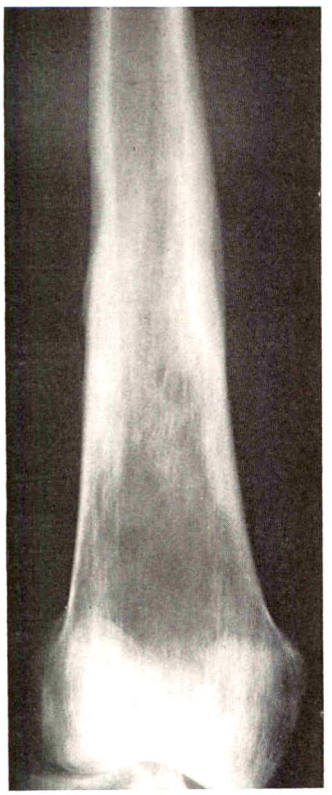


Fig. 3. Tomogram of distal femur. Thickening of the cortex can be seen, as well as patchy areas of medullary sclerosis.

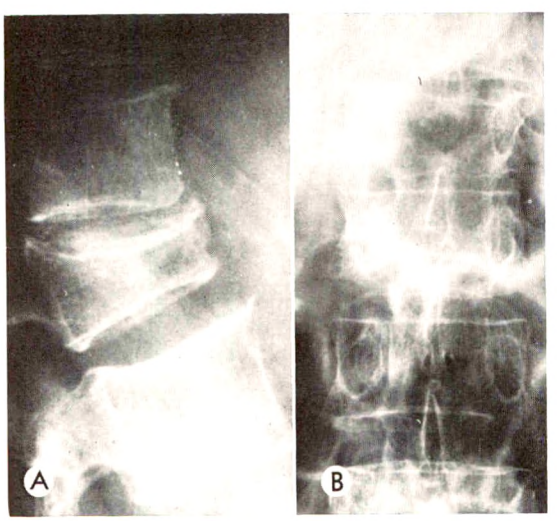


Fig. 4. (*A*) Lateral view showing wedge-like collapse of the first lumbar vertebral body. The intervertebral spaces are preserved. (*B*) Frontal view showing loss of bone density, which is generalized in the spine.

ment with the primary disease process. Thinning of the cortex is not uncommon. Scalloping of the inner margin of the cortex may also be seen. The cortex may be split, with an inner layer of new bone formation, which does not fuse with the cortex, giving a "bone within a bone" appearance (Fig. 2). This finding is also commonly seen in sickle cell anemia. The cortex may also be thickened (Fig. 3).

The periosteum may react with a solid or a lace-like type of periosteal new bone formation, particularly in areas of ischemic necrosis, where buttressing is commonly present, or owing to subperiosteal hemorrhage or pathologic fracture.

There may be expansion of bone, particularly with the characteristic "Erlenmeyer flask" type of deformity, or expansile trabeculated lesions of the long bones.

The skull is rarely involved, but may show a mottled destruction of the calvarium. Destructive lesions and cyst-like areas in the mandible are at times seen. The spine ordinarily shows a generalized loss of bone density. One or more of the vertebrae may collapse, giving a wedge-like deformity (Fig. 4, *A* and *B*) or a wafer-like deformity. Sclerosis of the collapsed vertebra may occur or the collapsed vertebra may appear osteopenic. In addition, herniation of the nucleus pulposus into a verte-

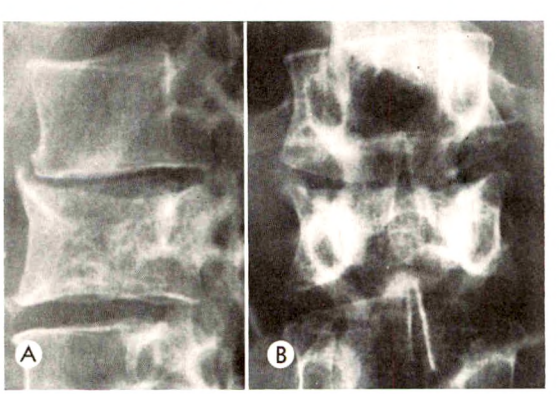


Fig. 5. (*A*) Lateral view. The intervertebral space between L1 and L2 is narrowed, with herniation into the body of L2, sclerosis and osteophyte formation. (*B*) Frontal view showing sclerosis of the second vertebral body.

QUADRIGEMINAL PLATE TUMORS

Strictly speaking, tumors of the quadrigeminal plate are included under brain stem tumors, although clinical and angiographic features differ considerably from pontine lesions (Fig. 14). Such tumors encroach posteriorly upon the quadrigeminal cistern and cistern of the precentral cerebellar fissure and superiorly upon the cistern of the vein of Galen and posterior part of the third ventricle. The most informative angiographic feature is posterior displacement of the precentral cerebellar vein which shows a localized posteriorly convex arcuate deformity (Fig. 14*B*). Similarly, the anterior culminate segment and precentral cerebellar branches of the superior cerebellar artery are displaced backward in arcuate fashion. The anterior superior marginal segments of the superior cerebellar arteries are, however, depressed (Fig. 14*A*). The *superior thalamic vein,* which originates from the medial superior part of the thalamus about 1 cm. behind the foramen of Monro, runs backward parallel, inferior and lateral to the internal cerebral vein to join the posterior part of the internal cerebral vein, or the basal vein of Rosenthal, or the vein of Galen. In mesencephalic tumors, especially with primary involvement of the tectum, pretectum and adjacent tegmentum, the posterior portions of the internal

pole is further backward than its upper pole. The tonsillo-hemispheric branch (Th) is displaced posteriorly and shows a broad sweep around the tonsil. The basilar artery (BA) and the distal part of the vertebral artery are displaced forward and downward. The thalamo-perforating branches (Tp) are prominent but have a normal configuration. The precentral cerebellar artery (Pc) is slightly displaced posteriorly and the distance between this vessel and the basilar artery is larger than normal. The vermian branch (SV) of the superior cerebellar artery is elevated. The vermian branch (V) of the posterior inferior cerebellar artery is displaced slightly backward. The anterior lateral marginal branch (ALM) of the superior cerebellar artery and the posterior margin (F_2) of the foramen magnum are also labeled. The anterior and posterior arches of C_1 are retouched.

(*B*) Arterial phase, Towne view. The posterior inferior cerebellar artery (PIC) on the left side shows a tortuous course. Its proximal portion extends slightly more laterally than is ordinarily seen. The significance of this finding is difficult to evaluate because of marked normal variations. The posterior medullary (PM) and supratonsillar segments (unlabeled arrow) are not laterally displaced. The vermian branch (V) is also normally located. The posterior medullary segment (Th) of the tonsillo-hemispheric branch is, however, slightly shifted toward the right. An anterior inferior cerebellar artery (AIC) is well seen on the right side and is unremarkable.

(*C*) Venous phase, lateral view. The anterior ponto-mesencephalic vein (small arrowheads) is elevated upward and forward with the interpeduncular cistern (unlabeled long arrow) narrowed from below. The pontine segment is close to the clivus (⏢). The precentral cerebellar vein (Pc) including its colliculo-central point (⏢) is displaced backward. The interbrachial vein (Ib), continuous with the lateral mesencephalic vein (LM), is displaced markedly upward and backward. The medial tonsillar tributary (MT) of the vein of the lateral recess of the fourth ventricle (LR) is displaced markedly posteriorly. The peduncular portion (LR) of the vein is elongated and arched inferiorly. The usual acute angle at the junction of the peduncular and floccular segments of the vein in the cerebello-ponto-medullary angle (*) is blunted. The retrotonsillar vein (Rt) is displaced slightly backward. The upper cervical cord is outlined by veins both anteriorly and posteriorly. The median posterior spinal vein (MPS) as it extends upward becomes the vein of the restiform body (RB) which is also displaced markedly posteriorly. The anterior medullary vein (black arrow, ↕), the upward continuation of the anterior spinal vein (↕), bulges forward. The vein running on the superior aspect of the declive (Dc) is unusually close to the straight sinus. The torcular (T) and the anterior and posterior margins (F_1, F_2) of the foramen magnum are also labeled.

(*D*) Venous phase, Towne view. The medial tonsillar tributary (MT) of the vein of the lateral recess of the fourth ventricle (LR, arrowheads) is displaced slightly medially and arched downward. The brachium pontis and restiform body are widened as indicated by the downward and medial displacement of the peduncular portion (↕) of the vein of the lateral recess. Upward displacement of the brachial tributary (B) of the petrosal vein (P) is minimal. The superior petrosal sinus (SPS) is also labeled.

bral body may occur, although the appearance of the spine with multiple biconcavities is not seen, such as is characteristic of sickle cell anemia (Fig. 5, *A* and *B*). Osteosclerotic changes in the spine may also be present. Gibbus formation is rare. Subchondral radiolucent lines may be present, owing to demineralization (Fig. 6).

One area that has not been emphasized in the recent literature is involvement of the sacroiliac joints. Marginal sclerosis may be present and actual obliteration of the sacroiliac joint can be seen. Two cases in our material had this finding (Fig. 7, *A–C*; and 8, *A* and *B*).

The pelvis may show an alteration of bone texture and also sclerotic changes. The symphysis pubis can also be involved (Fig. 9). The most consistent finding in this area is ischemic necrosis of the femoral head (Fig. 10).

Ischemic necrosis of the femoral head can occur in a wide variety of conditions. The process is basically similar from most etiologies. Initially, there is subchondral sclerosis, extending to a large sclerotic area involving the major portion of the femoral head, the so-called "snow-cap" appearance. Structural failure then occurs, initiated by a radiolucent crescentic subchondral fracture line, at the point of greatest stress. Collapse of the femoral head then follows, with irregularity of contour and patchy sclerotic and radiolucent areas. The cartilage in Gaucher's disease

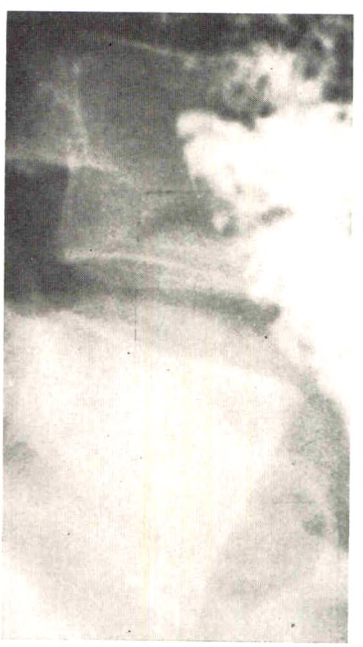


Fig. 6. Lower lumbar spine. Detail showing loss of bone density with radiolucent lines paralleling the endplates.

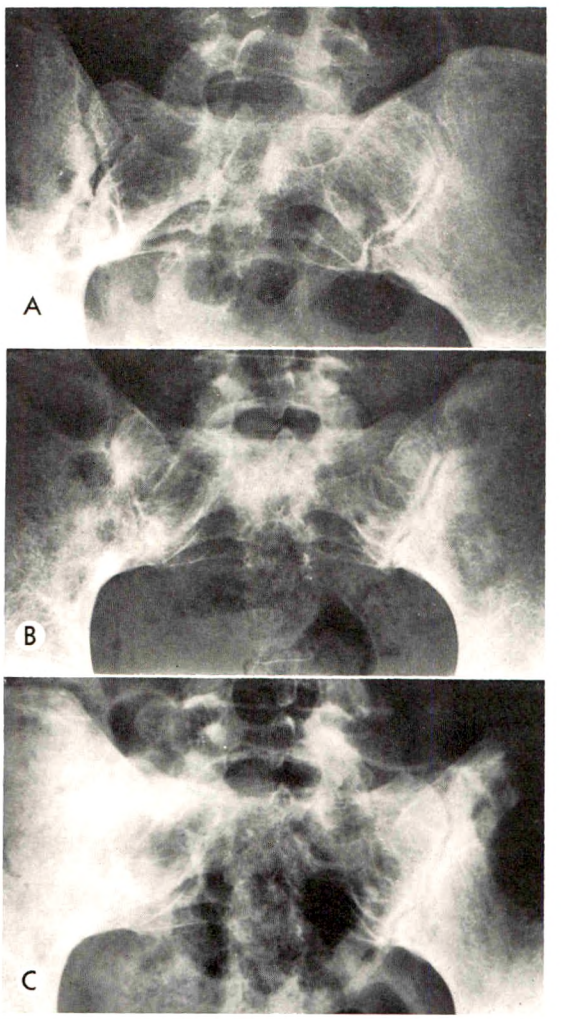


Fig. 7. Gaucher's disease showing progressive sacroiliac joint involvement. Female, aged 27 years. (*A*) Initial roentgenogram taken in 1965, showing both sacroiliac joints to be intact. (*B*) Roentgenogram taken in 1967, showing partial obliteration of the right sacroiliac joint, with 2 distinct areas of sclerosis. The left sacroiliac joint is intact. (*C*) Roentgenogram taken in 1970, showing complete obliteration of the right sacroiliac joint. The left sacroiliac joint remains intact.

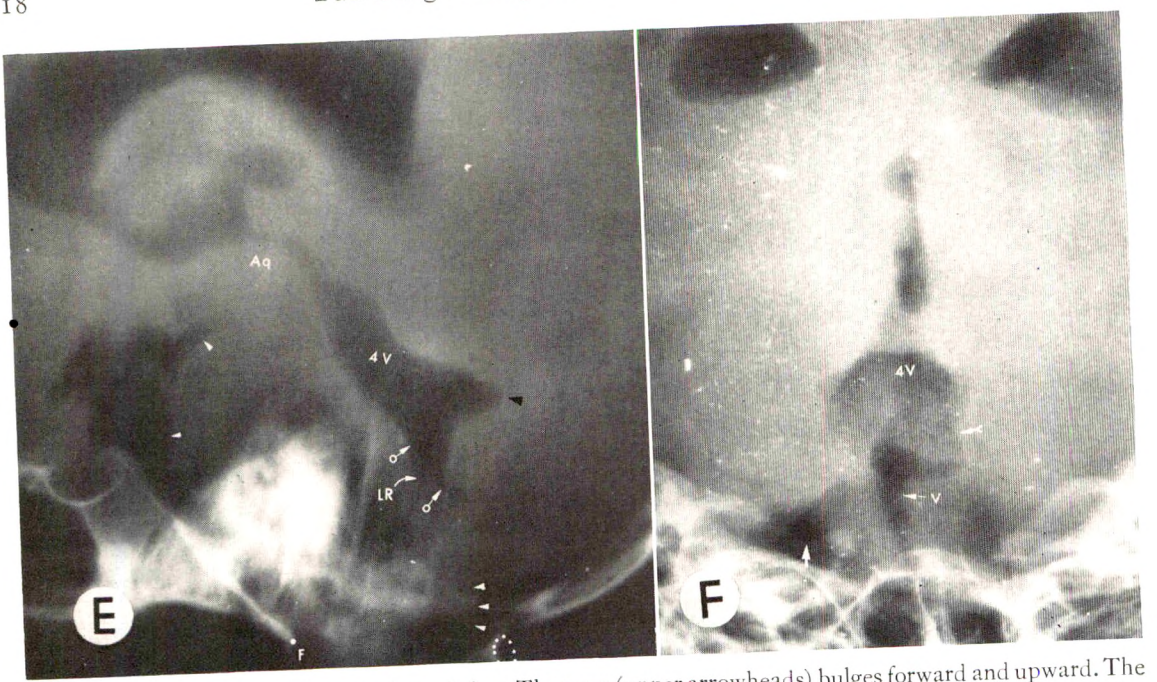


FIG. 8. (*E*) Pneumoencephalogram, lateral view. The pons (upper arrowheads) bulges forward and upward. The deep portion of the interpeduncular cistern is encroached upon from below. The most marked posterior widening of the brain stem is inferiorly located. The floor ( ) of the fourth ventricle (4V) is displaced backward. The lateral recess of the fourth ventricle (LR) can be identified and is displaced downward due to the enlarged brachium pontis and restiform body. The fourth ventricle is dilated presumably due to partial obstruction of outlets of the fourth ventricle. The height of the fourth ventricle (floor of the posterior superior recess, black arrowhead) is somewhat greater than normal. Marked posterior bulging of the medulla oblongata (lower arrowheads) is well demonstrated and corresponds to the course of the vein of the restiform body as seen in Figure 8*C*. The anterior margin of the foramen magnum (F) is also indicated. The posterior tubercle of C_1 is retouched.

(*F*) Pneumoencephalogram, reverse Towne view. The left lateral border ( ) of the fourth ventricle (4V) is flattened. The vallecula (V) is displaced toward the right by the enlarged tonsil. The lateral pontine cistern is well filled on the right side ( ) but poorly on the left. These findings also indicate that the lower brain stem is maximally involved by tumor which has extended into the left cerebellar hemisphere.

cerebral veins and superior thalamic veins are elevated (Fig. 14*B*). The pretectal segments of the medial posterior choroidal arteries are also elevated and their tectal segments are displaced outward. Often posterior portions of the basal veins of Rosenthal and posterior mesencephalic veins are elevated and displaced outward. The posterior thalamo-perforating vessels, arteries and veins are stretched and arched forward. The roof and mesencephalic segments of the anterior ponto-mesencephalic vein and the distal end of the basilar artery are not infrequently displaced forward and downward.

DIFFERENTIAL DIAGNOSIS OF BRAIN STEM FROM FOURTH VENTRICLE TUMORS

Both brain stem tumors and fourth ventricle tumors are located in the midline and produce relatively symmetric vascular displacements. In both instances, vessels located on the anterior aspect of the brain stem are displaced forward, vessels located behind the roof of the fourth ventricle are displaced backward, and vessels lateral to the brain stem and fourth ventricle are displaced laterally. Differential features must be based on recognizing landmarks related

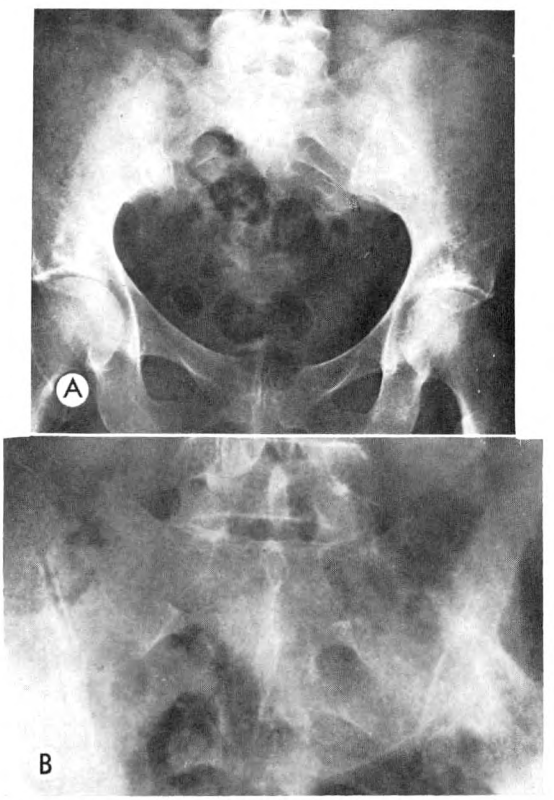


FIG. 8. Gaucher's disease with unilateral involvement of a sacroiliac joint. (*A*) Roentgenogram taken in 1961 shows partial obliteration of the left sacroiliac joint, with surrounding sclerotic change. The right sacroiliac joint is intact. (*B*) Roentgenogram taken in 1966 showing no significant change. Obliteration of the left sacroiliac joint is present, with an intact right sacroiliac joint remaining.

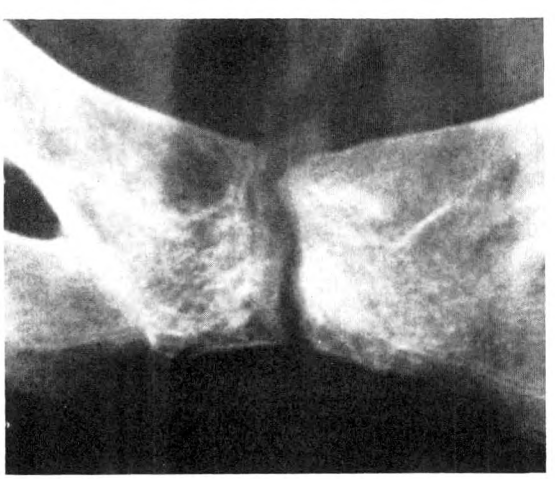


FIG. 9. Involvement of the symphysis pubis, with sclerosis and distortion; however, the symphysis is not fused.

is not primarily involved; however, there can be secondary osteoarthritic changes (Fig. 11). Periosteal buttressing along the inferior aspect of the involved site may also be present. Collapse and deformity of subchondral bone in other areas may also occur.

The distal femora show the most widely popularized aspects of bone involvement in this disease. There is usually bilateral involvement, although unilateral involvement may occur. The medial border of the distal femur is at first straightened and later assumes a convex contour. Expansion then proceeds to the typical "Erlenmeyer flask" deformity (Fig. 12). Expanding lesions may also be seen in the mandible, with a cyst-like expansile lesion surrounding the teeth, but not involving the periodontal membrane. Expansile lesions in the ribs, as well as destructive areas with pathologic fractures, may be present (Fig. 13).

The shoulders likewise may be involved, with ischemic necrosis similar to femoral head involvement. The phalanges may rarely be involved late in the disease process, with coarsened trabeculation. The



FIG. 10. Ischemic necrosis of the femoral head in a 27 year old female with Gaucher's disease. The femoral head shows irregularity of contour with several destructive areas and several sclerotic areas. Periosteal buttressing along the femoral neck is also seen. The joint space remains intact.

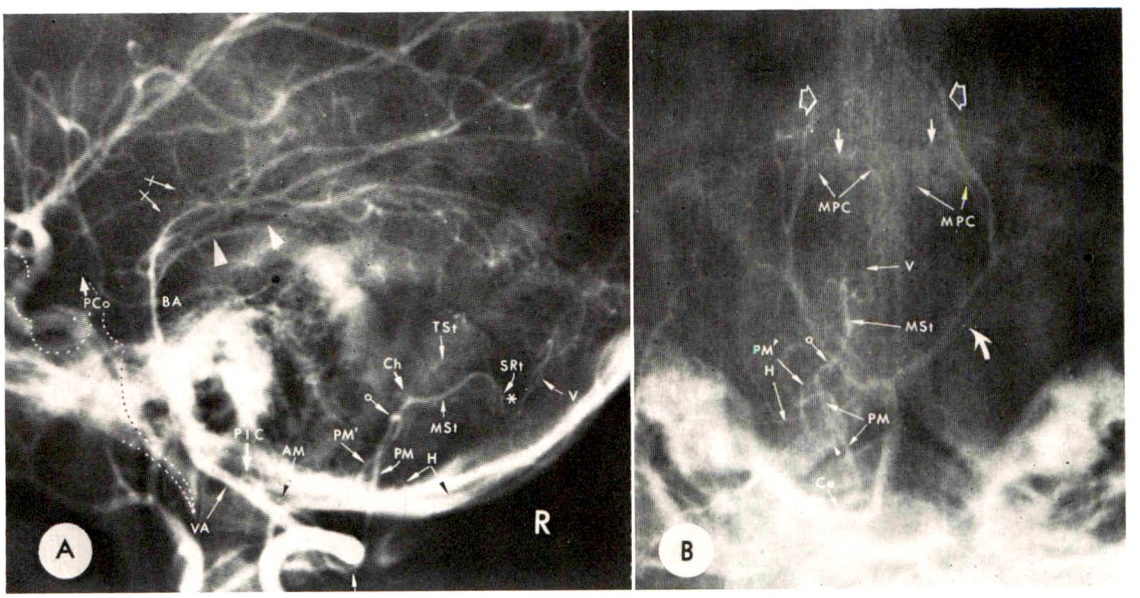


Fig. 9. Extensive brain stem astrocytoma.

(*A*) *Right* retrograde brachial angiogram, arterial phase, lateral view. The distal portion of the vertebral artery (VA) is displaced downward and forward and has an inferior convexity. The basilar artery (BA) is stretched, arched and appears taut. The proximal portions of the posterior cerebral and superior cerebellar arteries are stretched and elevated (large arrowheads). The distal branches of the posterior cerebral arteries are also stretched as a result of obstructive hydrocephalus. The anterior medullary segment (AM) of the posterior inferior cerebellar artery (PIC) is displaced downward. It takes a tortuous course and dips down into the upper part of the cervical canal (arrow, lower mid margin). The posterior medullary segment (PM) is markedly posteriorly displaced, particularly in the region of the choroidal point. At this point, a faint blush (Ch) due to choroid plexus can be identified. The superior pole of the cerebellar tonsil is outlined by a prominent medial supratonsillar segment (MSt), a faintly visualized transverse supratonsillar branch (TSt) and the superior retrotonsillar segment (SRt). The cerebellar tonsil appears widened and is also tilted posteriorly. The copula pyramidis (*) is compressed from in front. The vermian segment (V) is displaced backward. The tonsillo-hemispheric branch arises ( ) proximal to the choroidal point. The posterior medullary segment (PM′) of this branch is tortuous but also displaced posteriorly in its upper part. The hemispheric branch (H) is unusually close to and partly obscured by the base of the skull. There is considerable distance between the basilar artery and the posterior aspect of the clivus (retouched). The thalamoperforating arteries ( ) are displaced forward. The posterior communicating artery (PCo) is markedly stretched and straightened.

(*B*) Right brachial angiogram, arterial phase, Towne view. The right carotid artery was manually compressed in the neck to avoid superimposition of vessels. The proximal portions of the posterior cerebral, superior cerebellar and medial posterior choroidal arteries are displaced medially (curved arrow) on both sides. This is surprising since these vessels are also displaced upward (Fig. 9*A*), excluding uncal herniation as the cause of medial displacement. This paradoxical finding is probably due to tumor growth of pseudo-extra-axial type, *i.e.*, nodules protruding from the anterior part of the upper brain stem. More posteriorly, these vessels are separated indicating diffuse widening of the posterior aspect of the upper brain stem. This is well demonstrated by tracing the medial posterior choroidal arteries (MPC) posteriorly to their tectal segments ( ) which are markedly separated. More posteriorly, the posterior cerebral arteries ( ) are also separated from each other, presumably due to upward herniation of posterior fossa contents through a wide posterior tentorial notch. Various segments of the posterior inferior cerebellar artery, including its cervical (Ce), posterior medullary (PM), medial supratonsillar (MSt) and vermian segment (V) can be visualized and do not appear remarkable. The posterior medullary (PM′) and hemispheric segments (H) of the tonsillo-hemispheric branch originating ( ) from the distal part of the posterior inferior cerebellar artery can be identified and are not displaced laterally.

spleen is usually enlarged. The liver may be enlarged to a lesser degree. Gallstones have also been reported.[5]

DIFFERENTIAL DIAGNOSIS

The basic processes of bone involvement, as seen roentgenographically, are not specific to Gaucher's disease. Loss of bone density, particularly in the spine, can occur in a wide variety of diseases, including osteoporosis, metabolic bone disease, hematologic diseases, multiple myeloma and excess steroid therapy. The vertebral bodies do not show the cupping that is pathognomonic of sickle cell anemia. In steroid osteoporosis a characteristic finding is excess callus formation at the site of collapse of the bodies, a finding which does not occur in Gaucher's disease.

The skeletal involvement may progress to a predominantly sclerotic pattern, either patchy or more generalized. Differentiation from osteoblastic metastasis, osteopetrosis, mastocytosis, tuberous sclerosis, Hodgkin's disease, and myelofibrosis would be required. Myelofibrosis may present some difficulty, as enlargement of the spleen, in

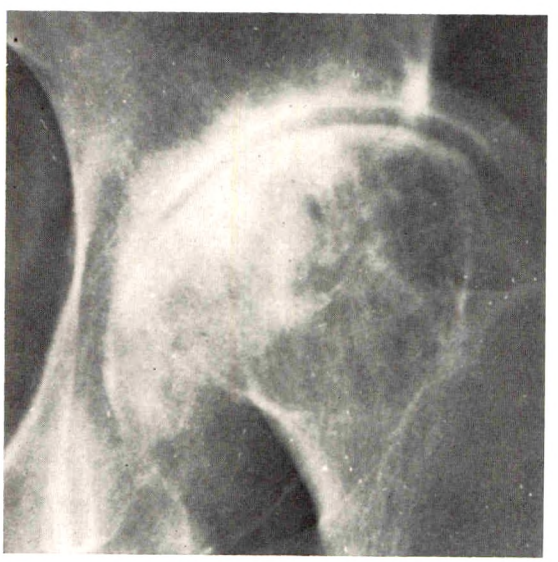


FIG. 11. Ischemic necrosis of the femoral head with loss of contour and patchy destructive and sclerotic areas. There has been secondary narrowing of the joint space, with reactive hypertrophic changes of the acetabulum.



FIG. 12. Lack of constriction or modeling of the distal femora and proximal tibiae. This is the so-called "Erlenmeyer flask" deformity.

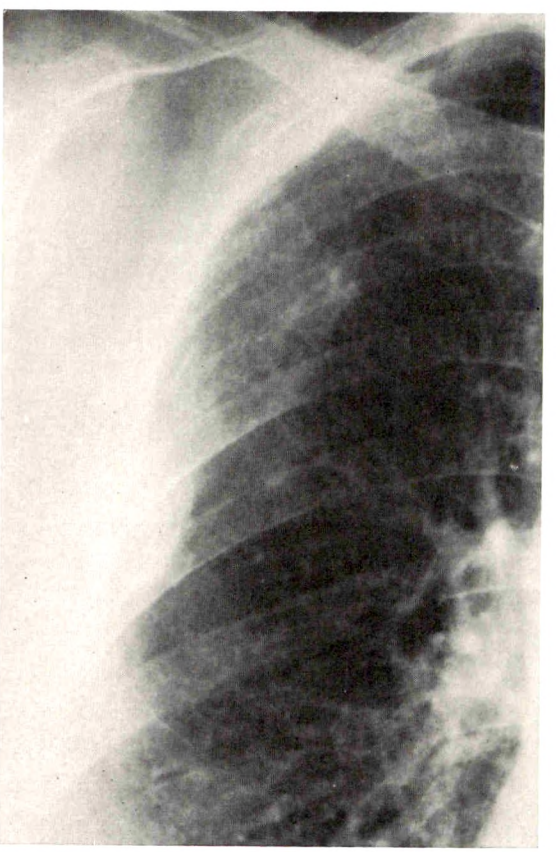


FIG. 13. Destruction of the ribs with pathologic fractures and an extrapleural sign.

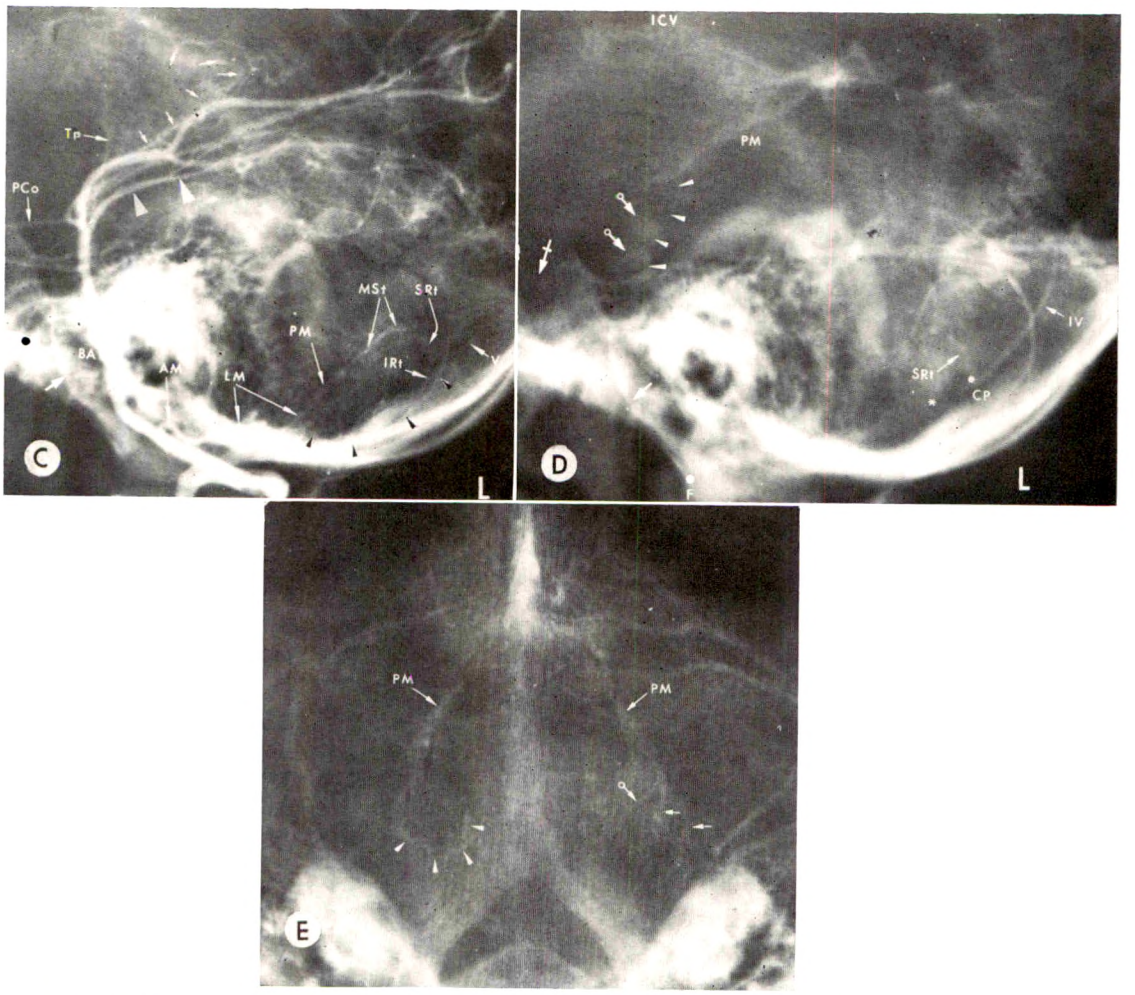


FIG. 9. (*C*) *Left* retrograde brachial angiogram, arterial phase, lateral view. Bone changes involving the clivus (↑), separation of the basilar artery (BA) from the clivus, stretching of the posterior communicating artery (PCo), upward and forward displacement of thalamo-perforating arteries (Tp), and elevation and stretching of the posterior cerebral and superior cerebellar arteries (large arrowheads) seen in Figure 9*A* are again noted. The medial posterior choroidal artery (row of unlabeled small arrows) is rather well seen. Its circumpeduncular portion is elevated while its tectal portion shows a more acute angle as it turns forward. The anterior medullary (AM) and lateral medullary segments (LM) are stretched. The posterior medullary segment (PM) appears more posteriorly displaced and tilted than that on the right side (Fig. 9*A*). The medial supratonsillar segment (MSt), superior retrotonsillar segment (SRt), the inferior retrotonsillar branch (IRt) and vermian segment (V) are posteriorly displaced. The hemispheric branch (black arrowheads) of the posterior inferior cerebellar artery is unusually close to the occipital bone.

(*D*) Left brachial angiogram, venous phase, lateral view. A large eroded sella (↕) and scalloping of the clivus (↑) are again noted. A portion of the anterior ponto-mesencephalic vein (row of arrowheads) can be faintly identified and corresponds exactly to the position of the basilar artery seen in the arterial phase (Fig. 9*A*). It is considerably separated from the clivus. A faint tumor blush (⇕) can be seen immediately anterior to the anterior ponto-mesencephalic vein. The posterior portion of the posterior mesencephalic vein (PM) is slightly elevated. The superior retrotonsillar tributary (SRt) of the inferior vermian vein (IV) is displaced markedly backward as is the copular point (CP). The normal location of the copular point (*), *i.e.*, 4 mm. below and behind the mid-point of the line connecting the anterior margin (F) of the foramen magnum to the torcular, is indicated. In this case, the copular point (CP) is located 9 mm. behind the expected position. The internal cerebral vein (ICV) is somewhat flattened anteriorly.

(*E*) Left brachial angiogram, venous phase, Towne view. The posterior mesencephalic veins (PM) are laterally displaced and separated from each other. Like the posterior cerebral arteries, one (⇕) of the peduncular veins is displaced medially and backward but others (▲, ↑) are displaced anteriorly and laterally.

addition to generalized bony lesions, is common to both. "Erlenmeyer flask" deformity and ischemic necrosis do not occur in myelofibrosis. Extramedullary hematopoiesis, which may occur in myelofibrosis, is not expected in Gaucher's disease. Hodgkin's disease would also not show ischemic necrosis and "Erlenmeyer flask" deformities. The "bone within a bone" appearance in osteopetrosis differs from that of Gaucher's disease.

In osteopetrosis there is symmetric condensation of increased bone density paralleling the metaphysis and the iliac crests, as well as the vertebral endplates. In Gaucher's disease there is a split layer of cortex which may involve more than one area, but is not symmetric.

Coarsening of the bony textural pattern may occur in a wide variety of diseases, including the congenital anemias, hyperparathyroidism, and myelomatosis. The short tubular bones of the hands and feet are less severely involved than in Cooley's anemia. The characteristic subperiosteal changes of hyperparathyroidism are not present in Gaucher's disease, and the other bone changes described in Gaucher's disease are not present in myelomatosis. Altered bony texture, or areas of sclerosis, in association with an "Erlenmeyer flask" deformity may also be present in fibrous dysplasia, and this may lead to some difficulties of diagnosis. However, ischemic necrosis, generalized diffuse loss of bone density and splenomegaly are not present in fibrous dysplasia. "Erlenmeyer flask" deformity can be seen in a very wide range of diseases, the classic being Pyle's disease. This can be the result of generalized disease, or toxic or traumatic injuries.[3]

Ischemic necrosis, particularly of the hips, occurs in very many conditions. Fifty-two various etiologies for a fragmented appearance of the femoral head have been summarized by the author.[3] It is a very common event in association with Gaucher's disease.

Splitting of the cortex, with formation of a distinctive inner layer, is also commonly seen in sickle cell anemia.

Patchy destructive changes, sometimes associated with vertebral collapse, are common and these must be differentiated from osteolytic metastasis.

Involvement of the sacroiliac joints, with marginal sclerosis and progressive obliteration of the joint space, was evident in our material and has also previously been described,[4] although not emphasized, in the recent literature on Gaucher's disease. This finding must be differentiated from ankylosing spondylitis and tuberculosis involving the sacroiliac joint. Ankylosing spondylitis usually shows bilateral sacroiliac joint obliteration, osteoporosis of the spine, squaring of the anterior aspects of the vertebral bodies, and calcification of the annulus fibrosus and the paraspinal ligaments. None of these changes occur in Gaucher's disease, except loss of bone density of the spine. Tuberculosis in the sacroiliac joint can be expected to present with a more destructive appearance.

The bone that is the key to the diagnosis of this disease is the femur, with ischemic necrosis of the proximal end and an "Erlenmeyer flask" deformity at the distal end.

SUMMARY

The roentgen bone changes of chronic adult Gaucher's disease are presented. These include osteopenia, osteosclerosis, bone destruction, pathologic fracture, ischemic necrosis, and cortical changes. Obliteration of the sacroiliac joint space, a finding not emphasized in the recent literature, is demonstrated in 2 cases.

The differential diagnosis is discussed.

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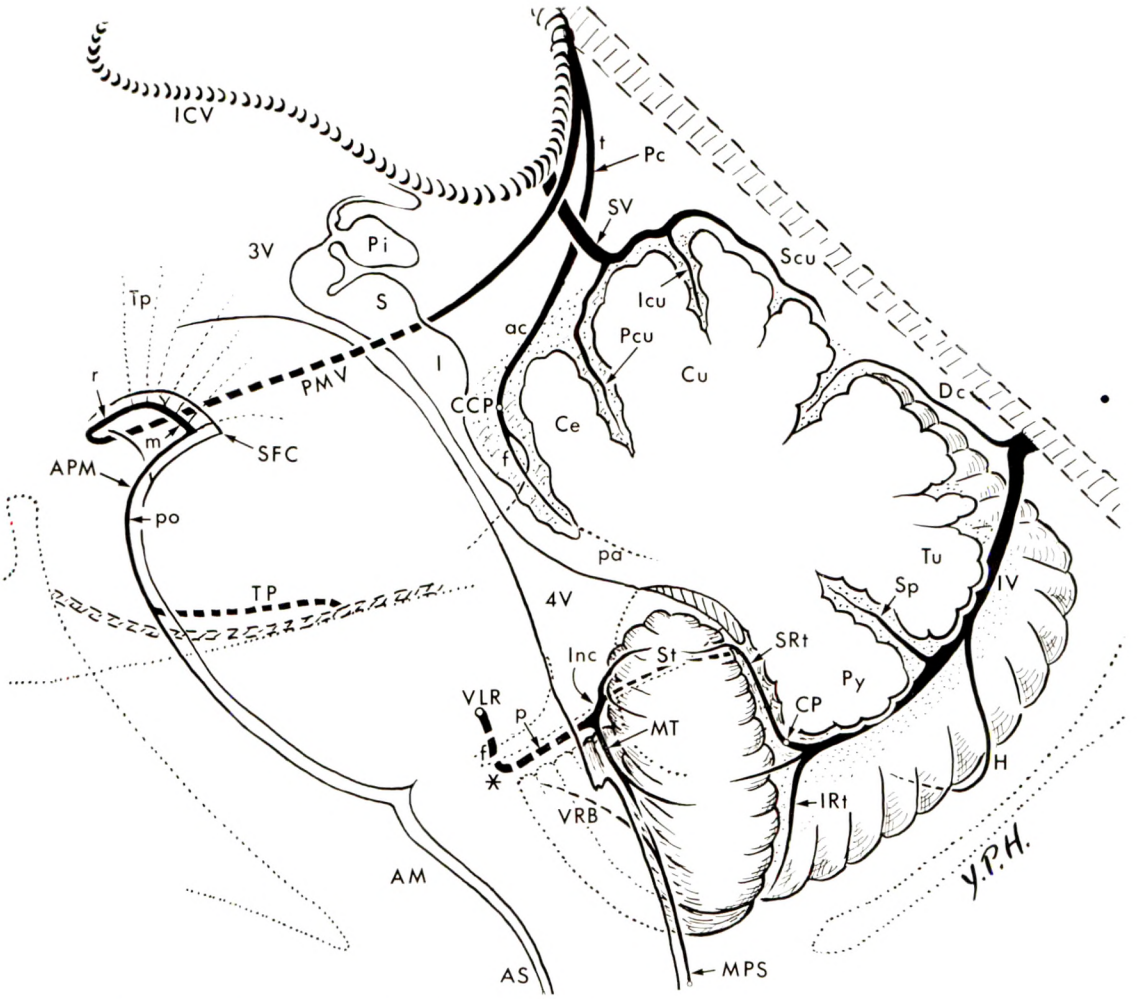


FIG. 10. Diagrammatic representation of "mid-sagittal section" showing normal courses of the posterior fossa veins important in the diagnosis of brain stem tumors.

The labeled structures include the anterior culminate segment (ac), anterior medullary vein (AM), anterior ponto-mesencephalic vein (APM), anterior spinal vein, (AS), central lobule (Ce), colliculo-central point (CCP), copular point (CP), culmen (Cu), declival vein (Dc), fissural segment (f), hemispheric vein (H), inferior colliculus (I), intraculminate vein (Icu), internal cerebral vein (ICV), incisura (Inc), inferior retrotonsillar tributary (IRt), inferior vermian vein (IV), mesencephalic segment (m), median posterior spinal vein (MPS), medial tonsillar vein (MT), peduncular segment (p), parenchymal segment (pa), precentral cerebellar vein (Pc), preculminate vein (Pcu), pineal gland (Pi), posterior mesencephalic vein (PMV), pontine segment (po), pyramid (Py), roof segment (r), superior colliculus (S), supraculminate vein (Scu), superior foramen caecum (SFC), suprapyramidal vein (Sp), superior retrotonsillar vein (SRt), supratonsillar vein (St), superior vermian vein (SV), terminal segment (t), thalamo-perforating veins (Tp), transverse pontine vein (TP), tuber (Tu), vein of the lateral recess of fourth ventricle (VLR), vein of the restiform body (VRB). The third and fourth ventricle (3V, 4V) and cerebello-ponto-medullary angle (*) are also indicated.

Note that the roof (r), mesencephalic (m) and pontine segments (po) of the anterior ponto-mesencephalic vein (APM) outline the size and configuration of the interpeduncular and pontine cisterns. The fissural (f) and parenchymal portions (pa) of the precentral cerebellar vein (Pc) are located immediately behind the inferior margin of the inferior colliculi (I) and the roof of the upper portion of the fourth ventricle. The superior vermis is outlined by the precentral cerebellar vein and tributaries of the superior vermian vein (SV). The supraculminate tributary (Scu) runs around the apex of the cerebellum. The distance between this vessel and the straight sinus indicates the size of the superior cerebellar cistern.

The veins related to the posterior aspect of the lower brain stem include the median posterior spinal vein (MPS), the vein of the restiform body (VRB) and the medial tonsillar tributary (MT) of the vein of the lateral recess of the fourth ventricle (VLR). The supratonsillar (St), superior and inferior retrotonsillar tributaries (SRt, IRt), and medial tonsillar vein (MT) outline the body of the cerebellar tonsil. Its antero-posterior diameter corresponds to the height of the fourth ventricle, specifically its posterior superior recess. The peduncular portion of the vein of the lateral recess is related to the posterolateral aspect of the brain stem although separated from it by the lateral recess. Note the rather acute angle at the junction of the peduncular (p) and floccular (f) segments of the vein of the lateral recess in the region of the cerebello-ponto-medullary angle (*).

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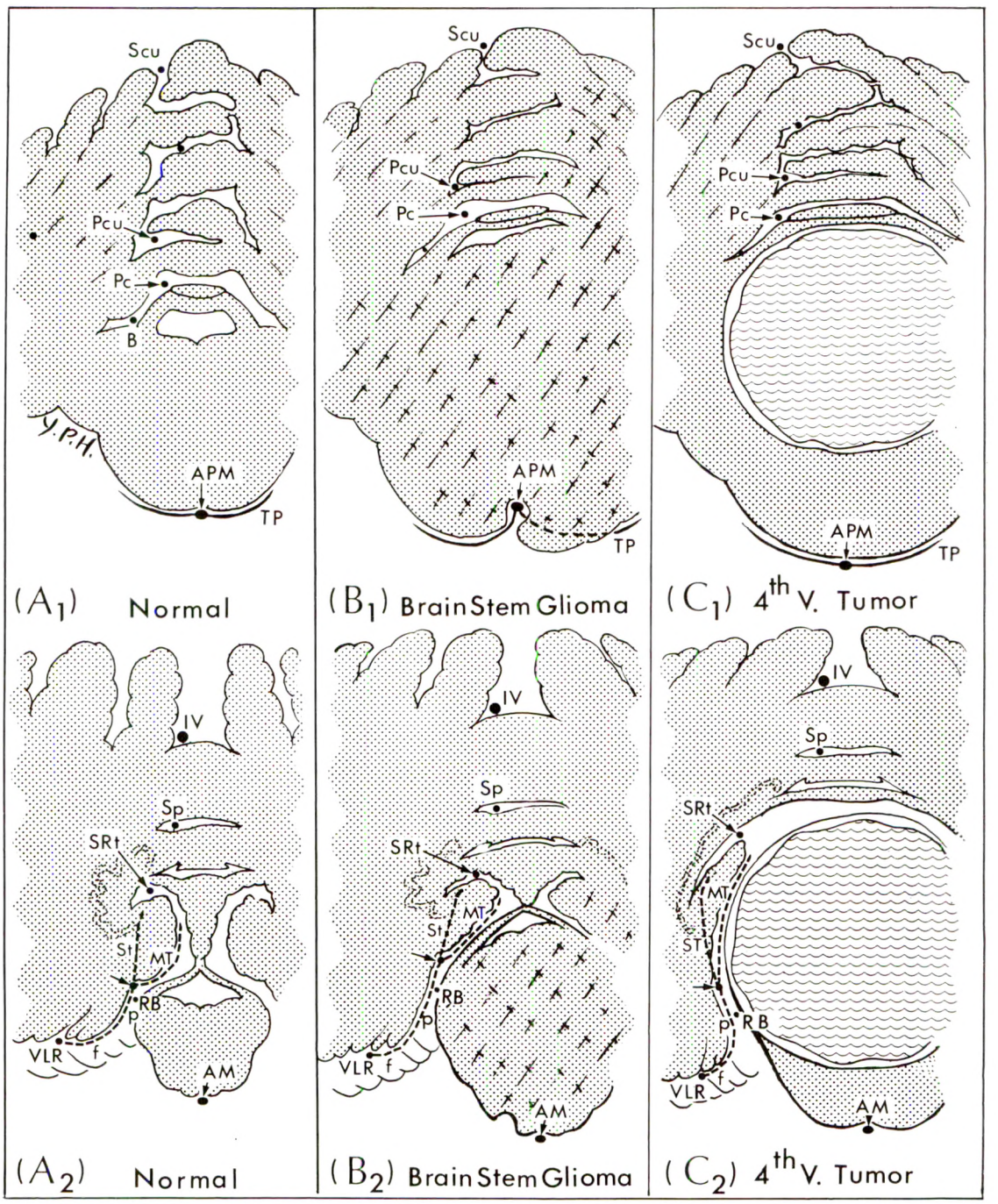


FIG. 11. Diagrammatic horizontal sections of the cerebellum and brain stem showing anatomic relationship of the posterior fossa veins in normal cases, in brain stem tumors, and in fourth ventricle tumors.

The labeled structures include the anterior medullary vein (AM), anterior ponto-mesencephalic vein (APM), brachial tributary (B) of the precentral cerebellar vein, floccular segment (f), inferior vermian vein (IV), medial tonsillar vein (MT), peduncular segment (p), precentral cerebellar vein (Pc), preculminate vein (Pcu), vein of the restiform body (RB), supraculminate vein (Scu), suprapyramidal vein (Sp), superior retrotonsillar vein (SRt), supratonsillar vein (St), transverse pontine vein (TP) and the vein of the lateral recess of the fourth ventricle (VLR).

GASTRIC TUBERCULOSIS*

REPORT OF A CASE WITH DISCUSSION OF ANGIOGRAPHIC FINDINGS

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DESPITE the ubiquitous presence of tuberculosis, it is surprising that gastric involvement is so rarely[3,4,5,6,7,11] seen even at autopsy, where the reported incidence is 0.16 per cent.[5]

A case of gastric tuberculosis is reported and its roentgenologic evaluation including angiography is presented to acquaint radiologists with this entity.

REPORT OF A CASE

D.S. (#B.H. 19-68-55) is a 32 year old Negro male with the chief complaints of cough, productive of thick greenish sputum, and fever and chills of 5 days' duration. He had nausea, vomiting and anorexia without hematemesis. No history of tuberculosis or previous abdominal complaints was obtained. He was febrile, tachypneic, acutely ill without significant lymphadenopathy, chest or abdominal findings initially. Approximately 1 month after admission, a tender mass was palpated in the left upper quadrant of the abdomen.

Admission chest roentgenogram was within normal limits. Four days later a left pleural effusion was noted. In the subsequent month the patient's clinical condition deteriorated while on penicillin.

Laboratory tests: Hematocrit 30 per cent, white blood cell count 11,000, sedimentation rate 80. Urinalysis, urine cultures, sputum cultures and blood cultures were noncontributory. Slight increase was noted in the alkaline phosphatase. VDRL and Kolmer tests were reactive, and febrile, typhoid and cold agglutinins, heterophil, latex fixation and antinuclear antibodies were all within normal limits. The intermediate PPD was 8 mm. in duration while the second strength PPD was 15 mm. Histoplasmin and coccidioidin skin tests were negative.

A pleural biopsy revealed acute and chronic inflammation without evidence of tuberculosis. A liver biopsy showed non-necrotizing granulomas without demonstration of the tuberculosis bacillus. Intravenous pyelography and intravenous cholangiography were noncontributory.

Roentgenologic findings. Fluoroscopic examination revealed hypoactive peristalsis and nondistensibility of the stomach. Figure 1*A* demonstrates denudement of the gastric mucosa, separating and fragmenting the barium, and multiple outpouchings along the lesser curvature. Since no hypersecretion was demonstrated, the separation and compartmentalization of barium was due to a pseudomembrane of necrotic debris.

A linear outpouching was observed along the greater curvature extending outside the projected lumen of the stomach (Fig. 1*A*; and 2, *A* and *B*). This was felt to be a fistulous tract protruding into a thickened gastric wall and ending as an intramural abscess. A large gastric ulcer was demonstrated (Fig. 2, *A–C*) which was neither typical of benign peptic disease nor of neoplastic ulceration.

Because of the patient's age and symptoms, as well as the atypical findings on gastrointestinal examination, celiac angiography was performed as a supplemental and complemental study to differentiate infiltrating neoplastic lesion from chronic inflammatory disease of the stomach.

The arterial phase of the celiac angiogram (Fig. 3*A*) revealed enlargement of the left gastric, short gastrics, and left gastroepiploic arteries with hypervascularity of their normal tapering branch arteries. The capillary phase (Fig. 3*B*) demonstrated a markedly thickened gastric wall and omentum, as well as hypertrophied gastric folds. A selective left gastric injection (Fig. 3*C*) showed a large left gastric artery with normal tapering branch vessels and again a markedly thickened gastric wall and omentum. Chronic inflammatory disease was suggested on the basis of the hypervascularity and thickened gastric wall and without evidence of neoplastic vasculature.

Clinical correlation of the thickened gastric wall and omentum was demonstrated at sur-

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specifically to the fourth ventricle and its floor (Fig. 3; and 11).

In fourth ventricle tumors (Fig. 15), the ballooning of the fourth ventricle displaces its floor forward with compression and anterior displacement of the brain stem. The roof is displaced backward. As tumor extends inferiorly between the tonsils and into the vallecula, the tonsils are separated, compressed on their medial aspects, and elongated anteroposteriorly.[19] In brain stem tumors, on the other hand, the brain stem is enlarged anteroposteriorly, the fourth ventricle flattened and displaced posteriorly. The tonsils are compressed from in front with a decrease in their anteroposterior diameter. Separation of the tonsils is not marked.

Unfortunately, vessels located precisely on the floor of the fourth ventricle cannot be recognized on angiography since they are extremely small.[11] However, there are several identifiable vessels on the anterior aspect of the body of the tonsil which correspond closely to the posterior aspect of the medulla or floor of the fourth ventricle. These include the posterior medullary segment of the posterior inferior cerebellar artery, the posterior medullary segment of the tonsillo-hemispheric branch, the medial tonsillar tributary of the vein of the lateral recess, the vein of the restiform body and the median posterior spinal vein (Fig. 1; and 10). Vessels outlining the posterior aspect of the cerebellar tonsil include the superior retrotonsillar segment of the posterior inferior cerebellar artery, retrotonsillar segment of the tonsillo-hemispheric branch, superior and inferior retrotonsillar tributaries of the inferior vermian vein. The two

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(A_1), (B_1), and (C_1), are sectioned through the upper part of the pons, whereas (A_2), (B_2), and (C_2) are through the upper part of the medulla oblongata.

(A_1) *Normal.* The anterior ponto-mesencephalic vein (APM) and transverse pontine vein (TP) outline the anterior aspect of the pons. The precentral cerebellar vein (Pc) and tributaries of the superior vermian vein (Pcu, Scu) outline various portions of the superior vermis.

(A_2) *Normal.* The junction between the supratonsillar (St) and medial tonsillar (MT) tributaries of the vein of the lateral recess (VLR) at the tonsillar incisura (unlabeled arrow) is located opposite the restiform body on which the vein of the restiform body (RB) lies. The peduncular portion (p) of the vein of the lateral recess of the fourth ventricle joins the floccular portion (f) in the cerebello-ponto-medullary angle. The superior retrotonsillar vein (SRt) is usually a tributary of the inferior vermian vein (IV). The distance between the superior retrotonsillar vein (SRt) and the tonsillar incisura (unlabeled arrow) indicates the anteroposterior diameter of the body of the cerebellar tonsil.

(B_1) *Brain stem glioma.* The midline anterior ponto-mesencephalic vein (APM) is located posterior to the transverse pontine vein (TP). The precentral cerebellar vein (Pc) and tributaries of the superior vermian vein (Pcu, Scu) are displaced backward.

(B_2) *Brain stem glioma.* The peduncular portion (p) of the vein of the lateral recess of the fourth ventricle (VLR) is elongated. The tonsillar incisura (unlabeled arrow) at the junction between the supratonsillar and medial tonsillar tributaries is posteriorly displaced. The vein of the restiform body (RB) is also posteriorly displaced. The superior retrotonsillar tributary (SRt) and suprapyramidal tributary (St) of the inferior vermian vein (IV) are also displaced posteriorly, but to a lesser degree.

(C_1) *Fourth ventricle tumor.* The anterior ponto-mesencephalic vein (APM) and transverse pontine vein (TP) are both displaced forward. The precentral cerebellar vein (Pc) and tributaries (Pcu, Scu) of the superior vermian vein are displaced backward. Displacements of the veins are similar to those seen in brain stem tumors, although more marked lateral displacement may occur in tumors of the fourth ventricle.

(C_2) *Fourth ventricle tumor.* The peduncular (p) and supratonsillar segments (ST) of the vein of the lateral recess (VLR) are displaced markedly laterally in arcuate fashion. The incisura (unlabeled arrow) is displaced anteriorly. The vein of the restiform body (RB) is also displaced forward. The supratonsillar segment is markedly elongated posteriorly as is the medial tonsillar vein. The superior retrotonsillar vein is displaced markedly backward and laterally.

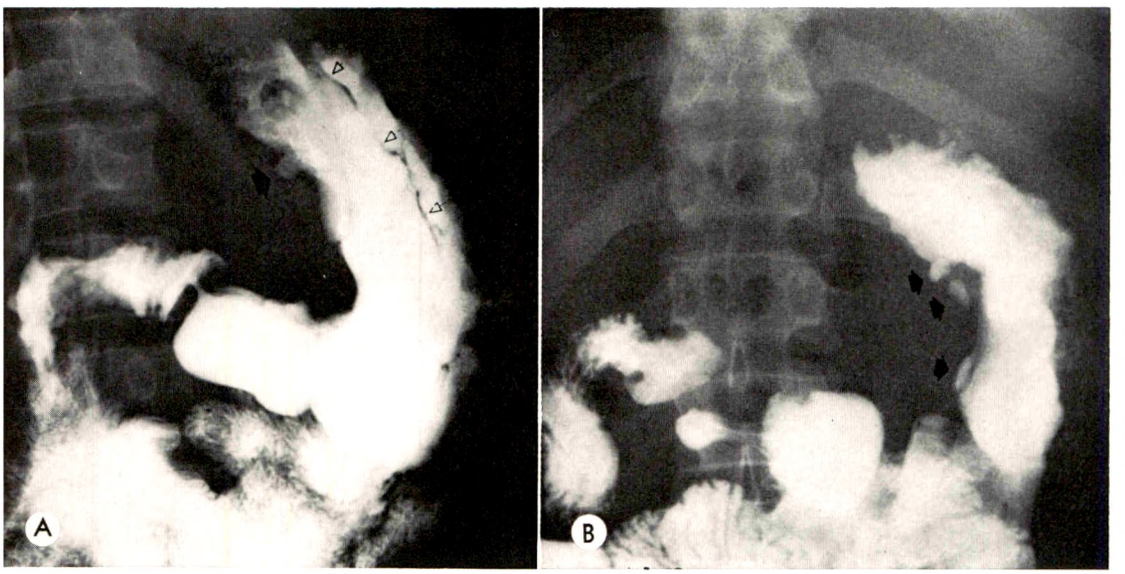


Fig. 1. Upper gastrointestinal series. (*A*) The stomach is relatively nondistensible without hypersecretion. Denudement of gastric mucosa is noted. Separation of barium is due to necrotic debris (←). Ulceration is observed on the lesser curvature (◀). A fistulous tract is demonstrated along the greater curvature. (*B*) Multiple ulcerations and effacement of the gastric mucosa are demonstrated (◀).

gical exploration where a "plastic" character to the left upper quadrant of the abdomen was found, and the omentum matted to the stomach. On biopsy, this was reported to contain tuberculous granulomas.

A repeat upper gastrointestinal series was done approximately 2 months after the patient was started on antituberculosis medication (Fig. 4). The examination failed to reveal the previously described ulcerations, fistulous tract, separation of barium or effacement of the gastric mucosal folds.

DISCUSSION

Pathologically, 6 forms of gastric tuberculosis have been described: (1) tuberculous ulcer; (2) miliary tubercles; (3) hypertrophic gastric tuberculosis; (4) tuberculous pyloric stenosis; (5) solitary tuberculoma; and (6) tuberculous lymphadenitis.

The most frequent form, the ulcerative type, represents 80 per cent of the reported cases.[6] Multiple ulcerations are more commonly found along the lesser curvature and in the pylorus. The edges of the ulcers are irregular, undermined, ragged, thickened and edematous. The base of the ulcer is covered with necrotic debris. Fistulous openings may be found through the mucosa.[1,10] The adjacent lymph nodes are usually caseous or necrotic. As healing ensues marked fibrosis and scarring occur. Since the pylorus is commonly involved, constriction and obstruction of the pyloric channel may result.

A rarer form of involvement is limited to the submucosa of the stomach, producing thickening of the gastric wall and usually an annular constriction of the lumen. Gastric hypertrophic tuberculosis almost exclusively involves the pylorus, although the infiltration may spread through the entire stomach similar to a linitis plastica.[10,11] Pathologically, it is an annular tumor with markedly thickened and fibrotic submucosa and granulomas in the regional lymph nodes. This form of tuberculosis can also lead to severe pyloric obstruction[1] and may be difficult to differentiate from lymphoma roentgenologically.[4]

The other forms of gastric tuberculosis are extremely rare. Miliary tubercles may be found in the terminal stages of generalized miliary tuberculosis, and solitary tuberculomas are basically hypertrophic in

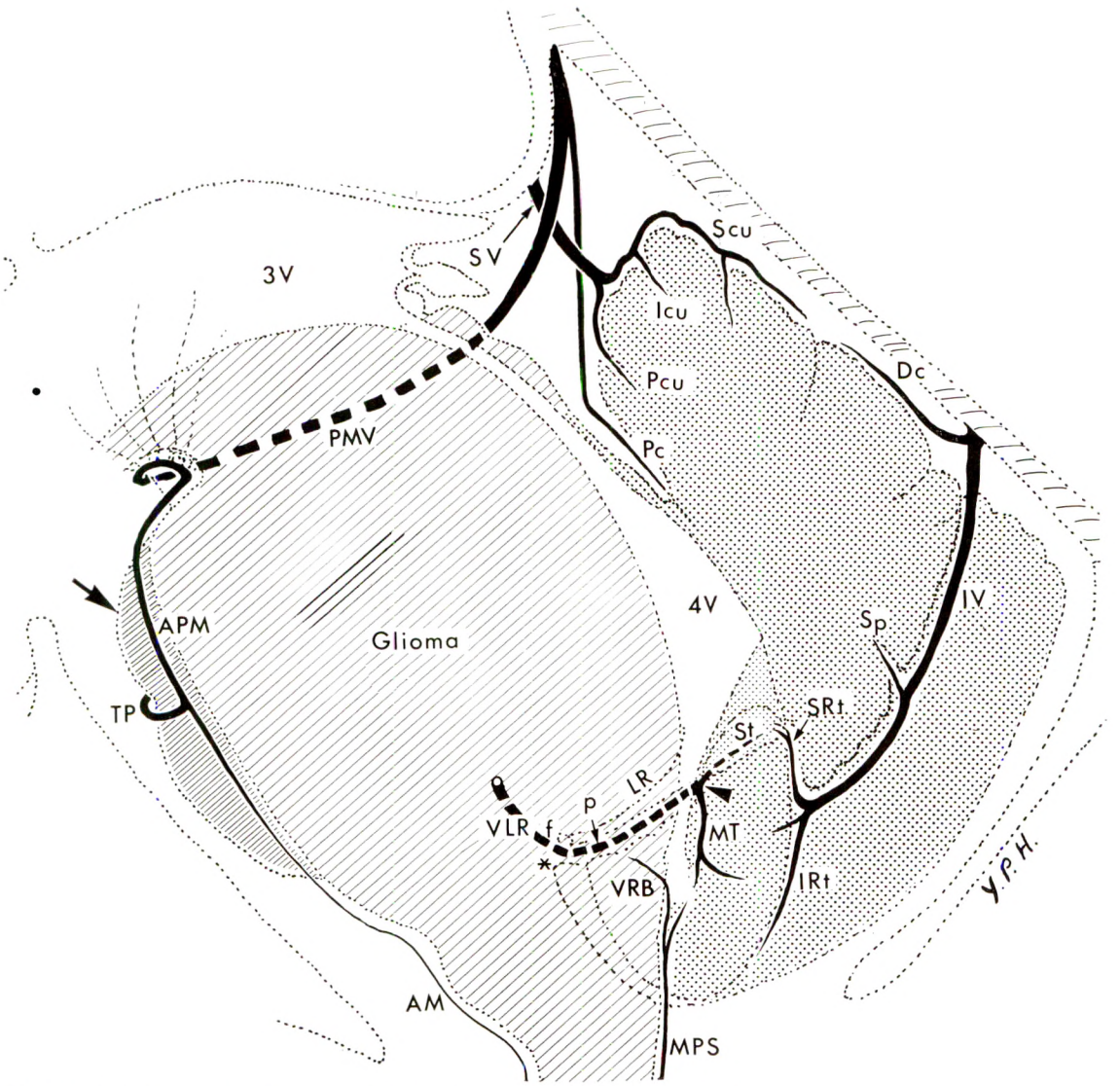


FIG. 12. Diagrammatic representation showing venographic changes in brain stem tumors.

The labeled structures include the anterior medullary vein (AM), anterior ponto-mesencephalic vein (APM), declival vein (Dc), intraculminate tributary (Icu), inferior retrotonsillar tributary (IRt), inferior vermian vein (IV), lateral recess of the fourth ventricle (LR), medial tonsillar tributary (MT), median posterior spinal vein (MPS), precentral cerebellar vein (Pc), preculminate tributary (Pcu), posterior mesencephalic vein (PMV), supraculminate tributary (Scu), suprapyramidal tributary (Sp), superior retrotonsillar tributary (SRt), supratonsillar tributary (St), superior vermian vein (SV), transverse pontine vein (TP), vein of the lateral recess of the fourth ventricle (VLR), and the vein of the restiform body (VRB). The third and fourth ventricles (3V, 4V) are also labeled. The large arrowhead (▲) indicates the location of the tonsillar incisura. Nodular bulging (↑) of a brain stem astrocytoma is illustrated. The pontine segment of the anterior ponto-mesencephalic vein (APM) is displaced forward. The transverse pontine vein (TP) is displaced further forward. Upward and forward displacement and compression of the interpeduncular fossa is depicted by elevation of the upper portion of the pontine segment, mesencephalic segment and roof segment of the anterior ponto-mesencephalic vein. The precentral cerebellar vein (Pc) and, to a lesser degree, the superior vermian (SV) and its tributaries are displaced backward. The distance between the anterior spinal and medullary veins (AM) and veins on the posterior aspect of the cord and medulla is increased. The junction (▲) between the medial tonsillar vein (MT) and the vein of the lateral recess of the fourth ventricle (VLR) is displaced backward.

The distance between the medial tonsillar vein and the retrotonsillar tributaries (SRt, IRt) is diminished. Due to extension of tumor through the brachium pontis and restiform body, the peduncular portion (p) of the vein of lateral recess is stretched, elongated and arched downward. The inferior vermian (IV) and its superior and inferior retrotonsillar tributaries are markedly posteriorly displaced and the copular angle formed by the inferior vermian vein (IV) and its superior retrotonsillar tributary (SRt) is decreased. Flattening and posterior displacement of the lower portion of the fourth ventricle is indicated by diminution of the anteroposterior diameter of the posteriorly flattened cerebellar tonsil outlined by the retrotonsillar veins and medial tonsillar vein. Also shown is anterior displacement and arching of the thalamoperforating veins exiting from the posterior perforated substance.

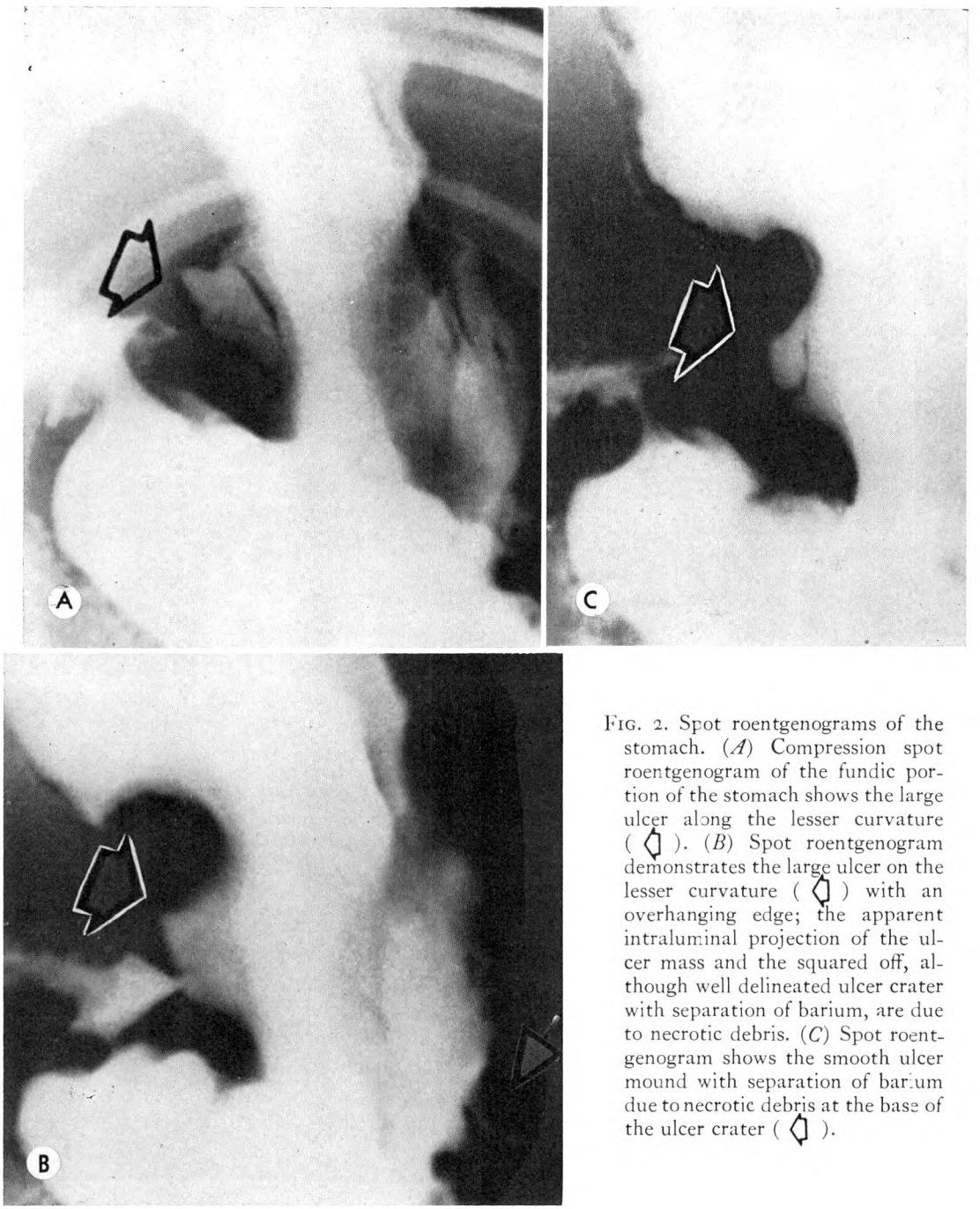


FIG. 2. Spot roentgenograms of the stomach. (*A*) Compression spot roentgenogram of the fundic portion of the stomach shows the large ulcer along the lesser curvature (◁). (*B*) Spot roentgenogram demonstrates the large ulcer on the lesser curvature (◁) with an overhanging edge; the apparent intraluminal projection of the ulcer mass and the squared off, although well delineated ulcer crater with separation of barium, are due to necrotic debris. (*C*) Spot roentgenogram shows the smooth ulcer mound with separation of barium due to necrotic debris at the base of the ulcer crater (◁).

type; *i.e.*, the involvement is limited to the submucosa and therefore presents as a tumor mass.[10]

There are no pathognomonic clinical or roentgenologic findings in gastric tuberculosis. Gastric tuberculosis should be suspected in a patient with medically unresponsive gastric ulcers involving primarily the pyloroduodenal segment and interfering little with peristalsis. Simultaneous involvement of both the pylorus and duodenum, as well as demonstration of fistu-

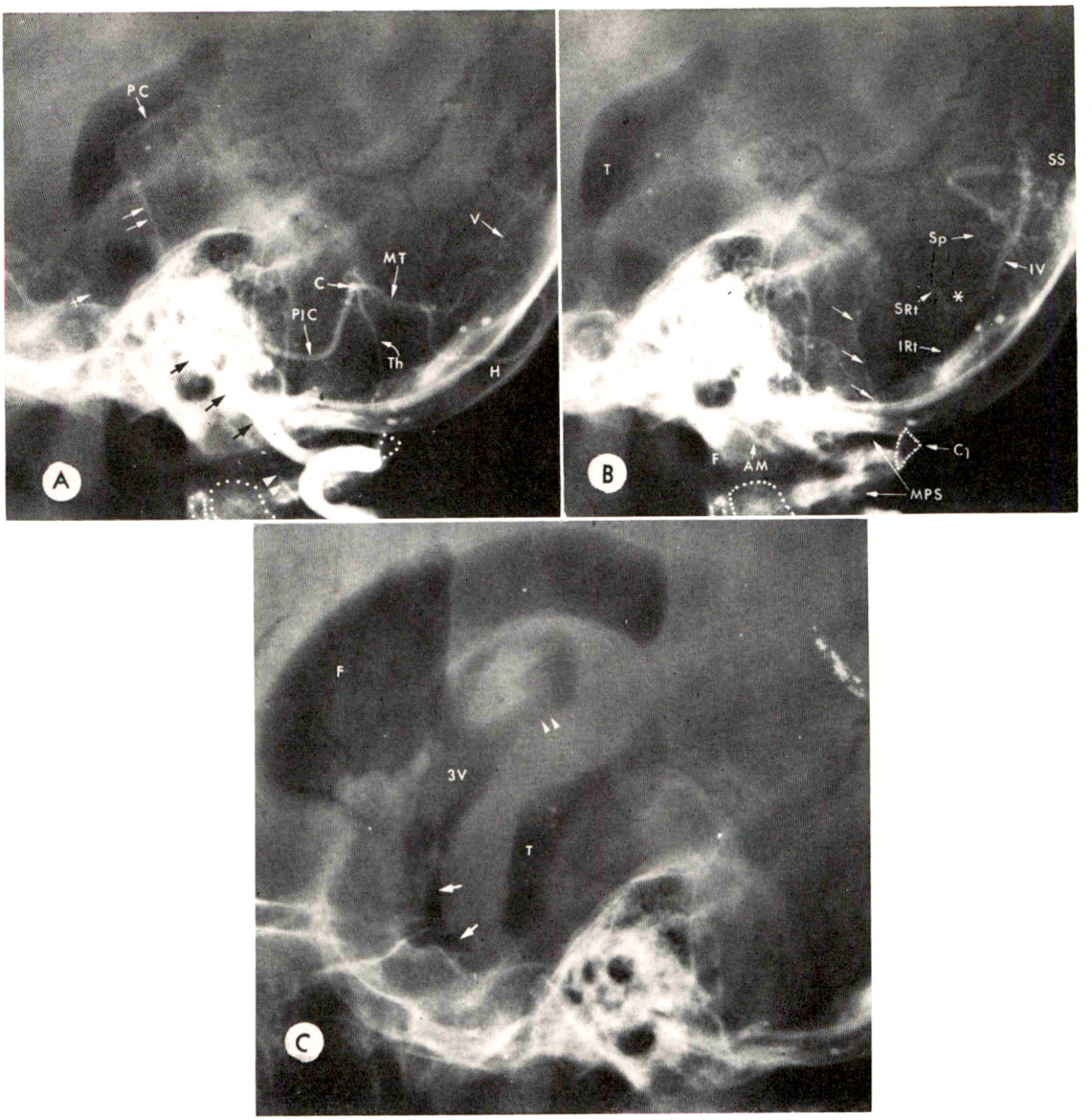


FIG. 13. Paradoxical posterior displacement of the basilar artery in huge brain stem astrocytoma.

(*A*) Left brachial angiogram following pneumoencephalography, arterial phase, lateral view. The basilar artery (↑ ↑) is stretched upward and markedly displaced posteriorly producing marked *increase* in the distance between this vessel and the clivus (‡). The posterior cerebral artery (PC) is faintly visualized and is markedly elevated. The distal portion of the vertebral artery (black arrows) is slightly posteriorly displaced. The upper portion of the anterior spinal artery (double arrowheads) is located closer to the anterior margin of the foramen magnum than the vertebral artery. The distance between the basilar artery and the choroidal point (C), although both are displaced posteriorly, is nevertheless markedly increased. The proximal portion of the posterior inferior cerebellar artery (PIC) is straightened. The length of the medial tonsillar segment (MT) indicates a normal anteroposterior diameter of the body of the tonsil presumably due to extension of the tumor into the tonsil. The vermian segment (V) of the posterior inferior cerebellar artery is posteriorly displaced. The tonsillo-hemispheric branch (Th) is displaced and curved posteriorly beyond the posterior margin of the arch of C_1 (retouched). This vessel outlines the posterior aspect of the bulging medulla.

(*B*) Venous phase, lateral view. The anterior medullary vein (AM) is displaced forward. The median posterior spinal vein (MPS) and its upward continuation (↑) into the cerebello-medullary fissure are displaced markedly backward. The distance between this vessel and the retrotonsillar tributaries (SRt, IRt) of the inferior vermian vein (IV), *i.e.*, the anteroposterior diameter of the tonsil, is within normal limits. The distance between the suprapyramidal tributary (Sp) and the superior retrotonsillar tributary (SRt) is increased indicating additional tumor infiltration into the vermis. This is confirmed by maintenance of the normal copular angle and curve (*). Air filled temporal horn (T), posterior arch of C_1, straight sinus (SS) and anterior margin of the foramen magnum (F) are also labeled.

(*C*) Pneumoencephalogram, lateral view, brow-up. Marked enlargement of brain stem, upward and forward, has practically effaced the basal cisterns (↑). The floor (▲) of the third ventricle (3V) is markedly elevated. The frontal (F) and temporal (T) horns are slightly enlarged.

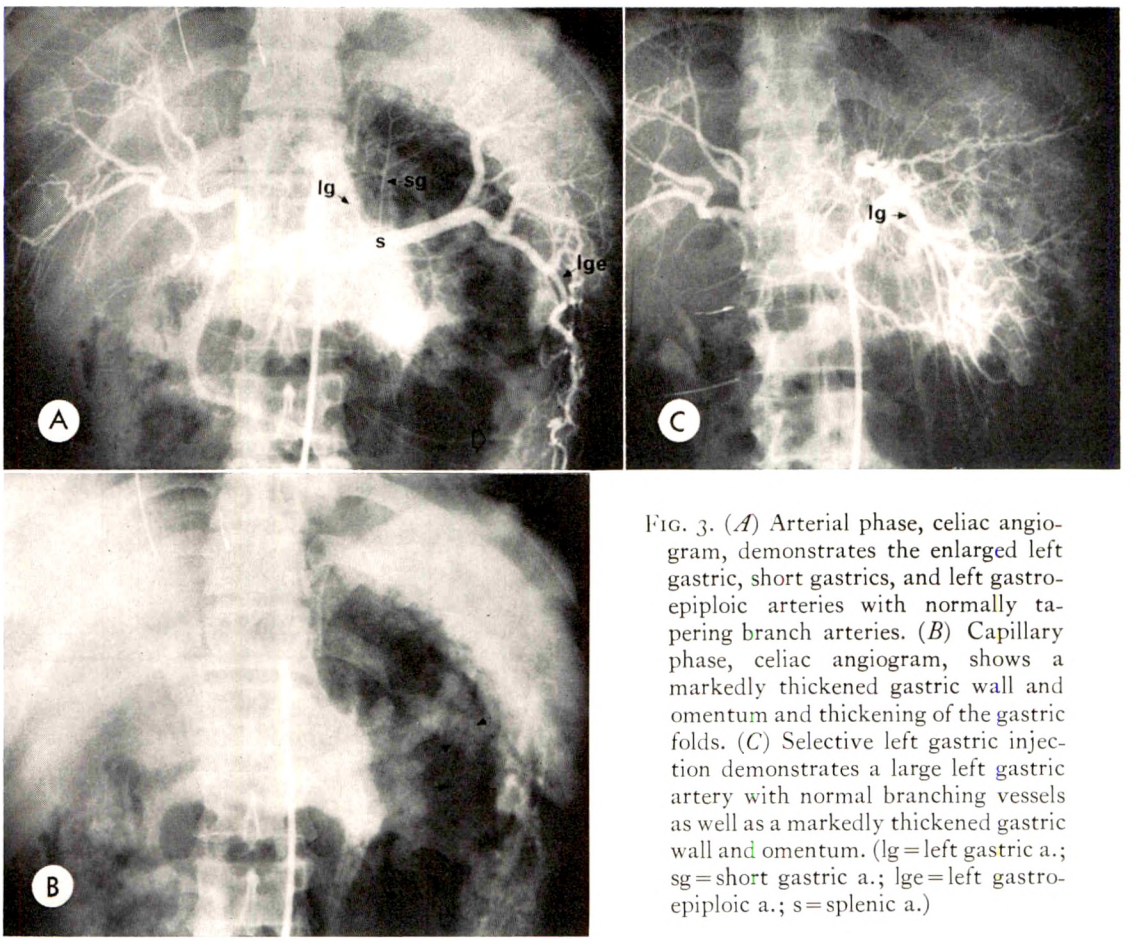


Fig. 3. (*A*) Arterial phase, celiac angiogram, demonstrates the enlarged left gastric, short gastrics, and left gastroepiploic arteries with normally tapering branch arteries. (*B*) Capillary phase, celiac angiogram, shows a markedly thickened gastric wall and omentum and thickening of the gastric folds. (*C*) Selective left gastric injection demonstrates a large left gastric artery with normal branching vessels as well as a markedly thickened gastric wall and omentum. (lg = left gastric a.; sg = short gastric a.; lge = left gastroepiploic a.; s = splenic a.)

lous tracts should suggest a tuberculous etiology, as it did in our case. Also the atypical appearance of the gastric ulceration, whether benign or malignant, in a young patient should suggest gastric tuberculosis. About 10 per cent of cases of gastric tuberculosis are associated with duodenal involvement and rarely produce a stenotic lesion. Most often a duodenitis is present and, as infiltration of the duodenum occurs, irregularity of the duodenal wall and dilatation are seen roentgenologically. Angiographic demonstration of marked hypervascularity as well as thickening of the gastric wall and omentum without specific signs of neoplastic infiltration should suggest tuberculosis.

Differential diagnosis should include lymphoma which more diffusely involves the stomach, whereas gastric tuberculosis is primarily localized to the pyloroduodenal area. Syphilis of the stomach can also be confused with hypertrophic gastric tuberculosis but serologic tests should help to establish the correct diagnosis. Leiomyosarcoma and gastric carcinoma have characteristic roentgenographic and angiographic patterns and interfere with gastric motility and peristalsis more so than does gastric tuberculosis. Differentiation from peptic ulcer disease is established by the response to appropriate therapy indicated in our case by the dramatic improvement when antituberculosis medication was instituted.

SUMMARY

A case of gastric ulcerative tuberculosis is reported with angiographic findings of hypervascularity and thickening of the gastric wall and omentum.

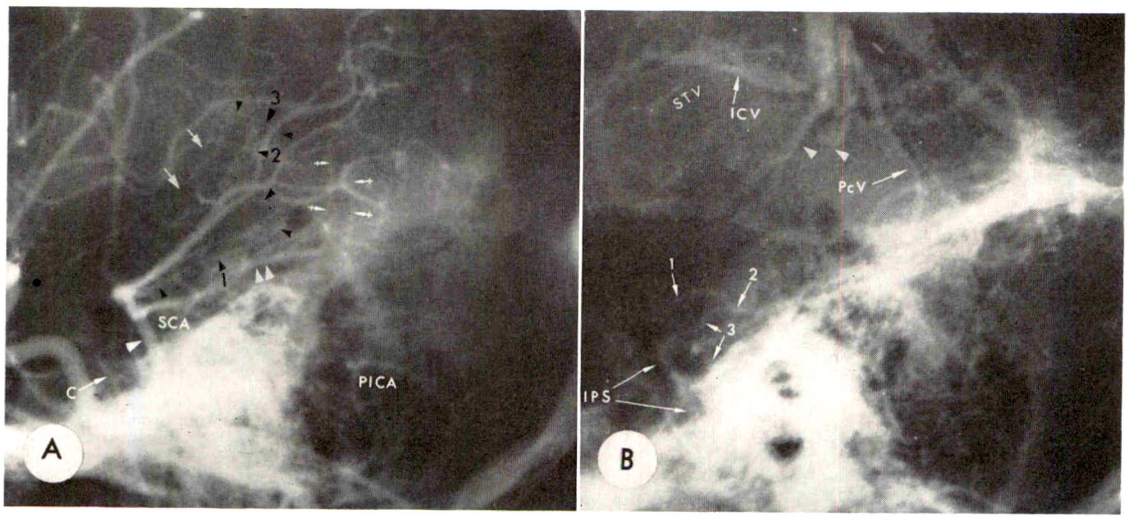


FIG. 14. Quadrigeminal plate tumor. Adjacent tegmentum and pretectum are also involved.

(*A*) Right brachial angiogram, arterial phase, lateral view. The distance between the basilar artery (▲) and the clivus (C) is unremarkable, although its distal end appears tilted forward. Striking findings are marked posterior displacement of the anterior culminate segments (↕) and depression of the anterior superior marginal segments (double white arrowheads) of the superior cerebellar arteries. The medial posterior choroidal artery (row of black arrowheads) shows several changes. Its first or circumpeduncular portion (1) is stretched backward; its second or tectal portion (2) is elongated upward; its pretectal portion (3) is markedly elevated. The infrapulvinar portions of the lateral posterior choroidal arteries are displaced forward (↑).

(*B*) Venous phase, lateral view. The precentral cerebellar vein (PcV) is arched and displaced backward. The posterior parts of the internal cerebellar vein (ICV) and superior thalamic vein (STV) and, to a lesser degree, the posterior portions of the posterior mesencephalic veins (arrowheads) are displaced upward. The roof (1), mesencephalic (2), and pontine segments (3) of the anterior ponto-mesencephalic vein are well visualized and demonstrate compression of the interpeduncular cistern from above and *behind*. The inferior petrosal sinus (IPS) is also labeled.

groups of vessels which outline the anterior and posterior margins of the body of the cerebellar tonsil can therefore be used to determine the anteroposterior diameter of the body of the cerebellar tonsil or height of the fourth ventricle. Vessels which bridge these two groups of vascular channels include the supratonsillar segment of the posterior inferior cerebellar artery and supratonsillar tributaries either of the vein of lateral recess of the fourth ventricle or of the inferior vermian vein. In fourth ventricle tumors,[19] vessels corresponding to the posterior aspect of the medulla oblongata (or anterior aspect of the body of the tonsil) are typically displaced forward and laterally; in brain stem tumors, they are displaced posteriorly with little lateral displacement. The supratonsillar segment of the posterior inferior cerebellar artery is markedly elongated anteroposteriorly and displaced laterally in arcuate fashion in fourth ventricle tumors; in brain stem tumors, this segment is crowded backward and usually shows little lateral displacement. The retrotonsillar group of vessels are displaced markedly posteriorly and also laterally in fourth ventricle tumors. Instead of being vertical, they may become transverse, effectively obliterating the copular angle completely, especially when the fourth ventricle tumor extends into the posterior cerebellar notch. In brain stem tumors, the retrotonsillar vessels are posteriorly displaced with relatively little lateral displacement. The copular angle is easily identifiable because it is more acute than normal. Lateral displacement of the peduncular portion of the vein of the lateral recess is greater in fourth ventricle tu-

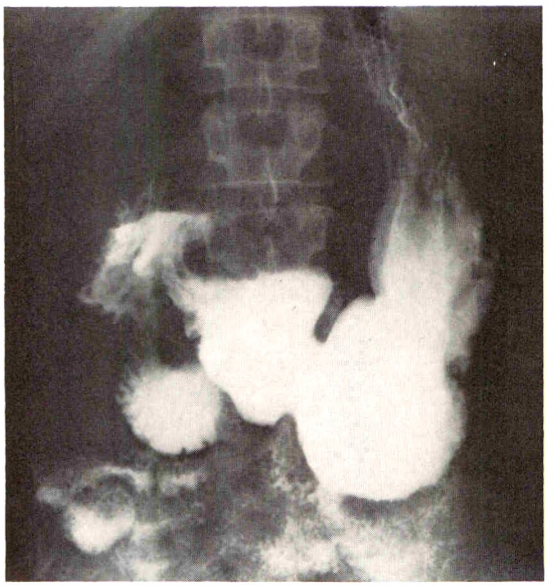


FIG. 4. Gastrointestinal series after about 1½ months on antituberculosis therapy shows complete healing of the ulcerations, fistulous tract, and effacement of the gastric mucosal folds.

Regression of the tuberculous lesions after administration of antituberculosis drugs is demonstrated.

Since the treatment of gastric tuberculosis is medical, the radiologist must be aware of this entity to advise appropriate treatment and avoid undue surgery.

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mors than in brain stem tumors. In the former, this segment of the vein may be displaced laterally beyond the brachial vein as seen in the Towne projection. This is particularly likely when the fourth ventricle tumor extends into and balloons the lateral recess. In contrast, when a brain stem tumor involves the brachium pontis and restiform body, the peduncular segment of the vein of the lateral recess is displaced downward and often medially. The normal angular course between the peduncular and floccular segments of the vein is blunted in brain stem tumors, whereas in fourth ventricle tumors this angle is maintained. This is best seen in the lateral view. In fourth ventricle tumors, with ballooning of the upper part of the fourth ventricle and ventricularization of the lower part of the aqueduct, the fissural and parenchymal portions of the precentral cerebellar vessels are markedly posteriorly and superiorly displaced. This results in a more acute colliculo-central angle, elevation of but not marked posterior displacement of the colliculo-central point. In brain stem tumors, the roof of the upper part of the fourth ventricle assumes a gentle posterior arching, resulting in posterior flattening of the precentral cerebellar vessels, a more obtuse colliculo-central angle, elevation and obvious posterior displacement of the colliculo-central point. In fourth ventricle tumors, vessels running in the superior paravermian sulcus (precentral cerebellar arteries, brachial tributaries of or a split precentral vein) are more markedly separated than in brain stem tumors. The thalamoperforating vessels in fourth ventricle tumors are not displaced or arched forward to any significant degree, whereas in brain stem tumors these small vessels are, with involvement of the midbrain or subthalamic region, displaced forward.

Asymmetric changes are features of brain stem tumors rather than fourth ventricle tumors. Paradoxical or pseudo-extra-axial displacements are not seen in fourth ventricle tumors and are almost pathognomonic of brain stem astrocytoma.

In occasional cases, fourth ventricle and brain stem tumors may show similar changes in the lateral view. In some instances of fourth ventricle tumors, the posterior medullary segment of the posterior inferior cerebellar artery may be displaced *posteriorly* instead of anteriorly and an apparent supratonsillar segment may be elongated and displaced *inferiorly*. These findings are the result of anatomic variations: prior to the development of the tumor the posterior medullary segment was probably located on the medial aspect of the tonsil rather than over the superior pole. An increase in the anteroposterior diameter of the tonsil may be due to a fourth ventricle tumor or a brain stem tumor with extension into the tonsil. The copular angle may not be more acute in brain stem tumor if growth has extended into the pyramidal portion of the inferior vermis. However, in the Towne projection there will always be *marked lateral displacement of all vessels related to the tonsil in fourth ventricle tumors*, while in brain stem tumors lateral displacement of these vessels is minimal or absent.

Extension of tumor into the mid-brain, subthalamic region or thalamus obviously does not occur with tumors of the fourth ventricle.

SUMMARY

1. Tumors of the brain stem show characteristic angiographic features in both arterial and venous phases. These result from the central location of the tumor and more or less symmetric expansion displacing adjacent vessels. Specific angiographic features depend on the location and size of the tumor. Pseudo-extra-axial or paradoxical displacements of vessels are emphasized as characteristic of brain stem astrocytomas.

2. Angiographic features of extension of a brain stem tumor into the cerebellar hemisphere, mesencephalon, floor of the third ventricle, tectum, medulla oblongata and upper cervical cord are described.

3. While both brain stem tumor and fourth ventricle tumors share several angio-

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TRANSFEMORAL SUPERSELECTIVE CELIAC ANGIOGRAPHY*

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SELECTIVE celiac angiography has become one of the most important methods of diagnosis in intraabdominal organs. This method, however, has the limitation that the finer vessels of the pancreas are poorly opacified and overlapped by other vessels, which is one reason why early malignant lesions escape detection. One way to prevent these shortcomings is to inject the contrast medium through catheters placed in the branches supplying only one specific organ. The tip of the catheter is positioned in the secondary branches of the celiac artery; *e.g.*, hepatic, splenic or gastroduodenal artery. This procedure may be called "superselective" catheterization.

This paper describes our technique of superselective celiac catheterization from the femoral artery and demonstrates the extent to which the pancreatic artery can be revealed when the tip of the catheter is positioned in the secondary branches of the celiac artery.

INSTRUMENTATION AND TECHNIQUES

A Muller-USCI deflector guide wire system and Variflex Positrol Catheter* are utilized. The catheter is 80 cm. long and 7 F size. The distal 4 cm. of it is tapered to 5 F. After percutaneous puncture of the femoral artery and introduction of a spring guide wire, the catheter is passed over the wire into the artery. Sometimes the vessel dilator is used to dilate the artery and facilitate the introduction of the catheter. The Muller guide system with an 80 cm. tip deflecting wire is introduced into the catheter. The angle of the catheter tip may be varied by controlling the deflectable guide. This tip may be maneuvered into the celiac artery under image intensified fluoroscopy. Before superselective catheterization is done, celiac angiography is performed in the standard manner. The ordinary celiac angiogram serves as a vascular map for further catheterizations.

An important attribute of this system is the ability to "pay out" the catheter over the deflected tip of the guide wire in order to obtain a more peripheral position beyond the bifurcation of the celiac artery (Fig. 1). This process allows the tip of the catheter to be advanced from the celiac axis to a more peripheral artery while keeping the force directed caudad, simulating the catheter introduced from the axillary artery, which will easily catheterize the celiac axis in many patients because of its caudad orientation. In patients with a cephalad directed celiac axis, the curve of the tip of the guide wire is decreased, and in those with a caudad directed celiac axis, the curve of the guide wire is accentuated. In order to prevent coagulation within the catheter, frequent intermittent injections of physiologic saline solution with heparin should be performed through the injector manifold throughout the procedure.

For investigation of the hepatic or splenic artery, 20 to 40 ml. of conraxin-H† is injected by hand. The gastroduodenal artery requires 20 to 25 ml. of the contrast medium. Roentgenograms are obtained on a rapid serial film changer at the rate of 2 films per second for 10 seconds.

CATHETERIZATION RESULTS

The various positions of the catheter tip

* United States Catheter and Instrument Corp., N.Y., U.S.A.

† Sixty per cent of sodium and N-methylglucamine salt of iodamide. Takeda Co., Osaka, Japan.

* From the Department of Radiology, Kanazawa University School of Medicine, Kanazawa, Japan.

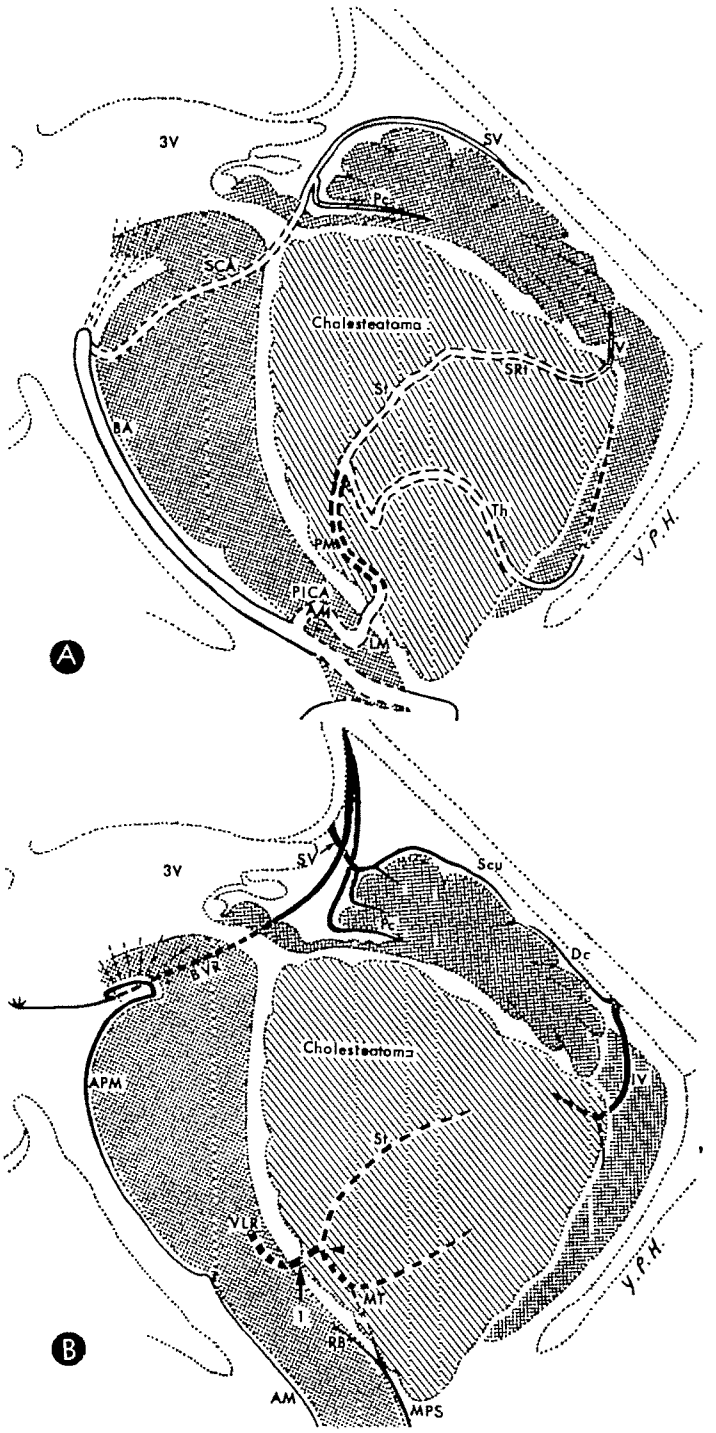


FIG. 15. Diagrammatic representation of "mid-sagittal section" of the brain stem and cerebellum showing displacements in a fourth ventricle tumor.

(*A*) Arterial phase. The labeled structures include the basilar artery (BA), lateral medullary segment (LM), precentral cerebellar artery (Pc), posterior inferior cerebellar artery (PICA), posterior medullary segment (PM), superior cerebellar artery (SCA), superior retrotonsillar segment (SRt), supratonsillar segment (St), vermian segment (SV) of the superior cerebellar artery, tonsillo-hemispheric branch (Th) and vermian segment (V) of the inferior cerebellar artery. The third ventricle is also labeled.

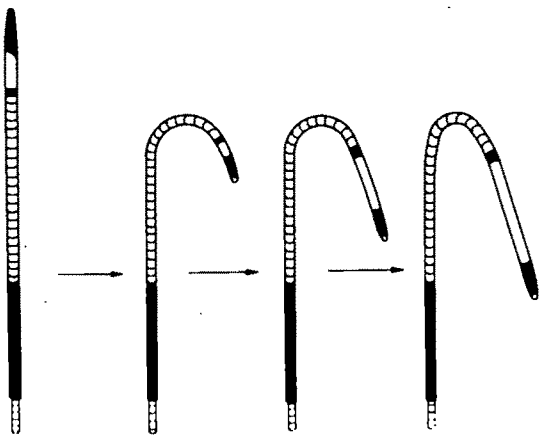


FIG. 1. After the tip of the catheter is deflected by a guide wire controlled by an external mechanism, "paying-out" the catheter over the deflected guide advances its tip to a more peripheral artery keeping the force directed caudad.

in 60 attempted superselective catheterizations are shown in Figure 2.

Twenty-two of the patients studied showed normal pancreatic vasculature. In these 22 patients, the results of pancreatic arterial filling at different injection sites are given tabularly. Table 1 shows that

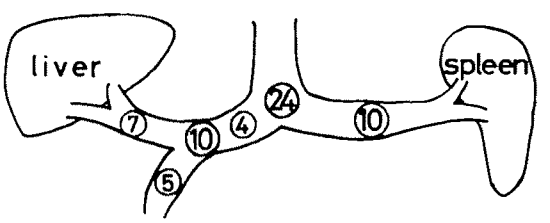


FIG. 2. The positions of the catheter tip in 60 attempted superselective catheterizations.

the arterial supply of the head of the pancreas was opacified by injecting the contrast medium into the gastroduodenal artery (GD), proper hepatic artery (PH) or the terminal portion of the common hepatic artery. The degree of filling of the intrapancreatic arteries was similar at the 3 different injection sites. Both the posterior pancreaticoduodenal arcade (PPDA) and the anterior pancreaticoduodenal arcade (APDA) were completely visualized consistently, and contrast medium even passed into the inferior pancreaticoduodenal artery (IPD) and superior mesenteric artery via those 2 arcades (Fig. 3 and 4). The small unnamed intrapancreatic branches in the head were consistently well filled.

By splenic injection (SA), visualization of the pancreatica magna artery and its branches in the tail of the pancreas was achieved (Fig. 5).

When the contrast medium was injected directly into the proper hepatic, gastroduodenal and the terminal portion of the common hepatic artery, the small arteries of the tail of the pancreas were not opacified as well as those of the head. In the same manner, when the injection was into the splenic artery, only a few arteries of the head were visualized.

DISCUSSION

Several feasible techniques by which secondary branches of the celiac artery are intentionally catheterized have been de-

TABLE I

COMPARISON OF VISUALIZATION OF THE PANCREATIC ARTERIES AT EACH SUPERSELECTIVE SITE

Position of Catheter Tip	Cases	PPDA / APDA	IPD	DP	TP	Small Vessels		
						Head	Body	Tail
GD	4	4	4	2	3	4	1	1
Bifurcation of PH and GD	4	4	3	2	3	3	2	1
PH	5	5	4	2	3	4	2	1
SA	9	0	0	0	8	0	2	9

GD=gastroduodenal artery; PH=proper hepatic artery; SA=splenic artery; PPDA=posterior pancreaticoduodenal arcade; APDA=anterior pancreaticoduodenal arcade; IPD=inferior pancreaticoduodenal artery; DP=dorsal pancreatic artery; TP=transverse pancreatic artery.

graphic features, there are sufficient differential criteria to permit correct diagnosis in the majority of cases. Differentiation is based on the recognition of vessels which serve as landmarks for the fourth ventricle and the cerebellar tonsils.

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The basilar artery (BA) and the distal portion of the vertebral artery are displaced forward. The anterior, lateral and posterior medullary segments (AM, LM, PM) of the posterior inferior cerebellar artery are displaced and crowded forward. The superior retrotonsillar segment (SRt) is displaced markedly upward and backward effacing the copular angle. This segment forms a continuous curve with the supratonsillar segment. The vermian segment is displaced markedly backward and upward. The intervening supratonsillar segment (St) is markedly stretched posteriorly, elongated and elevated. This is also true of the tonsillo-hemispheric branch (Th). The fissural segment of the precentral cerebellar artery (Pc) is displaced markedly upward producing an acute angle in the course of the artery. The colliculo-central point corresponding to the apex of the angle is displaced markedly upward. It is only slightly displaced posteriorly. The thalamo-perforating arteries are unremarkable.

(*B*) Venous phase. The labeled structures include the anterior medullary vein (AM), anterior ponto-mesencephalic vein (APM), basal vein of Rosenthal (BVR), declival vein (Dc), inferior vermian vein (IV), median posterior spinal vein (MPS), medial tonsillar vein (MT), precentral cerebellar vein (Pc), vein of the restiform body (RB), supraculminate vein (Scu), supratonsillar vein (St), superior vermian vein (SV) and vein of the lateral recess of the 4th ventricle (VLR). The first segment or peduncular segment (1) of the vein of the lateral recess, the location of the tonsillar incisura (▲) and the third ventricle are also labeled.

The anterior ponto-mesencephalic vein (APM) is displaced forward. The interpeduncular fossa is narrowed from below but usually not tilted forward. The fissural segment of the precentral cerebellar vein (Pc) shows changes similar to the precentral cerebellar artery. The tributaries of the superior vermian vein (SV) are crowded backward and upward. The peduncular segment of the vein of the lateral recess (VLR) is shortened and the junction between the markedly stretched and elongated supratonsillar vein (St) and the medial tonsillar vein (MT) is displaced forward. The supratonsillar vein is, in addition, elevated. The inferior vermian vein (IV) is displaced backward and upward.

vised. The first technique was described by Boijsen,[1,2] who introduced the catheter from the axillary artery and advanced it from the celiac axis to a more peripheral artery. The disadvantages of the axillary approach, especially the difficulty of performing the percutaneous puncture and the Seldinger technique in the relatively small vessels of the Japanese, prevents this from being a popular technique. Local thrombus formation is a frequent complication when this technique is employed. Paul *et al.*[3] described a technique in which a large catheter is placed in the celiac axis and then a smaller catheter is advanced through the large one into the secondary branches. This technique requires a large primary catheter and there is the risk of clot formation between the 2 catheters. More recently, superselective pancreatic catheterization, using the controled tip guide wire, was devised by Reuter.[4] Rösch and Grollman[5] reported that flexible preformed catheters with various secondary curves are the most suitable approach to superselective catheterization. In spite of the several advantages of a superselective approach, the disadvantages, including the extensive time required for manipulation and the resultant vascular complications, have recently elicited criticism.

In evaluating our technique, the catheterizations were divided into success and failure, according to the vessel configuration (Fig. 6). Many of the failures occurred

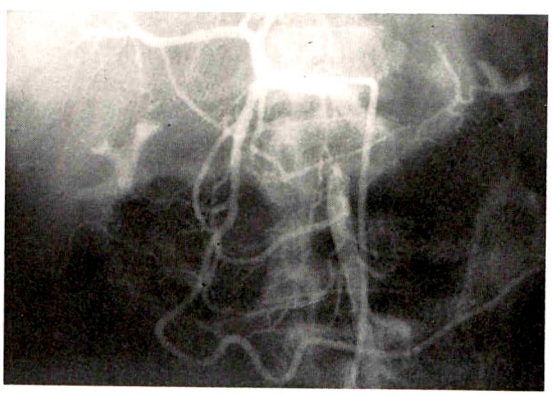


Fig. 3. Injection at the terminal portion of the common hepatic artery. There is excellent visualization of the branches in the head of the pancreas. The inferior pancreaticoduodenal and superior mesenteric arteries are also opacified.

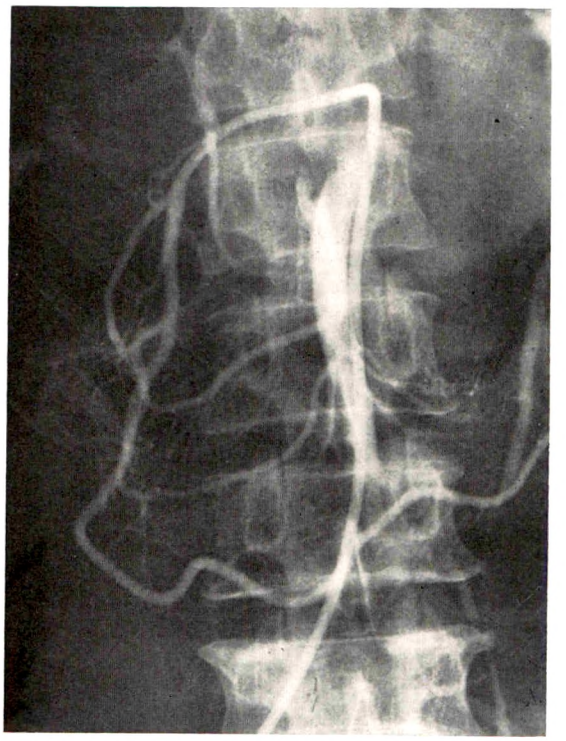


Fig. 4. Superselective gastroduodenal arteriogram. There is good visualization of the 2 arcades, the inferior pancreaticoduodenal and the superior mesenteric arteries.

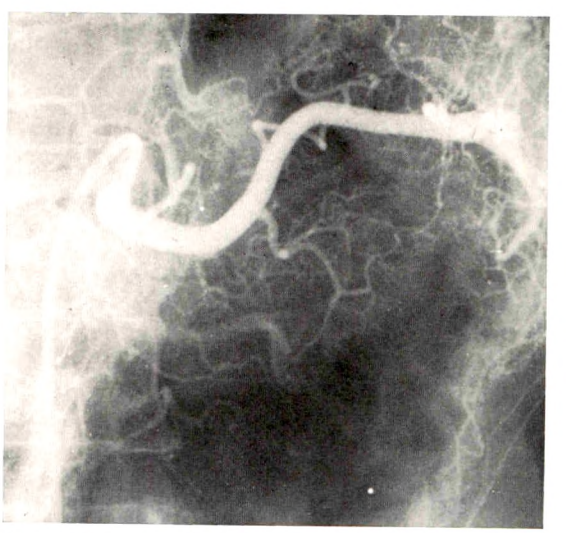


Fig. 5. Splenic injection. The small branches in the tail of the pancreas and the transverse pancreatic artery are well demonstrated.

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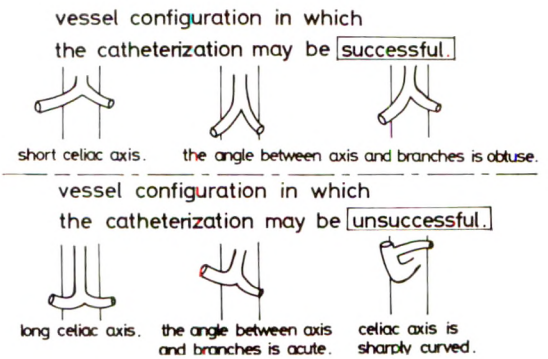


FIG. 6. Vessel configurations.

in the cases in which the angle between the celiac axis and the secondary branch is acute, or in which the celiac artery itself is sharply curved. Even in the former, when the celiac axis is short, it is possible to control the tip of the catheter and reach the proximal portion of secondary branches after introduction into the celiac axis. It is essential to obtain an exact anatomic evaluation of the celiac arterial system in each case before attempting to catheterize the vessels superselectively. The superselective approach is seldom successful in the branches which arise at an acute angle from a long celiac artery. But even in such cases, deep inspiration and expiration may aid the catheterization by modifying the angle between the celiac artery and its secondary branches (Fig. 7, *A* and *B*).

Examination time is not unduly prolonged by our technique, because the cases to be catheterized superselectively are selected after evaluating the branching of the celiac artery on the angiograms obtained by the conventional technique of injection.

No complications were observed following the procedure. It should be emphasized that the frequent intermittent flushing with physiologic saline and heparin solution is required to prevent clot formation at the catheter tip.

The results of visualization of each pancreatic artery at different superselective injection sites is shown in Table 1. They indicate that the arteries in the pancreatic head filled consistently with the gastroduodenal injection. These arteries were also filled by injection of the proper hepatic artery or the bifurcation of the proper hepatic artery and gastroduodenal artery. They were not filled at all by injection into the splenic artery. The arterial visualization of the pancreatic head with an hepatic artery injection is due to reflux of contrast medium into those arteries.

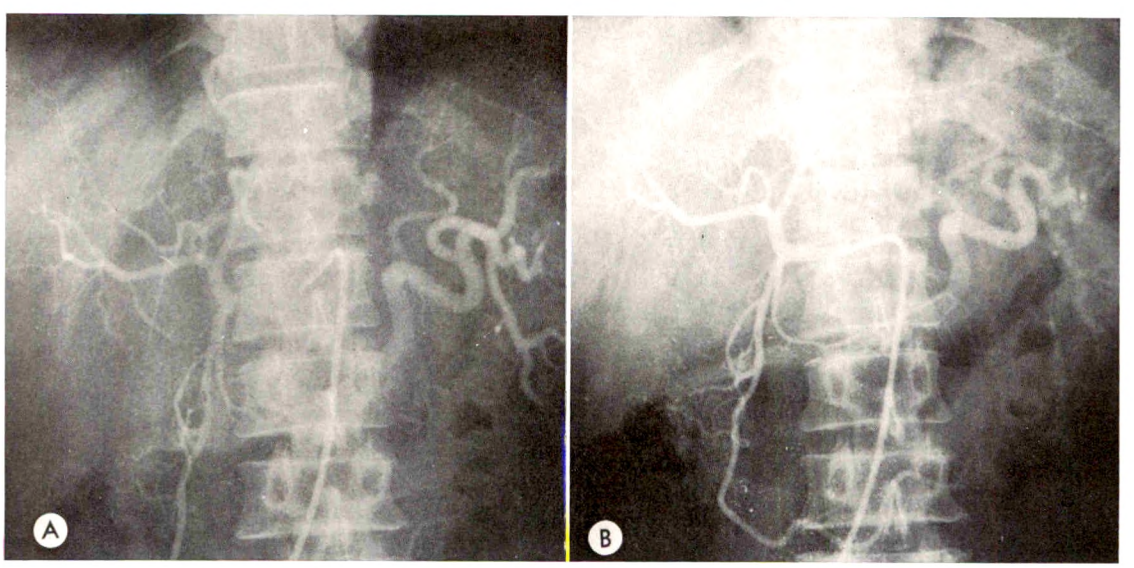


FIG. 7. (*A*) Inspiration. (*B*) Expiration. The angle between the celiac axis and the branch is changed with respiration. In spite of the unchanged position of the proximal celiac artery, the distal part of it may move up and down. Changing the angle between the aorta and the celiac axis is sometimes useful during catheterization.

EXTENSION OF POSTERIOR FOSSA TUMORS, PARTICULARLY INTRAVENTRICULAR FOURTH VENTRICLE TUMORS, INTO THE UPPER CERVICAL SPINAL CANAL*

By BERNARD S. EPSTEIN, M.D., JOSEPH A. EPSTEIN, M.D., *and* ROBERT CARRAS, M.D.
NEW HYDE PARK, NEW YORK

THE roentgenologic identification of direct extension of posterior fossa tumors into the upper cervical spinal canal has received little attention in the radiologic literature. It is well known that tonsillar herniation can be identified frequently in patients with increased intracranial pressure. However, the fact that similar and usually indistinguishable intrusion of tumor can occur with some posterior fossa neoplasms merits further discussion. To further investigate this, a review of 65 patients with posterior fossa tumors, including 16 with fourth ventricle intraventricular lesions, was made. There were 10 with verified tumorous extension into the upper cervical subarachnoid space. The underlying tumor originated within the fourth ventricle in 7, and 3 had extraventricular tumors. Our purpose was to study the anatomic mechanism of tumorous extension from the posterior fossa, especially from the fourth ventricle, into the cervical region and to correlate this with the clinical and roentgenologic data. The distribution of these tumors according to type and age is set forth in Table 1.

CLINICAL ASPECTS

The duration of symptoms varied from about 1 week to 2 months in 10 patients, between 6 months and 1 year in 6, and 2 to 4 years in the patient with a tonsillar hemangioblastoma and another adult patient with an auditory neurinoma. The latter patient had a slow hearing loss and slowly progressive dizziness for about 4 years before the diagnosis was made. It was intriguing to find a patient with a cerebellopontine angle tumor over 5 cm. in diameter with so few complaints.

The symptoms noted were those usually associated with posterior fossa tumors, including anorexia, nausea, vomiting, nystagmus and ataxia. A curious correlation existed between the relative mildness of symptoms and the size of the tumor, a frequent occurrence with intraventricular fourth ventricle tumors, which tend to be large. Papilledema was present in 13 patients. In 3 the early complaints included neck pain radiating to the occipital area. Two patients died suddenly because of massive intratumoral hemorrhages.

METHODS OF EXAMINATION

PLAIN FILM ROENTGENOGRAPHY

Sutural separation was present in 8 patients, all under 10 years of age. Increased digitations of the cranium were observed in 2. One patient with an ependymoma had sellar atrophy. Diagnostic bone changes were noted in the petrous pyramid and auditory canal of the patient with the eighth nerve neurinoma. Intratumoral calcifications were visible in 2 patients with ependymomas and in the patient with the ectopic pineal teratoma. Intratumor calcification was visible in the upper cervical canal in the patient with the ectopic pineal teratoma, and was clearly seen on the cervical air myelogram in one of the patients with tumorous extension of an ependymoma.

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Splenic artery injection gives consistently excellent filling of its pancreatic branches in the tail of the pancreas. The splenic vein, an important part of the pancreatic evaluation, is much better seen with the splenic artery injection.

The dorsal pancreatic artery (DP) is poorly visualized from all injection sites. The transverse pancreatic artery (TP) is well filled from both a gastroduodenal injection and a splenic injection, because of the anastomoses between them.

There is no great difference between our results and those of Boijsen[1,2] or Reuter[4] as regards the degree of normal pancreatic arterial filling at each superselective site. The results of those studies indicate that the branches in the head are well visualized from injection into the distal part of the common hepatic artery, and those in the tail, from injection into the splenic artery; only with both injections can the complete filling of all pancreatic arteries be achieved.

The superselective approach has several advantages: (a) more contrast material per unit time can be delivered to the organ under study which results in better angiographic detail; (b) there is no overlap of the vessels of other structures; and (c) the volume of contrast material per injection is decreased so that multiple injections and projections are possible.

It is necessary for serial magnification arteriography to prevent the appropriate vessels from overlapping and to achieve the complete filling of the smaller arteries. Also, the pharmacoangiography of the celiac arterial system may not become effective for detecting early malignant lesions until the catheter is introduced into the branches of the main trunk and the agent is injected only into the artery supplying one organ, as in renal pharmacoangiography. This point of view has developed in working with superselective angiography.

SUMMARY

The technique of transfemoral superselective celiac angiography using the Muller USCI deflector system is described.

The cases which can be catheterized superselectively with ease may be chosen by evaluating the vascular map of the celiac arterial system obtained from a conventional celiac injection. A superselective study should not be thought of as a replacement of conventional celiac angiography, but should supplement it.

An analysis of the opacification of the arteries in the various pancreatic areas obtained with the injection of contrast medium into the gastroduodenal artery, into the distal parts of the common hepatic artery, into the proper hepatic artery and into the splenic artery is made. The first 3 sites of injection give almost the same quality angiograms of the pancreatic head. A complete demonstration of the pancreatic arteries is achieved only by injecting the splenic artery as well as one of the other 3 sites.

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TABLE I

DISTRIBUTION OF TUMORS ACCORDING TO AGE OF PATIENT

Type of Tumor	Intraventricular Fourth Ventricle Tumors (total 16)			Extraventricular Tumors (total 3)
	16 mo.–10 yr.	11 yr.–19 yr.	20 yr.–39 yr.	40 yr.–54 yr.
Ependymomas	3	1	3	
Medulloblastomas	7			
Choroid plexus papilloma	1			
Ectopic pineal teratoma		1		
Hemangioblastoma				2
Acoustic neurinoma				1

BRAIN SCANNING

Brain scans were available in 5 patients, and were positive in all. Three had ependymomas, 1 had a medulloblastoma and 1 had an acoustic neurinoma.

LUMBAR PNEUMOENCEPHALOGRAPHY

Lumbar pneumoencephalograms were available in 7 patients. Of these, 5 were "failed pneumoencephalograms," which nevertheless disclosed narrowing of the pontine cistern and changes in the pericallosal and ambient wing cisterns indicative of hydrocephalus. In 2 patients, a 4 year old boy and a 5 year old girl with intraventricular ependymomas, air entered the fourth ventricle and outlined tumor in the dilated chamber. Moderate hydrocephalus was demonstrated.

In patients with "failed pneumoencephalograms," cervical air myelograms were interpreted as showing tonsillar herniation in 4. Calcification in the distal tip of one of these, a 20 year old woman with an ependymoma (Fig. 1, *A–D*), suggested that the intrusion was tumorous rather than tonsillar displacement. The presence of tumorous extension rather than tonsillar herniation was identified at necropsy in a 6 year old girl with a papilloma (Fig. 2, *A* and *B*). In the other 2 patients tonsillar herniation was disclosed at operation.

VENTRICULOGRAPHY

Ventriculograms were available in 11 patients. Nine had gross hydrocephalus. In 9 the third ventricle was markedly dilated, and impingement against its posterior aspect incident to bulging of the in-



FIG. 1. This 20 year old woman complained of headache, throbbing occipital pain radiating to the parietal region and to the neck, nausea, vomiting and blurred vision, for about 1 year. Bilateral papilledema was present. Skull plain roentgenograms were negative.

(*A*) Air cervical myelogram shows a soft tissue mass extending from the foramen magnum to the level of the superior margin of the lamina of C2. Just beneath the neural arch of C1 a pointed calcareous shadow is visible.

CELIAC EPINEPHRINE ENHANCED ANGIOGRAPHY*

By JÜRI V. KAUDE, M.D.,† *and* GEORGE W. WIRTANEN, M.D.‡
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ADRENOTROPIC receptors are hypothetical structures or systems located in, on, or near muscle or gland cells affected by epinephrine. They are termed alpha and beta receptors and they react variously to epinephrine, the sympathomimetic agents, and to the effects of sympathetic nerve stimulation.[2] The vascular reaction is exerted on the smaller arterioles and precapillary vessels, and also on larger arteries and veins.[8] In the liver and spleen, epinephrine constricts the vessels by reaction on alpha receptors, while the vessels in the pancreas appear to lack this constrictive response to epinephrine.[6]

The nonconstrictive response of neoplastic vessels to intraarterially applied epinephrine has been found to be useful in angiographic diagnosis of renal, gastrointestinal, and other abdominal tumors.[1,4,9–11]

Boijsen and Redman[6] described the effect of epinephrine in angiography of the celiac axis and superior mesenteric artery, and in some patients demonstrated abdominal tumors better, following epinephrine injection, but they concluded that the study could not give definitive answers about the general usefulness of the method.

The present study was undertaken to investigate the use of epinephrine in a larger number of hepatic, biliary, and pancreatic neoplasms.

MATERIAL AND METHOD

We examined 70 patients with known or suspected pancreatic, hepatic, or biliary neoplasms angiographically, with and without intraarterially administered epinephrine injection. Four patients had examination of 2 vessels, so that the material available for review represents 148 angiograms (Table 1).

TABLE I

SITE OF INJECTION IN EPINEPHRINE ANGIOGRAPHY IN 70 PATIENTS*

Celiac axis	34
Common hepatic artery†	21
Proper hepatic artery†	6
Right hepatic artery	6
Left hepatic artery	1
Splenic artery	2
Superior mesenteric artery	4
Total examinations	74

* Four patients examined with injection into 2 arteries.
† Terminology as used by Lunderquist.[13]

TABLE II

SUMMARY OF PATHOLOGIC ENTITIES EXAMINED

1. Hepatic Tumors		
A. Primary tumors		5
Hepatoma, malignant	3	
Hemangiosarcoma	1	
Leiomyosarcoma	1	
B. Metastatic tumors*		41
2. Pancreatic tumors		14
Carcinoma of the pancreas	12	
Insulinoma, benign	1	
Zollinger-Ellison tumor	1	
3. Carcinoma of the gallbladder or of the bile ducts		7
4. Recurrent adrenal carcinoma		1
5. Neurofibroma of the mesentery		1
6. Other conditions		4
Cirrhosis of the liver	3	
Chronic pancreatitis	1	
7. Normal studies		11
Totals		84

* Seven patients with primary pancreatic carcinoma, 6 patients with gallbladder or bile duct carcinoma, and 1 patient with recurrent adrenal carcinoma, are included here as well as under Headings 2, 3, and 4.

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Supported in part by National Cancer Institute, Public Health Service Grant 5-P02-CA-06295-RAD.
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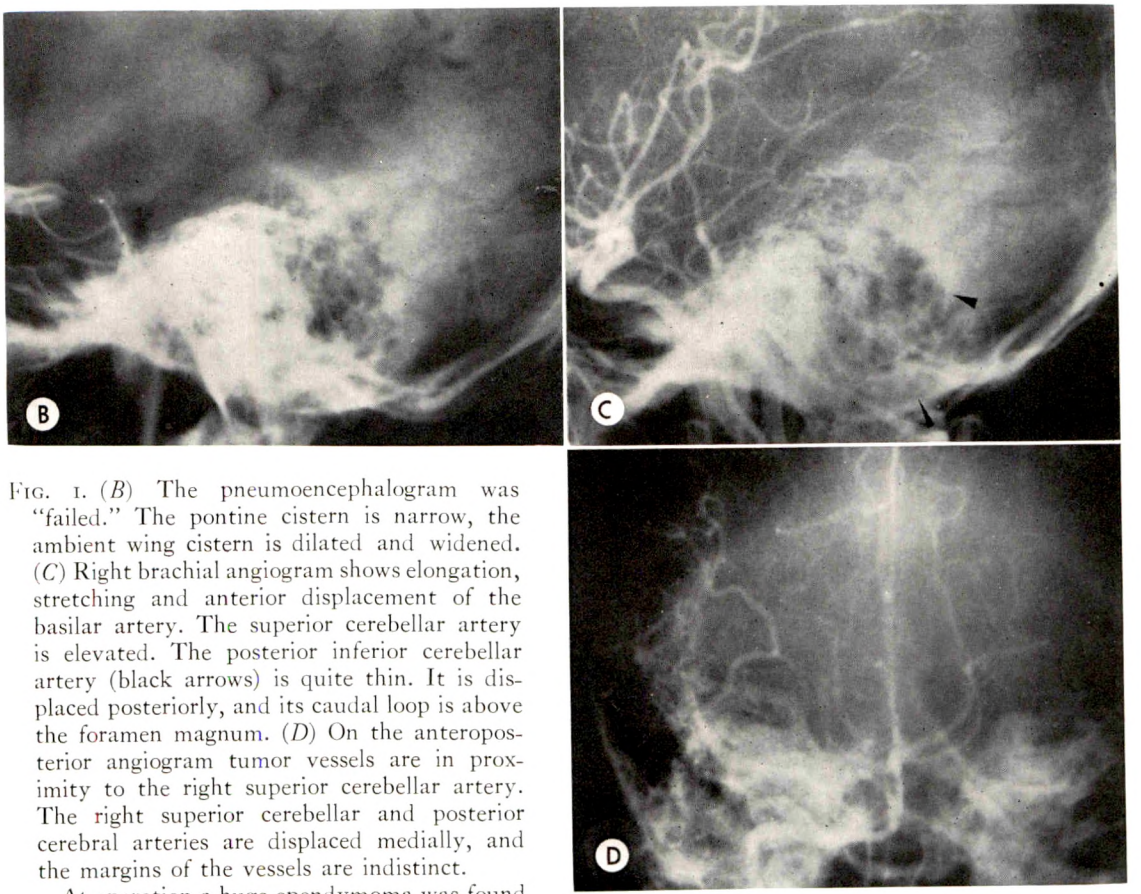


Fig. 1. (*B*) The pneumoencephalogram was "failed." The pontine cistern is narrow, the ambient wing cistern is dilated and widened. (*C*) Right brachial angiogram shows elongation, stretching and anterior displacement of the basilar artery. The superior cerebellar artery is elevated. The posterior inferior cerebellar artery (black arrows) is quite thin. It is displaced posteriorly, and its caudal loop is above the foramen magnum. (*D*) On the anteroposterior angiogram tumor vessels are in proximity to the right superior cerebellar artery. The right superior cerebellar and posterior cerebral arteries are displaced medially, and the margins of the vessels are indistinct.

At operation a huge ependymoma was found completely filling the fourth ventricle. It arose from the floor, and extended through the foramen of Magendie well down into the cervical spinal canal. The cerebellar tonsils were elevated and displaced laterally. The vermis was flattened and sharply elevated.

traventricular fourth ventricle tumor was noted in 3. In 6 the sylvian aqueduct was shortened, dilated, relatively horizontal in position, and presented a slight irregularity along its superior surface. The distal end of the aqueduct was slightly wider than its proximal portion. Gas entered the fourth ventricle in 9 patients, outlining the superior, the posterior and anterior aspects of the cephalad surface of the tumor. This "capping" of the mass localized the lesion, and was accompanied by pronounced dilatation of the chamber. Inasmuch as air did not pass out of the fourth ventricle because of impaction of the tumor against the foramina of Magendie and Luschka, it was impossible to identify tonsillar or tumorous extension into the cervical spinal canal. The fourth ventricle maintained a relatively normal position, but a lateral shift was seen in frontal projections in 4 patients in whom the tumor involved one side more than the other.

ANGIOGRAPHY

Posterior fossa angiograms were available in 6 patients, including 3 with ependymomas, 1 with a medulloblastoma, 1 with a tonsillar hemangioblastoma and 1 with an acoustic neurinoma. Tumor staining was visible with certainty in 2 patients with ependymomas, and was questionable in another. The patient with the acoustic neurinoma had a well defined tumor stain. In the patient with the vermis hemangioblastoma the tumor was well outlined, and

The patients comprise 2 distinct groups. The University of Wisconsin material represents 34 patients, all with known primary or metastatic hepatic tumor. Percutaneous transbrachial catheter exchange was carried out with nonradiopaque polyethylene catheters with internal diameters of 1.4 mm. and external diameters of 1.9 mm. Films were exposed in anteroposterior projection on an Elema-Schönander roll film changer, employing 45–50 cc. of 76 per cent meglumine diatrizoate, injected at a rate of approximately 5 cc. per second. The film rate was 1 film every 2 seconds for 20 seconds, and 1 film every 5 seconds for 10 seconds. This technique with slow injection and film rate was used to avoid recoil of the relatively long end-hole catheter, which was left in place for intra-arterial chemotherapeutic infusion.[15] Intra-arterial epinephrine injection in doses of 6 μg. was used in all patients, except in 1 with massive hepatomegaly, in whom a dose of 10 μg. was used. The University of Florida material represents 36 patients who had transfemoral percutaneous Kifa red thin-wall catheter exchange, employing 32–38 cc. of 76 per cent meglumine diatrizoate, injected in 4 seconds. Serial angiography was carried out with patients in anteroposterior or right posterior oblique projection with an Elema-Schönander cut film changer, making 3 exposures per second for 4 seconds, followed by 1 exposure per second for 4 seconds, and an additional 8 to 10 exposures in 4 or 5 seconds. Epi-

TABLE III

SITE OF PRIMARY LESION IN 41 PATIENTS WITH HEPATIC METASTASES

Carcinoma of the colon	9
Carcinoma of the rectum	2
Carcinoma of the gallbladder or bile ducts	6
Carcinoma of the pancreas	7
Carcinoma of the kidney	1
Carcinoma of the adrenal	2
Leiomyosarcoma of the stomach	1
Bronchogenic carcinoma	4
Carcinoma of the breast	6
Anaplastic carcinoma, primary lesion unknown	3
	41

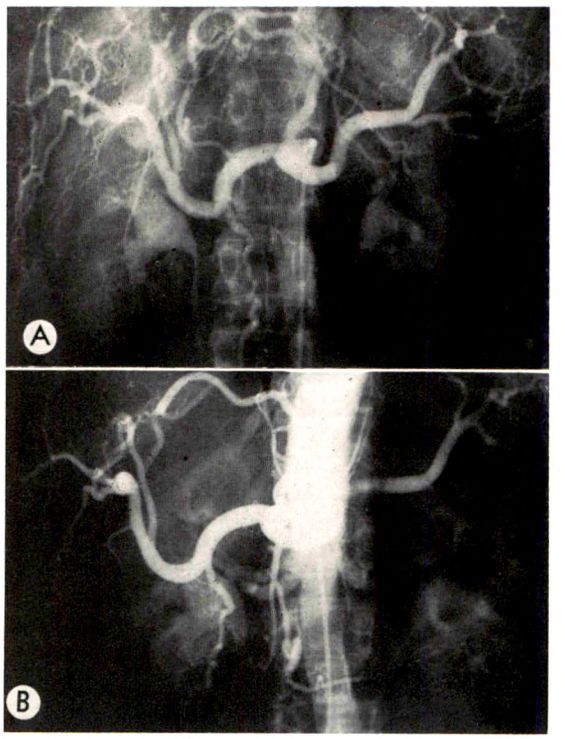


FIG. 1. Epinephrine effect in celiac angiogram in a patient with carcinoma of the head of the pancreas with liver metastases. Films are exposed 3 seconds following the injection of contrast medium. (*A*) Irregular stenosis of the gastroduodenal artery is present and faintly opacified pancreaticoduodenal vessels are visualized without epinephrine. The liver is enlarged and multiple metastatic lesions are present. (*B*) The repeat angiogram was done 20 seconds following the injection of 5 μg. epinephrine with the patient in right posterior oblique position. There is constriction of the peripheral hepatic, splenic and gastric vessels with contrast reflux into the aorta. The inferior phrenic arteries fill. The proximal arterial segments have not reacted and are filled with contrast medium in greater concentration. Tumor involvement of the gastroduodenal artery is evident, pancreatic neovascularity is better demonstrated, and with dorsal pancreatic artery opacification, tumor involvement in its distal segment is demonstrated. The cystic artery is more easily identified; the gallbladder is enlarged. Hepatic metastases could not be demonstrated with epinephrine with the peripheral hepatic artery vasoconstriction, and with diminished flow of contrast medium to the spleen, the splenic vein was not visualized.

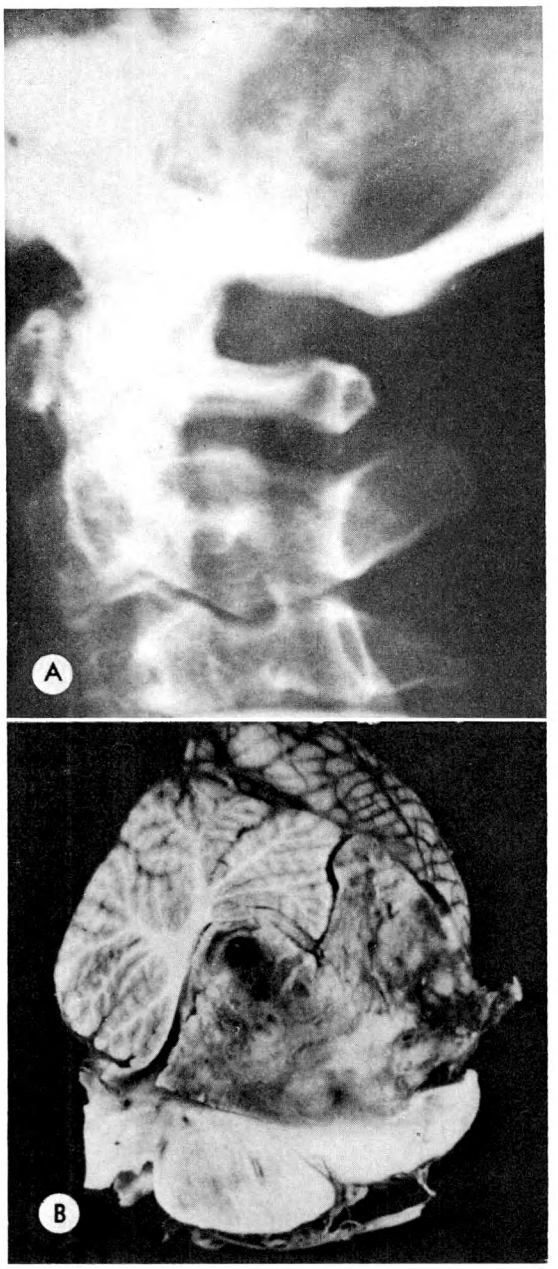


FIG. 2. A 6 year old girl with a papilloma of the choroid plexus which completely filled the fourth ventricle, emerged from the foramen of Magendie, and extended to a lesser extent laterally through the foramina of Luschka. The cerebellar tonsils were practically obliterated. The vermis was sharply elevated and flattened. Death was caused by marked brain stem compression. A lumbar pneumoencephalogram was "failed."

(*A*) A soft tissue density is seen immediately beneath the foramen magnum. This had been diagnosed as tonsillar herniation. (*B*) Sagittal cut of the tumor *in situ*.

a portion of its cystic component extended well below the foramen magnum (Fig. 3, *A* and *B*). The basilar artery was displaced anteriorly in 5 patients. In 1, a 6 year old boy with a medulloblastoma, neither tumor staining nor vascular displacement was visible. This patient had a "failed pneumoencephalogram."

The position of the posterior inferior cerebellar artery was of special interest because of its relationship to the medulla, the cerebellar tonsils and the vermis. In the patient with the tonsillar hemangioblastoma which extended below the foramen magnum the posterior inferior cerebellar artery, which supplied the tumor, was dilated and elevated by the tumor (Fig. 3*A*). Herniation of the tumor through the foramen magnum occurred anterolaterally in the patient with the acoustic neurinoma. This patient also had tonsillar herniation, and the caudal loop of the posterior inferior cerebellar artery was displaced downwards.

Two patients with ependymomas and herniation of tumor into the cervical spinal canal had changes in the configuration and position of the posterior inferior cerebellar arteries. In 1 the caliber of the vessel was diminished, and its caudal loop was shifted anteriorly and upwards. The cephalad loop was displaced posteriorly and downwards (Fig. 1*C*). In the other the artery was prominent. Its caudal loop was at the level of the foramen magnum, but its cephalad loop was altered so that the posterior medial branch, the supratonsillar and the vermian branches were depressed (Fig. 4, *A–D*). A third patient with an ependymoma had tonsillar herniation only. Here the posterior inferior cerebellar artery was well formed. Its caudal loop was elevated and the cephalad loop, particularly the posterior medullary, the supratonsillar and the vermian segments were displaced downwards, posteriorly and laterally (Fig. 5, *A–C*).

ANATOMIC OBSERVATIONS

All the intraventricular fourth ventricle tumors in this series were large. Points of attachment were found in the floor of the

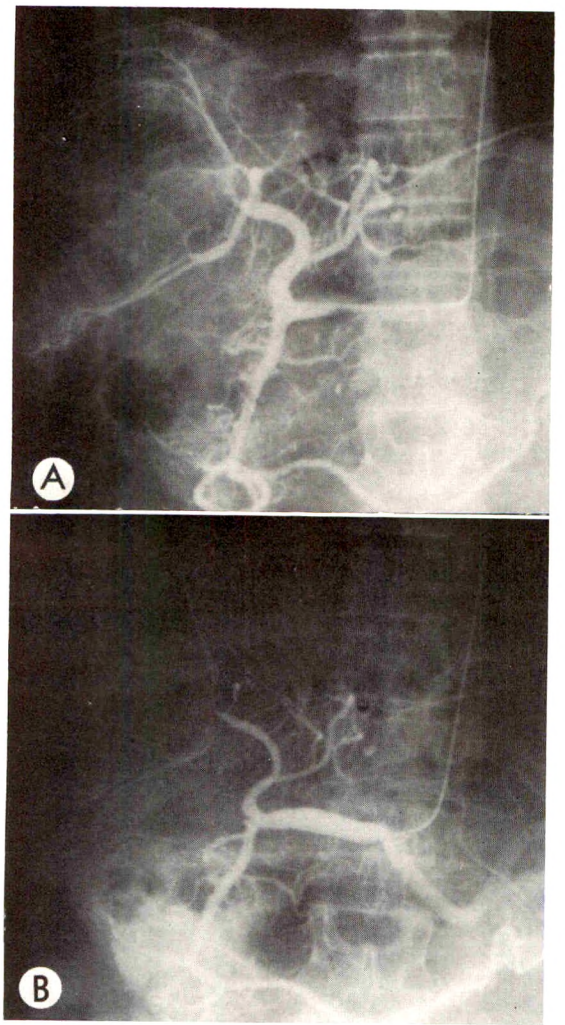


FIG. 2. A 59 year old male with undifferentiated liver metastases from an unknown primary lesion.

Hepatic angiograms (*A*) without and (*B*) with epinephrine. Following epinephrine the hepatic arteries demonstrate a moderate proximal and distal constriction with diminished contrast flow into the liver, resulting in loss of information regarding the liver metastases. The contrast medium refluxes to the proximal portion of the common hepatic artery and into the splenic artery and there is intense duodenal contrast staining.

nephrine injection dosage varied from 4 to 8 μg.

In both patient groups, angiographies were repeated with the same film techniques and sequences, with contrast material injected 20 seconds following the epinephrine injection.

RESULTS

Table II lists the pathologic entities i cluded in this study, and Table III sur marizes the origin of tumor in the 41 p tients with metastatic hepatic carcinom The positive angiographic diagnosis w confirmed histologically in all cases, exce in 1 patient roentgenographically dia nosed as carcinoma of the pancreas, wh was considered a poor surgical risk. In examinations, the peripheral hepatic ar splenic vessels were constricted in all i

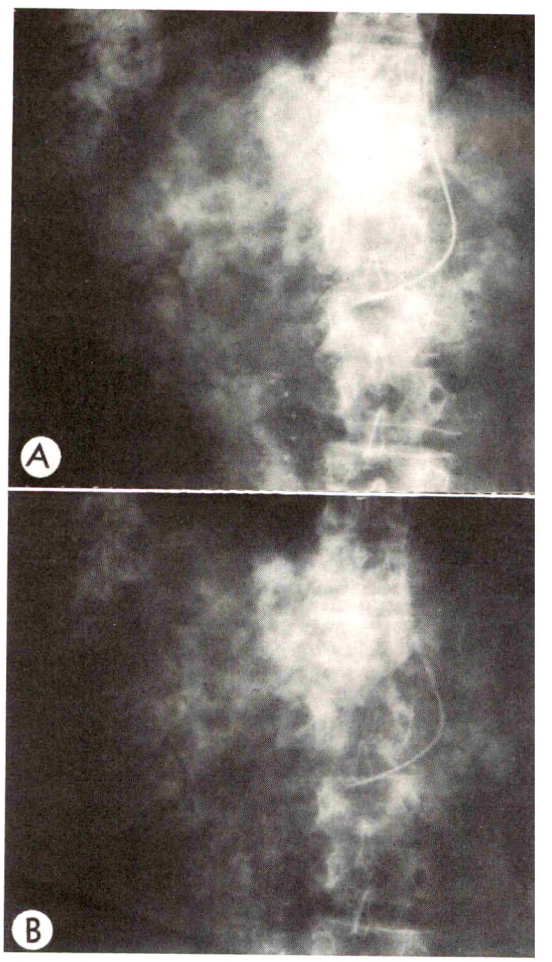


FIG. 3. A 60 year old female with hepatic metastase from an unknown primary carcinoma.

(*A*) There is extensive neovascularization dem onstrated throughout both lobes of the liver whe contrast material is injected into the prope hepatic artery.[13] (*B*) Following the intraarteria administration of 6 μg. epinephrine, the volume o the neovascular tumor is reduced.

fourth ventricle in 2 patients with ependymomas. In the 2 patients with medulloblastomas the tumors were adherent to the floor of the fourth ventricle. The roof and lateral walls also were involved in 3 other patients with ependymomas which had invaded the peduncles and the cerebellum. The other 9 tumors also were large. Most of them formed casts of the fourth ventricle with variable invasion of adjacent structures. This permitted surgical excision of some of these tumors, with a fairly good prognosis in those with ependymomas. The pineal teratoma had grown into the sylvian aqueduct as well as down the posterior aspect of the cervical canal, and had invaded the peduncles. Fatal hemorrhages into the tumor occurred in 1 patient with a medulloblastoma and another with an ependymoma.

A wide variety of tumors originate within the fourth ventricle. Ependymomas, medulloblastomas and papillomas are most frequent. Hemangioblastomas, astrocytomas, cholesteatomas[7,8] and oligodendrogliomas[6] also have been found. An intraventricular ectopic pinealoma was observed in the present series.

The most characteristic tumor, the ependymoma, has a tendency to originate in the midline of the floor of the fourth ventricle. It grows slowly, remains circumscribed to a greater or lesser extent, fills and bulges the chamber and causes foraminal obstruction and dilatation. Other intraventricular tumors may have similar characteristics, but may be more invasive. The tendency for tumors within the fourth ventricle to protrude through the foramina of Magendie and Luschka has been mentioned briefly. Cushing[3] and Russell and Rubenstein[9] published illustrations of ependymomas with spinal tongues. Boldrey and Miller[1] observed extension through the foramen of Magendie to the level of the lamina of the second cervical vertebra. Wickbom and Hanafee,[11] in a survey of 18 intraventricular fourth ventricle tumors occurring in a series of 153 posterior fossa tumors, found extension below the foramen magnum in 7. In 1 of the cases of cholesteatoma reported by Patriquin *et al.*[8] tumor extended into both cerebellopontine angles and passed laterally to the midline, surrounding the pons and the medulla.

Extraventricular tumors also may extend beyond the foramen magnum into the upper cervical spinal canal. Wickbom and Hanafee[11] mentioned that of 36 cerebellar hemispheric tumors there were 22 with depressed tonsils and 3 with tumor extending below the foramen magnum. Of 13

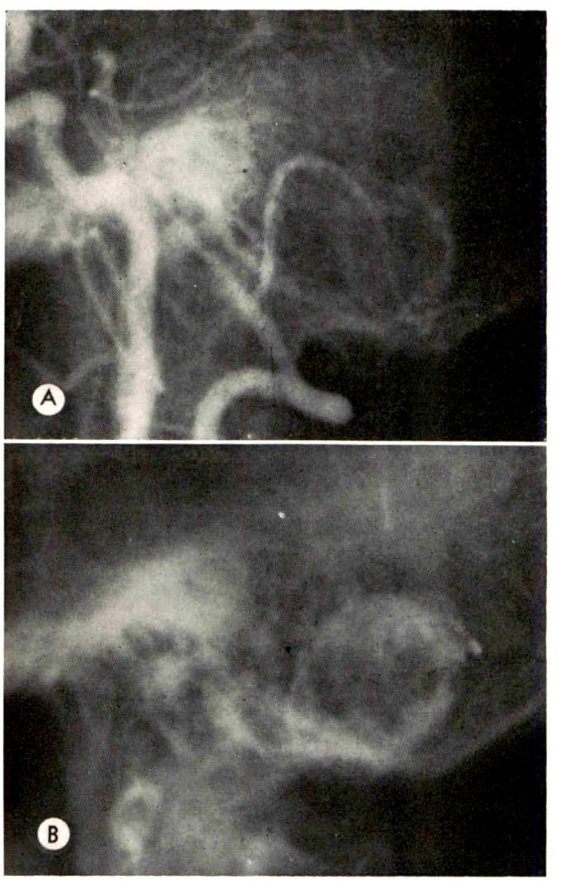


Fig. 3. A 52 year old woman with a hemangioblastoma which originated in the right cerebellar tonsil and extended into the hemisphere and vermis. The solid portion of the tumor was imbedded in the cerebellum, but a portion of the cystic element extended below the foramen magnum.

(*A*) Right brachial angiogram shows dilatation of the posterior inferior cerebellar artery, with posterior displacement of the caudal loop. Tumor vessels outline the posterior portion of the cyst. (*B*) In the later phases of the examination the inferior margin of the cyst extends below the foramen magnum.

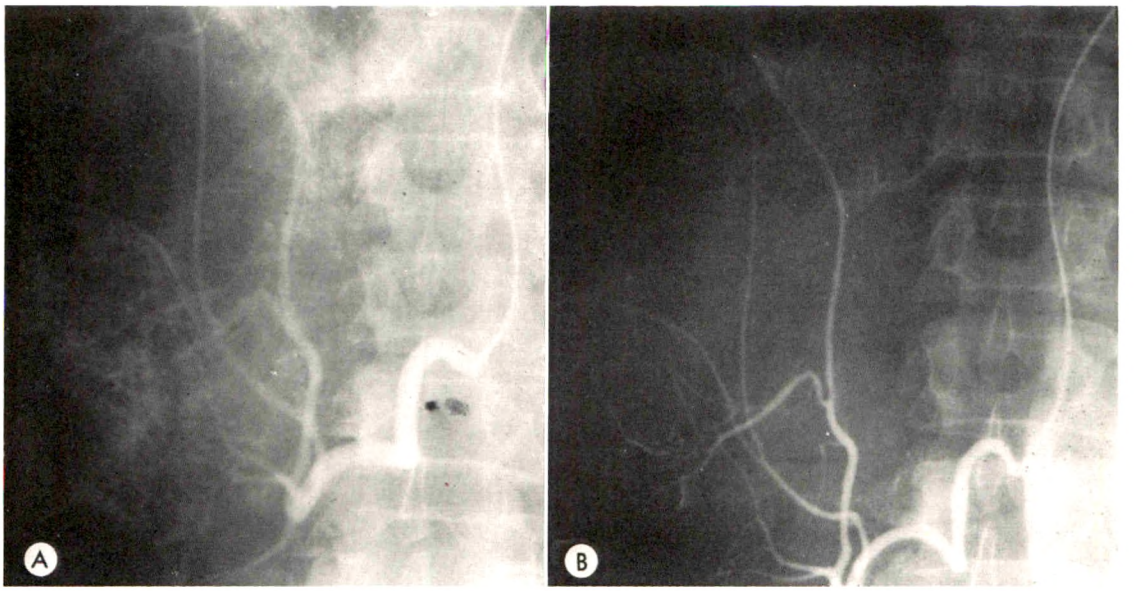


Fig. 4. A 67 year old male with adenocarcinoma of the colon, metastatic to the liver.
(*A*) The accessory right hepatic angiogram demonstrates large areas of neovascularity in the right lobe of the liver. The hepatic veins were opacified during the venous phase. (*B*) A 6 μg. intraarterial epinephrine injection produced an approximate 50 per cent reduction in the diameter of the artery. Most of the contrast medium refluxed into the superior mesenteric artery and opacified the superior mesenteric and portal vein in the venous phase.

stances except 2, when epinephrine was injected into the celiac axis (Fig. 1, *A* and *B*). The degree and site of constriction were variable, persisting for approximately 25–45 seconds. A marked vasoconstriction of the proximal larger arteries was noted in 6 studies. Arterial flow time was prolonged in relation to the degree of vasoconstriction, and some reflux of contrast medium into the aorta usually occurred, which resulted in poorer visualization of the splenic and portal veins. With selective injection of epinephrine and contrast medium into the hepatic arteries in 34 examinations, the diameter of the proximal larger vessels was reduced approximately 25–60 per cent in 24 patients (Fig. 2, *A* and *B*; and 4, *A* and *B*), unchanged in 9, and dilated in 1. Ten patients had marked vasoconstriction of the peripheral vessels. The contrast medium was diverted to the spleen, pancreas, duodenum, stomach, diaphragm, and adrenals, with selective hepatic artery injection following epinephrine. This made examination of the arterial phase in the liver less advantageous, but filling of more proximal axis vessels made the pancreatic vessels sufficiently contrast medium loaded to allow angiographic evaluation of this organ, and during the venous phase the portal vein opacified. The size of the liver was reduced in 2 patients following epinephrine injection, with a decrease in the volume of pathologic vasculature in 1 patient (Fig. 3, *A* and *B*). With epinephrine injected into an accessory right hepatic artery, there is an approximate 50 per cent reduction in the diameter of the vessel, with reflux into the superior mesenteric artery, and subsequent opacification of the superior mesenteric and portal veins (Fig. 4, *A* and *B*). In 2 examinations, no difference in size of the arteries, before or after the injection of epinephrine, was noted. In 55 examinations with injection into the celiac axis or into the common hepatic artery, the gastroduodenal artery reacted with vasoconstriction in only 8 cases, but usually constriction of the proximal right gastroepiploic artery occurred immediately after its origin from the

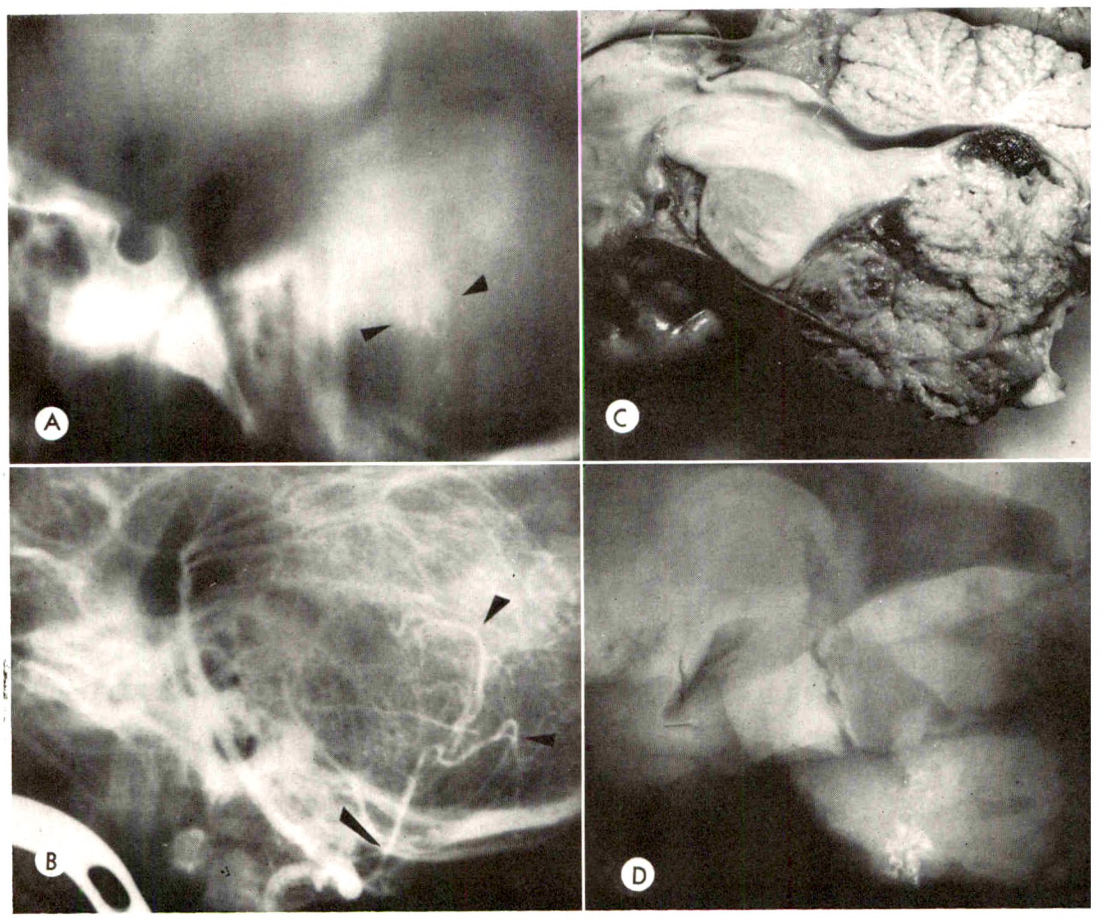


FIG. 4. A 4 year old boy with a history of anorexia, personality changes, and a mild degree of ataxia for ab 2 months, had an ependymoma which completely replaced the fourth ventricle. A lumbar pneumo cephalogram was "failed."

(*A*) Midline laminagram reveals calcification in the posterior fossa (black arrows). The cisterna ponti narrowed. (*B*) Right brachial angiogram shows the dilated posterior inferior cerebellar artery displa posteriorly. The caudal loop is at the level of the foramen magnum. Tumor vessels emerge from the verm and hemispheric branches. On later studies a definite tumor stain appeared in this area. (*C*) A h ependymoma completely occupied the fourth ventricle, and had grown through it to invade the medi and pons. (*D*) Roentgenogram of the specimen shows calcification well below the level of the foramer Magendie. The tumor had emerged from the foramina of Magendie and Luschka, practically wrapped self around the brain stem, invaded the peduncles and blocked the sylvian aqueduct by extending upwa

vermis tumors, tonsillar herniation occurred in 10, but there were none that traversed the foramen magnum. That this can occur is illustrated by the vermis hemangioma in the present series.

DISCUSSION

Intraventricular fourth ventricle tumors, because of their size and position, spread the cerebellar tonsils apart and elevate the inferior portion of the vermis. This process is exaggerated when tumor projects out the foramina, particularly the midl foramen of Magendie. In some patients t net effect of such intrusion not only exerted on the cerebellar structures l also involves the pontomedullary area a may extend out into the cerebellopont angles. Complete encirclement of the n dulla may be encountered when tun emerges from both the foramina of Lusch and Magendie. In the patients in the pr

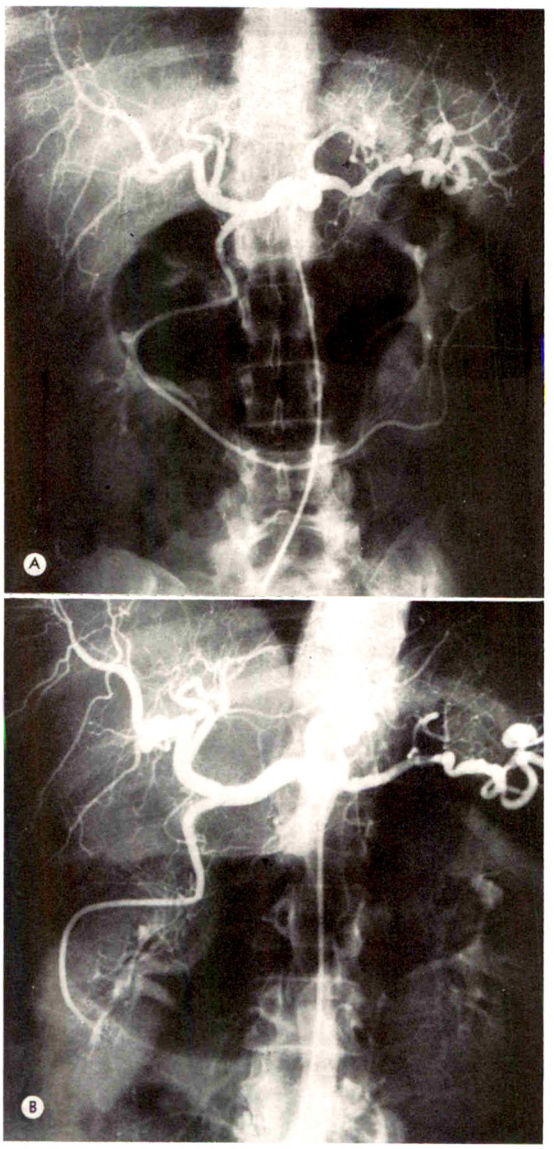


FIG. 5. A 48 year old female with extensive carcinoma of the pancreas.

(*A*) Celiac angiography demonstrates a long, irregular stenotic segment of the splenic artery. The pancreatic vessels are poorly visualized. Numerous thin stretched vessels are present in the head of the pancreas. The patient had hepatic metastases evident on late angiograms and a splenic artery aneurysm. (*B*) The angiogram following the administration of 8 μg. epinephrine in slight right posterior oblique position accentuates the involvement of the splenic artery. There is also irregular stenosis of the thin superior posterior pancreaticoduodenal artery. Marked vasoconstriction of the right gastroepiploic artery was present in most epinephrine enhanced angiograms.

gastroduodenal artery (Fig. 5, *A* and *B*). In the absence of vasoconstriction of the arteries supplying the duodenum and pancreas, therefore, better filling of the pancreaticoduodenal arteries was obtained, with accumulation of contrast being especially intense in the duodenum (Fig. 2, *A* and *B*). Individual tumor nodules were frequently more clearly defined, as in the detail from the left lobe of the liver in 1 patient (Fig. 6, *A* and *B*) and in the right lobe in another patient (Fig. 7, *A* and *B*).

The cystic artery was identified angiographically in 42 of 65 patients, and reacted with moderate vasoconstriction in 11 patients. The gallbladder and the cystic artery were more easily identified with epinephrine vasoconstriction of the arteries in the right lobe of the liver, as were bile duct and gallbladder carcinomas (Fig. 8, *A* and *B*).

Selective splenic epinephrine angiography diverted contrast material into the hepatic arteries, so that less contrast medium was available for opacification of the splenic and portal veins, and, in 1 patient, a measurable reduction in the size of the spleen was observed.[8,11]

The superior mesenteric artery was examined in 4 patients, and moderate constriction of the peripheral branches was noted; this also resulted in aortic regurgitation of contrast material. Epinephrine did not enhance visualization of the inferior pancreaticoduodenal arteries.

In 3 patients, some information was added, and some lost, as in an angiogram of a carcinoma of the pancreas in which the tumor was better demonstrated with epinephrine, and the liver metastases from the same tumor were better demonstrated without epinephrine (Fig. 1, *A* and *B*).

Epinephrine angiography added significant diagnostic information regarding the extent of a pancreatic tumor in 3 patients. In 5 additional patients, the infiltration of vessels by tumor and the pathologic vascularity were accentuated with the epinephrine study[12] (Fig. 1, *A* and *B*; and

ent series extension of tumor varied from a fairly large nodule traversing the foramen of Magendie to a huge mass of encircling tumor. Downward extension into the cervical spinal canal occurs with tumorous passage into the vallecula and thence as low as the second cervical vertebra.

Protrusion of tumor tissue from within the fourth ventricle was observed at operation in 10 patients, and further confirmed at necropsy in 5 of these. In 6 patients tumor protruded from both the foramina of Magendie and Luschka, while in 2 protrusion appeared only at the foramen of Magendie. In 1 patient tumor emerged from both foramina of Luschka only, and in 1 other from only one of the lateral foramina. Tumor escaping from the foramen of Magendie was much more bulky than from the lateral foramina. When tumor passed out of the lateral foramina it curled around the pontomedullary area. Tumorous extension dilated the respective foramina. There were 6 patients in whom intraventricular fourth ventricle tumors approached and occluded the openings from the chamber. In these, also, the tonsils were separated and the vermis elevated, but to a lesser extent than some of the bulky tumors which had entered the intertonsillar area.

Among the extracerebellar tumors to be considered in differential diagnosis are included spinocranial and craniospinal tu-

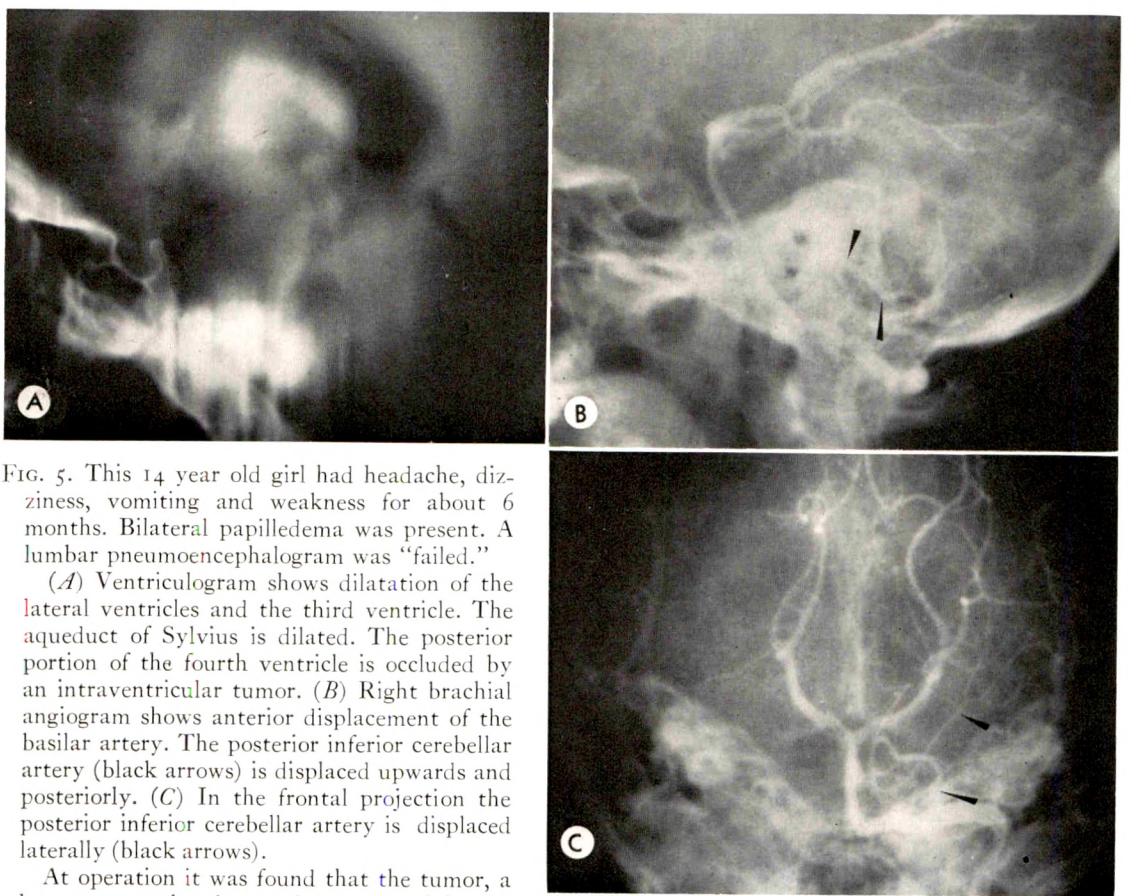


Fig. 5. This 14 year old girl had headache, dizziness, vomiting and weakness for about 6 months. Bilateral papilledema was present. A lumbar pneumoencephalogram was "failed."

(*A*) Ventriculogram shows dilatation of the lateral ventricles and the third ventricle. The aqueduct of Sylvius is dilated. The posterior portion of the fourth ventricle is occluded by an intraventricular tumor. (*B*) Right brachial angiogram shows anterior displacement of the basilar artery. The posterior inferior cerebellar artery (black arrows) is displaced upwards and posteriorly. (*C*) In the frontal projection the posterior inferior cerebellar artery is displaced laterally (black arrows).

At operation it was found that the tumor, a large encapsulated ependymoma, originated from the floor of the fourth ventricle. It practically filled but did not extend outside the chamber. Tonsillar herniation reaching the superior rim of the neural arch of the second cervical vertebra was present. However, the tumor displaced the tips of the tonsils posterolaterally as well as downward, thereby shifting the posterior inferior cerebellar arteries upwards and posteriorly.

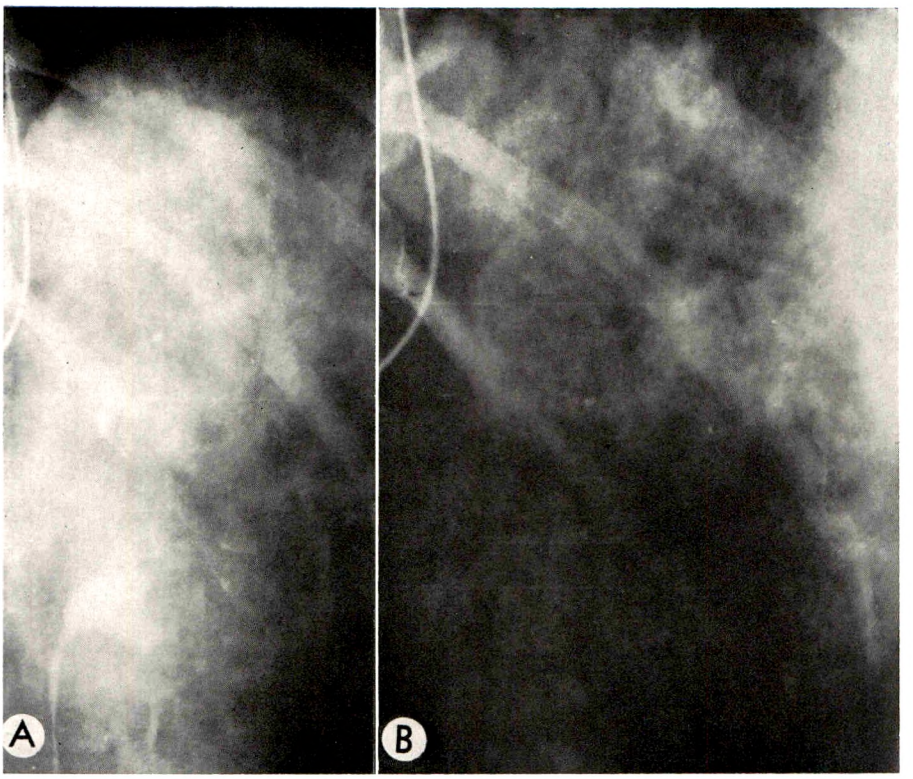


FIG. 6. A 32 year old female with adenocarcinoma of the colon, metastatic to the liver.

(*A*) The left lobe of the liver is enlarged, and the irregular, nonhomogeneous hepatogram indicates metastatic involvement. (*B*) Individual tumor nodules are better demonstrated, utilizing 6 μg. epinephrine intra-arterially prior to the injection of contrast material.

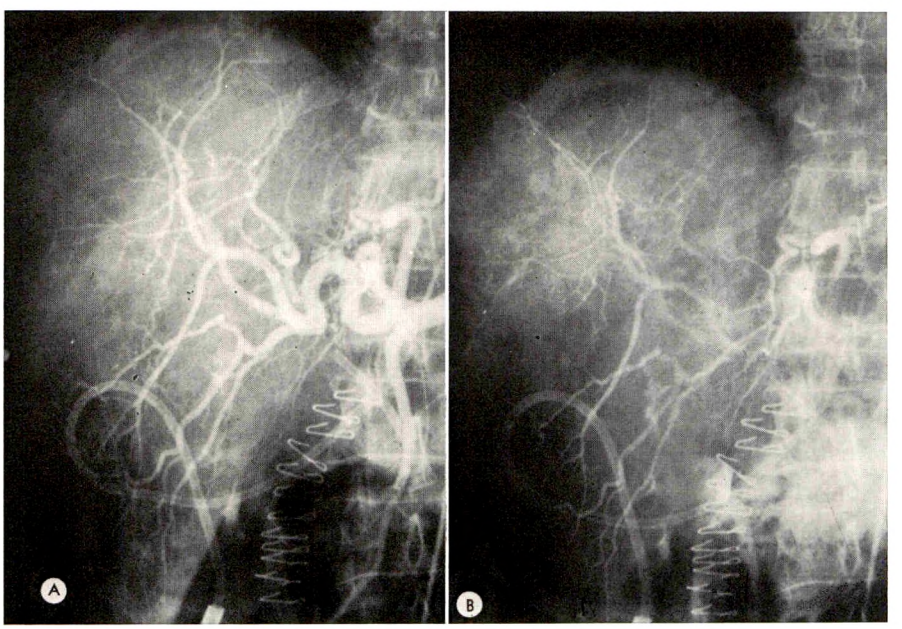


FIG. 7. A 67 year old male with bile duct carcinoma and liver metastases.

(*A*) The hepatic arteries are displaced by poorly vascularized expanding lesions in the angiogram without epinephrine. (*B*) Following 6 μg. epinephrine, contrast medium is diverted into a large area of metastases laterally in the right lobe.

mors,[4,10] particularly neurofibromas and meningiomas, and large aneurysms of the vertebral artery.[2] The Arnold Chiari malformation also should be kept in mind in the consideration of soft tissue masses in the upper cervical spinal canal. The present series includes a patient with a huge auditory nerve neurinoma which passed anterolaterally through the foramen magnum, and had tonsillar herniation as well.

The demonstration of calcification within tumors of the fourth ventricle is not uncommon in ependymomas, and has been found in oligodenrogliomas. It was also noted in the patient with an ectopic pineal teratoma in the present series. However, calcification within spinal extension of tumors emerging from the fourth ventricle has not been mentioned previously insofar as we know. Two such instances are reported here.

SUMMARY

Intraventricular fourth ventricle tumors may be accompanied by emergence of tumor from the foramen of Magendie into the cervical spinal canal. This produces an appearance on air myelograms indistinguishable from tonsillar herniation, which also may be present. In 2 cases seen here, calcification present in tumor intruding into the cervical canal was visible on plain film roentgenograms. Because of the spreading of the tonsils and upward displacement of the vermis incident to intraventricular fourth ventricle tumors, the posterior inferior cerebellar arteries may be shifted upwards, posteriorly and laterally. If the tumor is supplied by these vessels, they may become dilated as well as displaced. If the tumor compresses the branches of the posterior inferior cerebellar artery, they become attenuated.

When a combination of a soft tissue mass in the cervical canal, failure of descent of the posterior inferior cerebellar arteries, and a midline tumor stain is noted, the possibility of an intraventricular fourth ventricle tumor bears consideration. This usually can be substantiated by means of pneumoventriculography.

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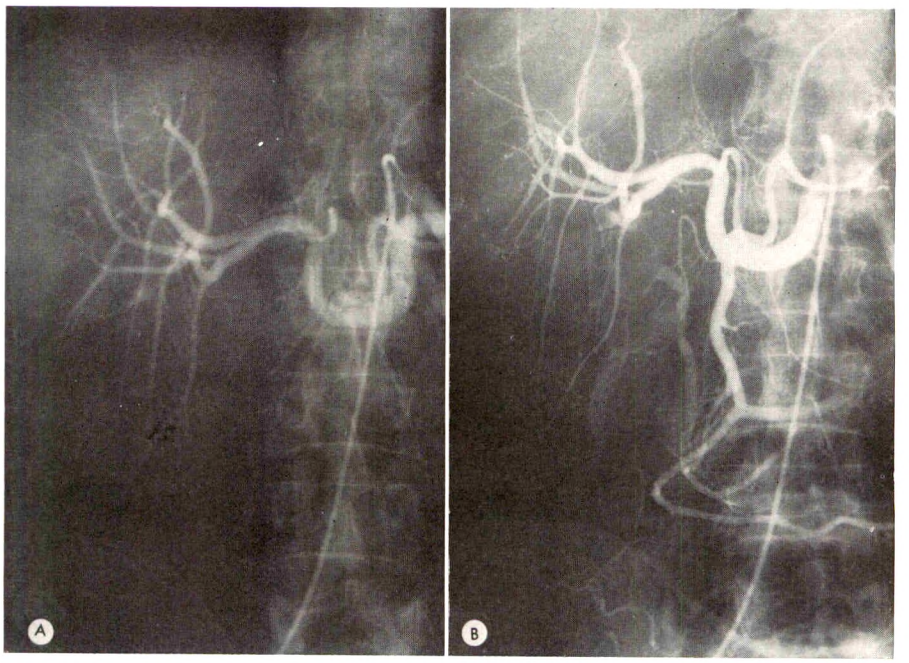


FIG. 8. A 67 year old male with obstructive jaundice and hepatomegaly secondary to intrahepatic bile duct carcinoma.

(*A*) The mid portion of the left lobe of the liver is poorly vascularized without definite evidence of tumor vasculature. The cystic artery is faintly opacified. The gastroduodenal artery and pancreatic vessels are not visualized because of reversed blood flow from the superior mesenteric artery to supply the enlarged liver. (*B*) Following 6 μg. epinephrine, small pathologic vessels are filled medially in the left lobe of the liver. The cystic artery opacifies and the gallbladder is small and its wall is partially collapsed, indicating proximal bile duct obstruction. The gastroduodenal artery and pancreaticoduodenal arteries are now filled and, except for enlarged arteries in the lower pancreatic arcade secondary to increased flow from the superior mesenteric artery, no pathologic changes are present in the pancreas.

5, *A* and *B*). It is of interest that the irregular stenotic portions of major arteries involved by the carcinoma reacted to epinephrine with vasoconstriction. This suggests that the changes in the arterial wall may not be due to direct invasion by tumor, but may be caused by perivascular tumor growth, or by tumor growth along the lymphatics of the arterial wall, as proposed by Boijsen and Reuter.[7] The improvement in the diagnostic quality in the angiography of the pancreas approached that of pancreatic angiograms done with selective catheterization of the gastroduodenal artery.[3,5,14]

Retrograde filling of the portal vein in the presence of liver metastases was present in 1 angiogram done only without epinephrine (Fig. 9, *A* and *B*). This communication was either closed when the drug was used, or, as is more likely, was the result of smaller quantities of contrast material reaching the level of flow reversal. In 1 patient with adrenal carcinoma, epinephrine was useful in better visualization of recurrent tumor following surgery by providing contrast medium reflux into the inferior phrenic artery. Diagnostic information was added in 11 patients utilizing epinephrine (Table IV), while in 7 patients lesions were better demonstrated without epinephrine (Table V).

CONCLUSIONS

Intraarterially administered epinephrine injection in the celiac axis or its branches, in a dose range of 4–10 μg., produced vasoconstriction by an alpha receptor response in the gastric, hepatic, and splenic arteries. The cystic artery demonstrated a

THE VEINS OF THE POSTERIOR CRANIAL FOSSA: AN ANGIOGRAPHIC STUDY UNDER PATHOLOGIC CONDITIONS

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THE introduction of new angiographic techniques (percutaneous retrograde brachial angiography, selective catheterization of the vertebral artery, subtraction method) has favored the routine investigation of the vertebral-basilar system. Vertebral angiography has therefore come into widespread use, not only to diagnose lesions of the posterior cranial fossa, but as a complement to the study of supratentorial vascular and neoplastic lesions as well. The knowledge of angiographic patterns in this region has thus improved greatly, but such progress has unfortunately been limited almost solely, for several years, to the angiographic phase of the examination. Variations in number, morphology, caliber and course of the veins of the posterior cranial fossa have hindered for a considerable time their complete and satisfactory description even under normal conditions, and thus have limited their correct evaluation in pathologic cases. In more recent years, the normal anatomy and angiographic appearance of the veins in the posterior cranial fossa have been described in detail by Huang and co-workers[3–5] and by Wolf and co-workers.[9] In this review of our series of tumors of the posterior cranial fossa, we shall therefore limit ourselves to our experience with the pathologic phlebogram only.

In previous papers[1,2] on the deep and superficial cerebral phlebogram, we have mentioned the frequent difficulties in recognizing on the angiographic films some single venous vessels because of their thinness or obscuration by other vascular (supratentorial superficial veins, veins of the extracranial occipital plexus) or osseous

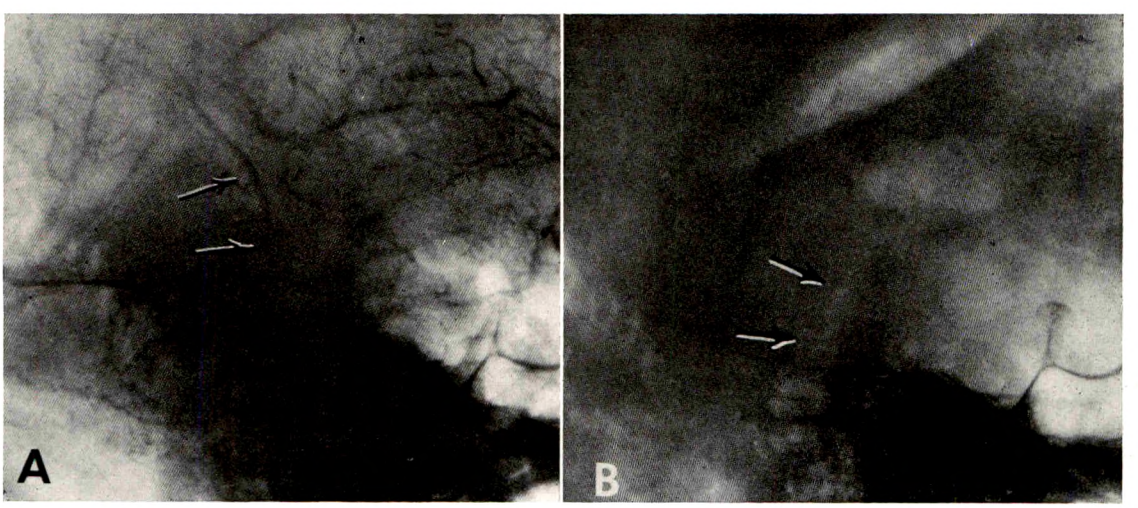


FIG. 1. (*A* and *B*) Right cerebellar glioma. The forward displacement of the precentral cerebellar vein (straight arrows), noticeable on the phlebogram (*A*), is matched by a corresponding anterior displacement of the aqueduct (straight arrows) on encephalography (*B*).

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Table IV

DIAGNOSTIC INFORMATION ADDED WITH EPINEPHRINE ANGIOGRAPHY

Individual tumor nodules in the liver	4
Metastases in the spleen	1
Extent of pancreatic cancer	3
Recurrent adrenal cancer with liver metastases	1
Intrahepatic bile duct carcinoma	1
Hepatic vein visualization in presence of liver metastases	1
	11

Table V

LESIONS OR CHANGES BETTER DEMONSTRATED WITHOUT EPINEPHRINE

Metastases in the liver	4
Hepatic vein opacification in presence of liver metastases	1
Hepatic artery-portal vein shunt in presence of liver metastases	1
Insulinoma of the pancreas	1
	7

similar response in 11 of 42 patients, and the gastroduodenal in 8 of 55. No definite vasoconstriction could be appreciated in the pancreaticoduodenal arteries, or in the pancreatic arteries arising from the splenic artery, except in 1 patient in whom vasoconstriction of the dorsal pancreatic artery occurred. This may be a reflection of the small quantity of epinephrine reaching these vessels, or fewer alpha receptors may be present in these duodenal and pancreatic arteries. In any event, this results in better angiographic visualization of the pancreatic vessels by diverting contrast material to the pancreatic vascular bed, with vasoconstriction and nonvisualization of confusing overlying hepatic, splenic, and gastric arteries.

Epinephrine diverts blood flow away from the vessels upon which it acts, to less reactive neovascular areas, to vessels receiving smaller doses of epinephrine enhancing duodenal and pancreatic visualization, and to vessels proximal to the end of the catheter. It may also, however, divert contrast material away from abnormal

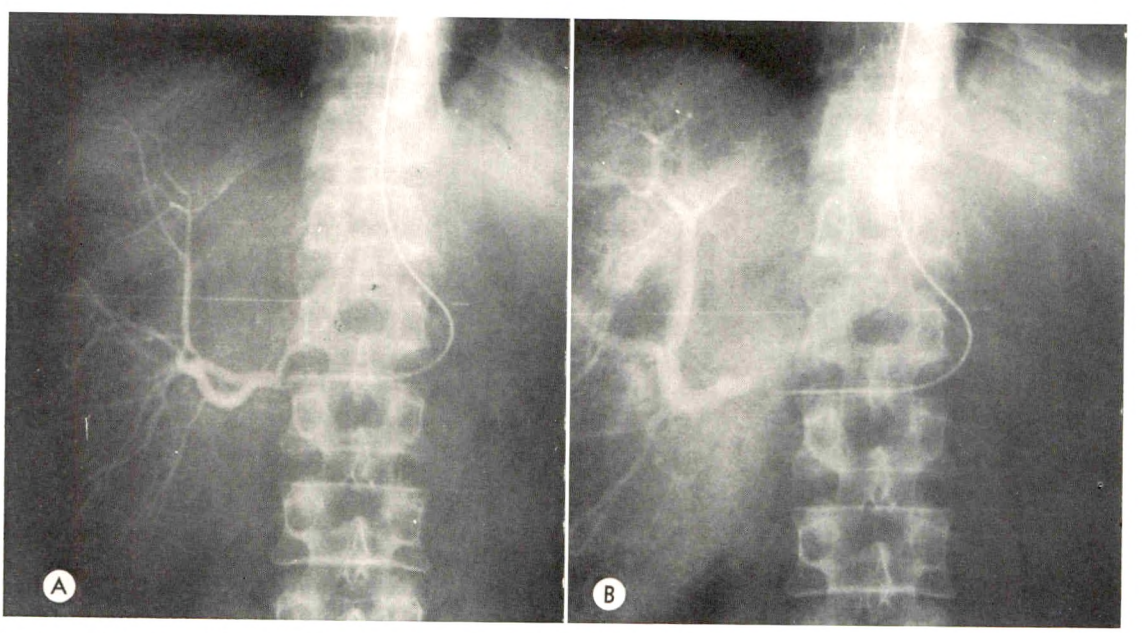


Fig. 9. A 54 year old male with squamous cell carcinoma of the lung, metastatic to the liver.
(*A*) Hepatic arteriogram without epinephrine demonstrates opacification of the hepatic artery, and (*B*) there is shunting of contrast material from the hepatic artery into the portal vein, with tumor staining noted throughout the right lobe of the liver. The comparable arteriogram with epinephrine produced diversion of contrast material away from the right lobe, so that the portal vein did not opacify.

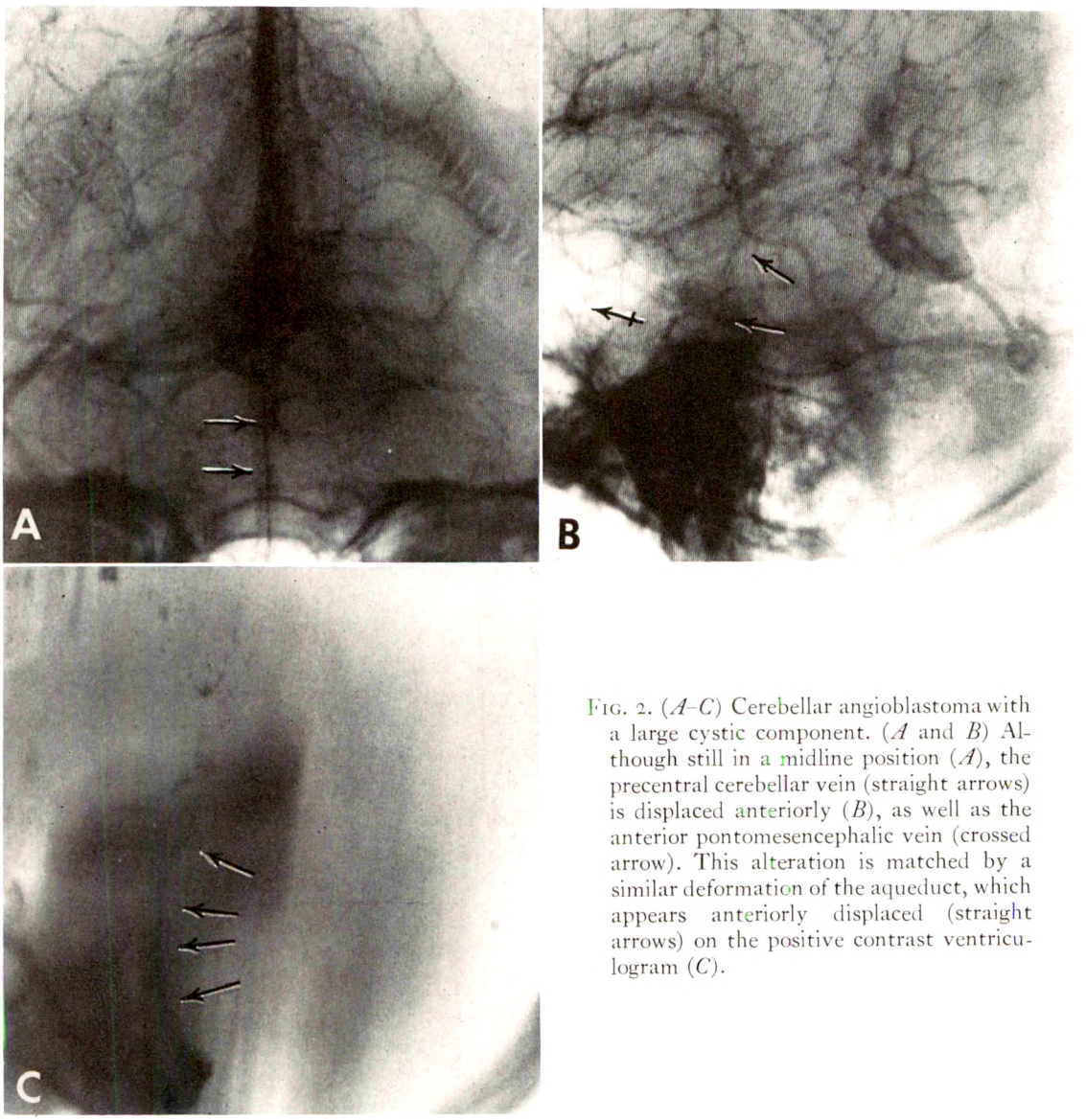


FIG. 2. (*A–C*) Cerebellar angioblastoma with a large cystic component. (*A* and *B*) Although still in a midline position (*A*), the precentral cerebellar vein (straight arrows) is displaced anteriorly (*B*), as well as the anterior pontomesencephalic vein (crossed arrow). This alteration is matched by a similar deformation of the aqueduct, which appears anteriorly displaced (straight arrows) on the positive contrast ventriculogram (*C*).

structures, which not even the subtraction method can always eliminate adequately. In addition, the almost identical course of some veins (basal, posterior mesencephalic, marginal and anterosuperior cerebellar veins) or supraimposition by their homologous vessel on the other side in the lateral view, may often preclude an exact identification of the single components of this venous system. However, daily experience in the observation of the venous pattern of the posterior cranial fossa may enable one to recognize some of the most important venous channels and to evaluate their possible alterations from pathologic processes, if one is familiar with their normal appearance and relationship with the nervous structures and if one correlates additional data available from the arterial phase of the examination and, possibly, from fractional gas encephalography.

In our experience, the most useful of all veins in the galenic system is the *precentral cerebellar vein*, whose caliber and characteristic course, following in the lateral views the course of the aqueduct and of the upper part of the fourth ventricle as shown by ventriculography, are essential elements

areas, resulting in loss of angiographic information. It would appear that if conventional angiography does not give a definitive diagnosis, information may be added by proceeding with epinephrine angiography.

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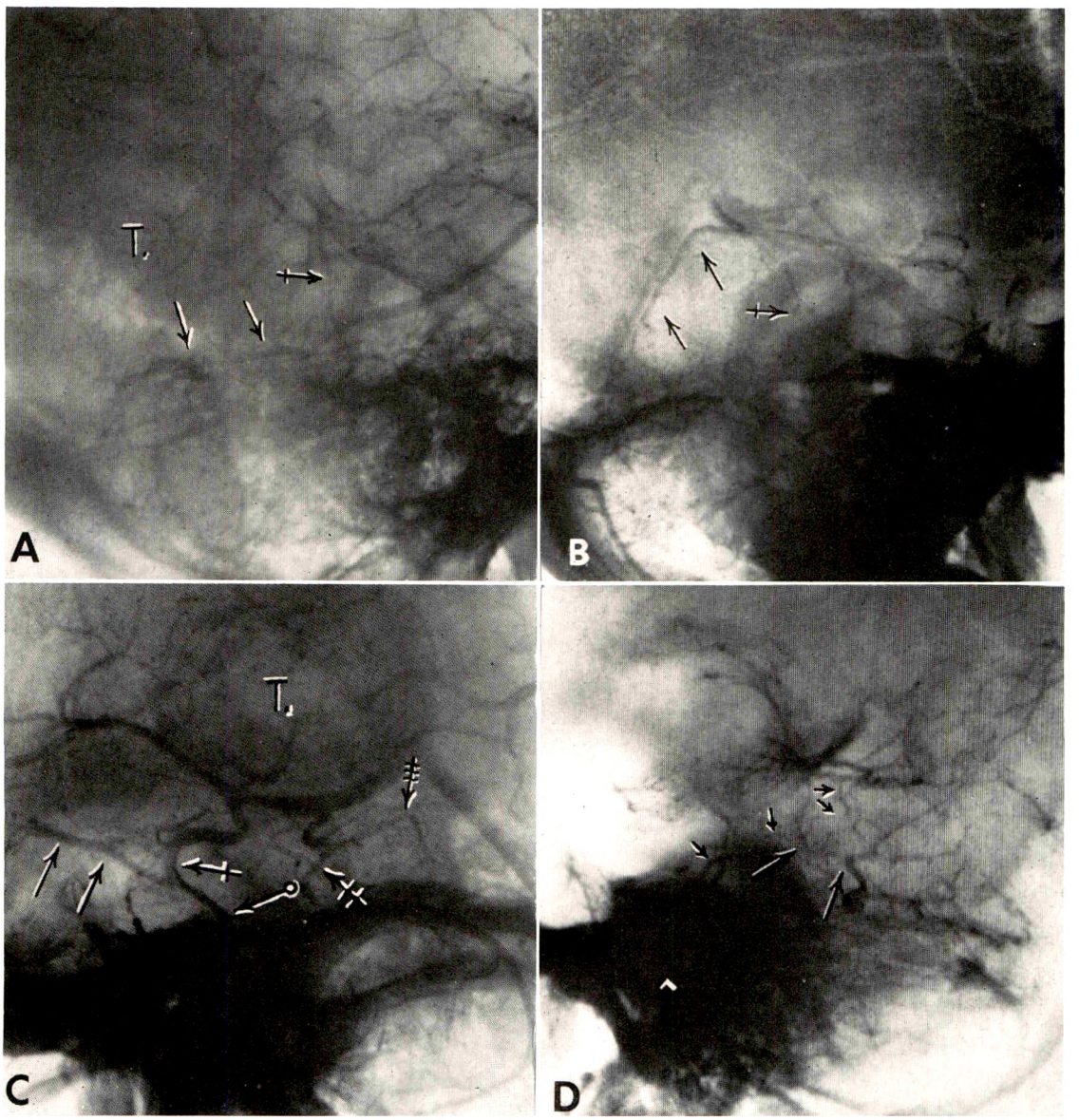


Fig. 3. (*A*) Meningioma of the falx-tentorium angle. Depression of the superior vermian vein (straight arrows), which appears to be separated more than usual from the straight sinus. The precentral cerebellar vein (crossed arrow), the anterior curvature of which is reduced at the level of the cullicula, is displaced anteriorly and verticalized. (*B*) Metastatic neoplasm of the vermis. The superior vermian vein is displaced posteriorly and superiorly (straight arrows), with loss of its characteristic festooned appearance. The precentral cerebellar vein (crossed arrow) is displaced anteriorly. (*C*) Tumor of the mid-posterior part of the corpus callosum. Marked depression of the internal cerebral vein; the precentral cerebellar (doubly crossed arrow) and the superior vermian (triple crossed arrow) veins, joining into a common terminal channel, appear to be "squashed" and displaced inferiorly. An anastomotic lateral mesencephalic vein (circle ended arrow) takes its origin at the junction of the anterior (straight arrows) and of the posterior (crossed arrow) parts of the basal vein. (*D*) Tumor of the fourth ventricle. Marked elevation of the initial part of the precentral cerebellar vein (short straight arrows). Note the marginal vein (long straight arrows), joining a tributary of the superior vermian vein with the petrosal vein.

INTESTINAL LYMPHANGIECTASIA*

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EXCESSIVE loss of serum proteins into the gastrointestinal tract (protein-losing enteropathy) is a common cause of hypoproteinemia and edema. Protein-losing enteropathy has been associated with over 70 diseases.[23] A cause being recognized with increasing frequency is the recently described syndrome of intestinal lymphangiectasia.[22]

Intestinal lymphangiectasia reflects a generalized disorder of the development of lymphatic channels. The hallmark lesion of the disorder, seen in all cases, is gross dilatation of the lymphatics of the lamina propria of the small bowel. These frequently distort and enlarge individual villi; however, there is no villous atrophy. Seen at laparotomy or autopsy, the affected small bowel is edematous and has dilated serosal and mesenteric lymphatics. The mesentery may also be edematous.

Clinically, intestinal lymphangiectasia is characterized by the usual early onset of massive edema, frequently asymmetric in distribution. Chylous effusions develop during the course of the disease in 45 per cent of patients. All patients have hypoalbuminemia and hypogammaglobulinemia. A proportionately less marked reduction of serum fibrinogen, transferrin and ceruloplasmin is also frequently present. The mechanism of the hypoproteinemia is excessive loss of serum protein into the intestine. This is demonstrable by radiolabelled macromolecules, such as Cr^{51} serum proteins or I^{131} polyvinylpyrrolidone (I^{131} PVP). Lymphocytopenia secondary to loss of lymphocytes into the bowel through the abnormal lymphatics is present in over 90 per cent of patients. The gastrointestinal protein loss may also occur because of ruptured submucosal lymphatics. In some cases serum protein levels and albumin metabolism improve when patients are placed on a low-fat diet in which middle chain triglycerides are substituted for long chain triglycerides.[5]

Gastrointestinal symptoms are usually mild but may be severe or absent. These include diarrhea (mild in 60 per cent of patients; severe in 20 per cent), vomiting (in 15 per cent of patients) and abdominal pain (in 15 per cent of patients). Mild steatorrhea is often present, severe steatorrhea is seen in 20 per cent of patients. Carbohydrate absorptive tests, including glucose, D-xylose and lactose tolerance tests are usually normal.

The pathogenesis of the lymphatic abnormalities in intestinal lymphangiectasia may be due to multiple causes. A congenital malformation is probably responsible in those patients presenting with this syndrome as infants. In older patients the syndrome may be acquired secondary to retroperitoneal fibrosis or pancreatitis.[22,23]

Patients with intestinal lymphangiectasia are often puzzling diagnostic cases of hypoproteinemia and edema, chylous effusions, or malabsorption. The purpose of this report is to review the roentgen findings in 20 patients with intestinal lymphangiectasia and indicate the value of roentgen studies in this disorder.

MATERIAL AND METHOD

Twenty patients with intestinal lymphangiectasia have been studied at the National Institutes of Health since 1957. All patients have had hypoproteinemia (both hypoalbuminemia and hypogammaglobulinemia) with serum survival of intra-

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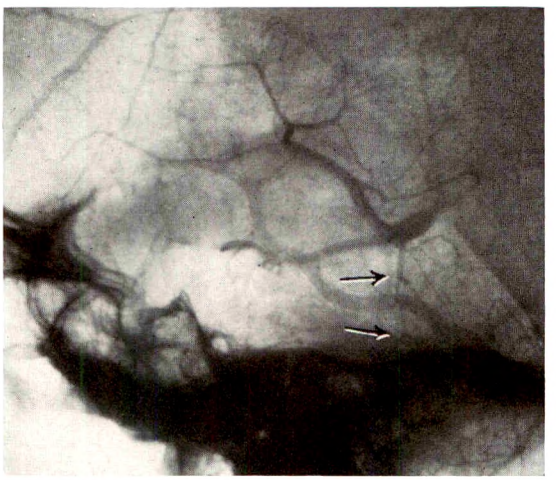


Fig. 4. Unverified tumor of the brainstem. The precentral cerebellar vein (straight arrows) is displaced posteriorly, with inversion of its normally anterior curvature.

for an easy recognition. The diagnostic importance of this vein is also given by its midline position to the nervous structures of the posterior fossa. Therefore, space-occupying lesions in this area will affect the shape and course of this vein, similar to those undergone by the aqueduct and by the upper part of the fourth ventricle. The most striking pattern can be observed in the lateral angiograms, in which supra-imposing veins can be easily identified as superficial.

In our series, the precentral cerebellar vein was found to be displaced anteriorly by tumors of the posterior part of the vermis or of the cerebellar hemisphere (Fig. 1, *A* and *B*; and 2, *A–C*); or to be verticalized, with reduction of its normal, anteriorly convex curve, by neoplasms of the upper part of the vermis or from tentorial tumor (Fig. 3, *A* and *B*). Tumors located more anteriorly (brainstem or cavity of the fourth ventricle) produce a lifting and posterior displacement of the vein (Fig. 3*D*; and 4), while the "genu" of the same vein is flattened downwards by supratentorial tumors, such as meningiomas of the falx-tentorium angle and gliomas of the posterior part of the corpus callosum (Fig. 3*C*). A posterior displacement of the precentral cerebellar vein is also produced by tumors of the third ventricle or by stenosis of the aqueduct (Fig. 5, *A–D*).

In the anteroposterior angiogram, the identification of the vein and the evaluation of its pathologic behaviour are made more difficult by the supraimposition of the inferior vermian veins, often running in the midline, and of the vein of Galen, or by a more lateral course of the vein itself as one of its normal variations.[3]

Only in exceptional cases were we able to observe a pathologic shift of the precentral cerebellar vein from subtentorial tumors (Fig. 6, *A* and *B*). In such cases, an exact diagnosis had already been made on the basis of other findings and such a feature could only assume a secondary role.

The pathologic alterations undergone by the *posterior mesencephalic vein* are, in our opinion and experience, more difficult to interpret, and thus of a limited diagnostic value. In fact, the extreme thinness of its caliber makes its adequate injection a rare event; furthermore, even under normal conditions, these veins are often asymmetric, which makes it difficult to judge pathologic shifts or deformities.

In supra- or subtentorial lateral expanding lesions, the homolateral posterior mesencephalic vein may be depressed or elevated (Fig. 5, *A–D*; and 8, *A* and *B*). In the anteroposterior view, such shifts are matched by an asymmetry in the 2 branches of the "inverted V" formed by the posterior mesencephalic veins. In none of our brainstem tumors, however, were we able to find a clear-cut, pathognomonic alteration of the posterior mesencephalic veins, such as it might be expected to be produced by tumors in such a location. It is possible that pressure from the tumor and from edema of the neighboring nervous structures may be such as to cause compression and inadequate angiographic filling of these veins.

The strict relationship of the *superior vermian vein*, the third element in the superior system of drainage (galenic draining group), with the dorsal surface of the

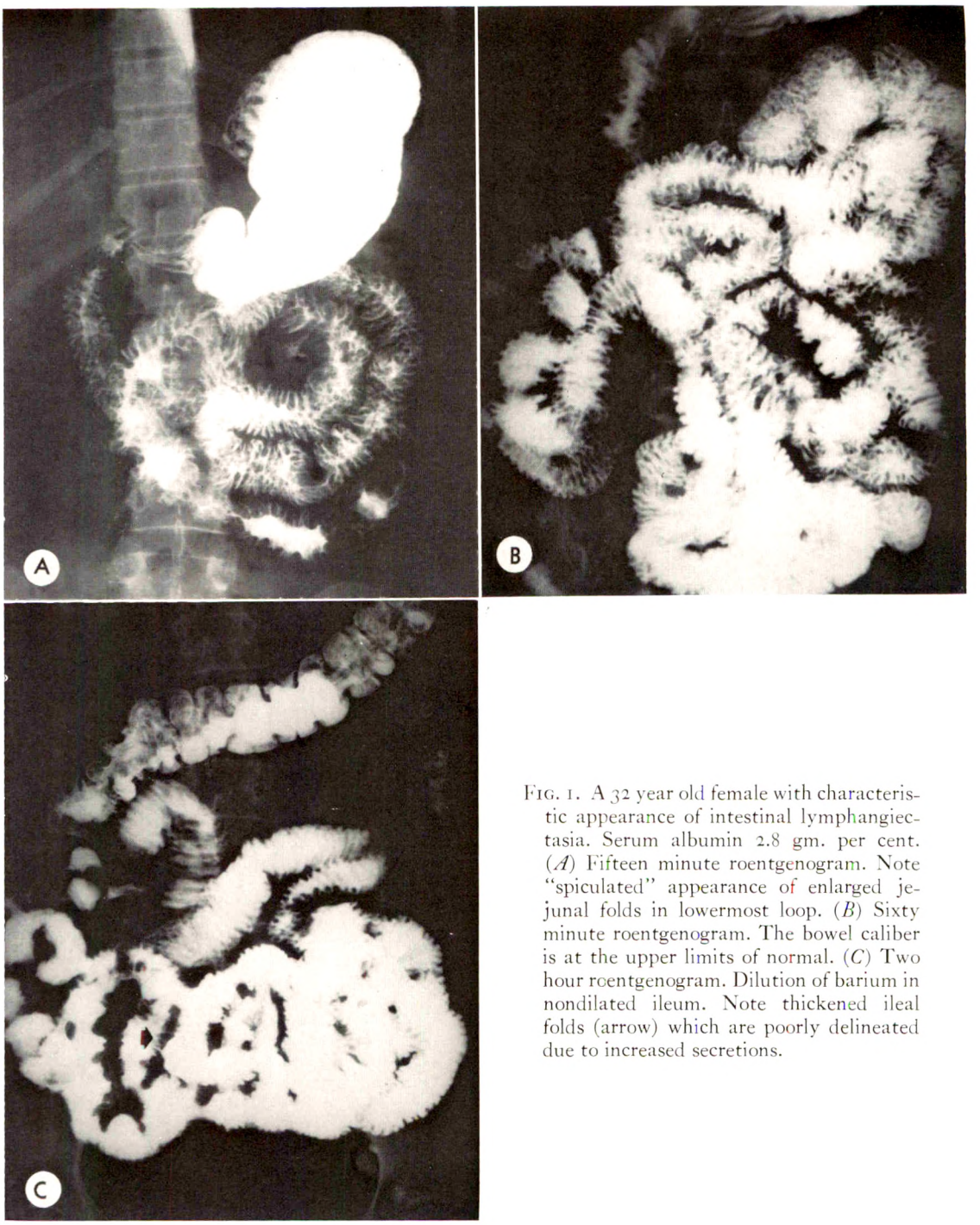


FIG. 1. A 32 year old female with characteristic appearance of intestinal lymphangiectasia. Serum albumin 2.8 gm. per cent. (*A*) Fifteen minute roentgenogram. Note "spiculated" appearance of enlarged jejunal folds in lowermost loop. (*B*) Sixty minute roentgenogram. The bowel caliber is at the upper limits of normal. (*C*) Two hour roentgenogram. Dilution of barium in nondilated ileum. Note thickened ileal folds (arrow) which are poorly delineated due to increased secretions.

venously administered I^{125} albumin from 1/10 to 1/2 normal values. In all cases excessive gastrointestinal protein loss has been demonstrated with Cr^{51} albumin or I^{131} PVP. The diagnosis in all has been confirmed by demonstration of dilated submucosal lymphatics of the small bowel in histologic specimens obtained at autopsy, laparotomy or by peroral jejunal biopsy. All 20 patients had chest roentgenograms,

cerebellar vermis (culmen and declive) gives it the characteristic appearance of a festooned arc with an upper convexity, allowing an easy recognition on the lateral films of the vertebral phlebogram and thus giving this vein a great value in the diagnosis and localization of expanding lesions. For instance, the superior vermian vein was clearly depressed and, more significantly, separated from the straight sinus in

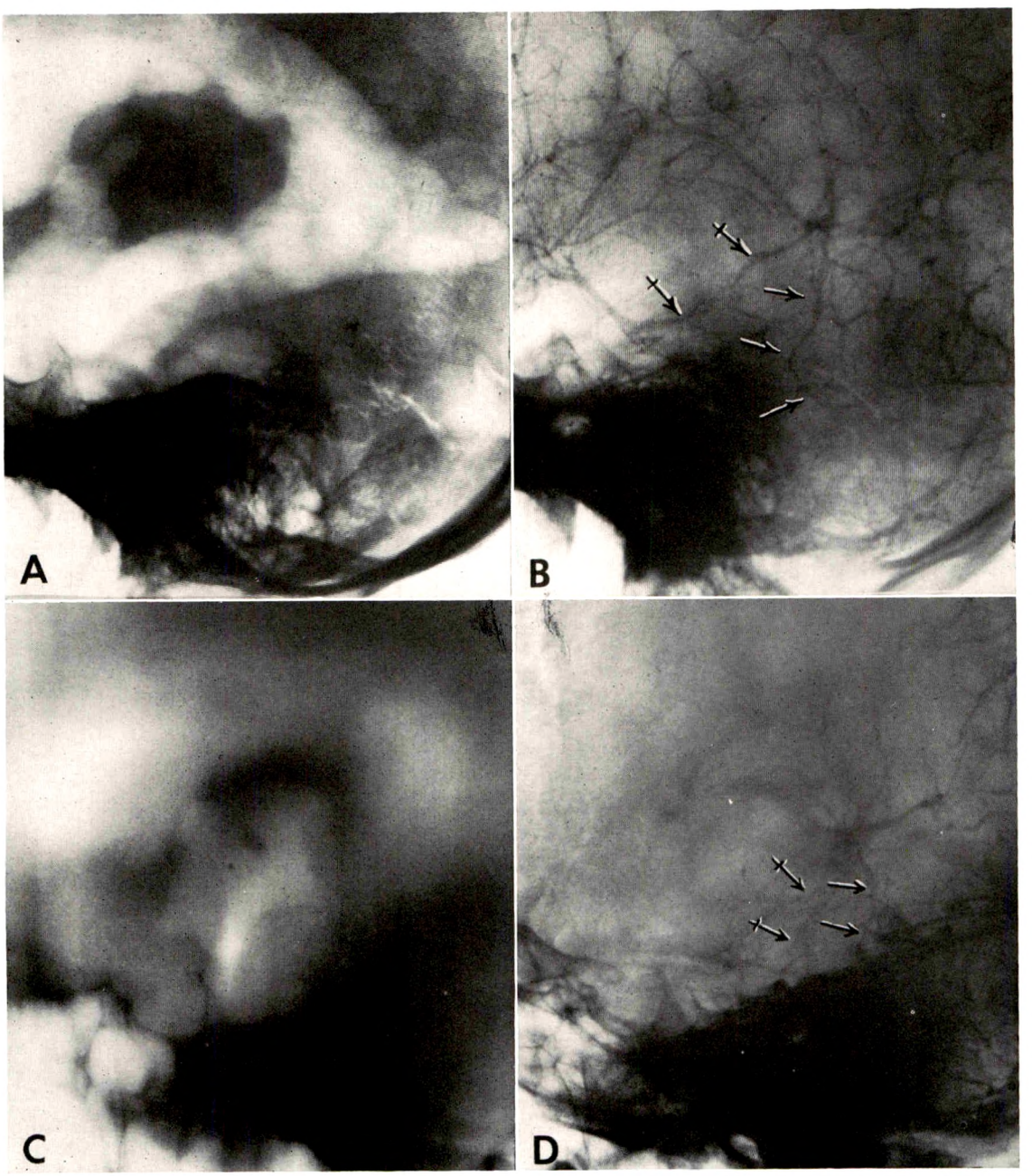


Fig. 5. (*A–D*) Two cases of tumor of the posterior part of the third ventricle. Marked elevation of the internal cerebral vein; posterior displacement of the precentral cerebellar vein (straight arrows), which follows in its terminal part a course which is more straight than usual. The posterior mesencephalic veins (crossed arrows) are displaced posteriorly and inferiorly.

upper gastrointestinal and small bowel examinations; 14 patients had barium enema examinations and 9 had pedal lymphangiography. All studies mentioned were available for review and form the basis of this report.

Our small intestinal examination employs a large volume of flocculation-resistant barium (*e.g.*, for adults 8–10 ounces of micropaque mixed with an equal volume of water). Our standards for normal are in agreement with other authors:[10,19] (1) the maximum width of the jejunum should be less than 3 cm., and of the ileum less than 2.5 cm. These figures are obtained by averaging measurements of the 2 or 3 best distended loops on a single roentgenogram chosen for maximal filling of the region; (2) the maximal jejunal and ileal fold (*i.e.*, valvulae conniventes) thickness should be less than 2.0 mm. This figure is an average of 5 or 6 measurements of the largest appearing folds chosen from many loops on the roentgenograms selected for the bowel width measurements; (3) the presence of increased intestinal secretions is established only if there is dilution of the barium column distally.[4] Often this diluted column has a fine granular appearance and the intestinal folds are poorly delineated. Fragmentation of barium is not used as an indication of increased secretions.

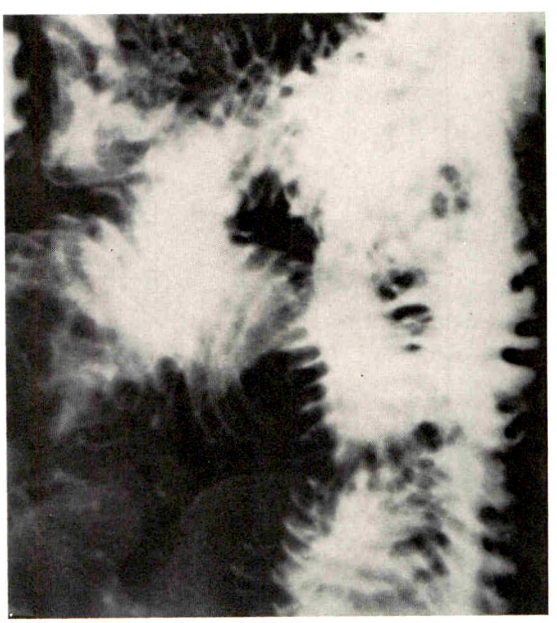


Fig. 2. Detail of proximal jejunum. The intestinal folds are enlarged, straightened, closely spaced and parallel to one another. (Same patient as in Fig. 11.)

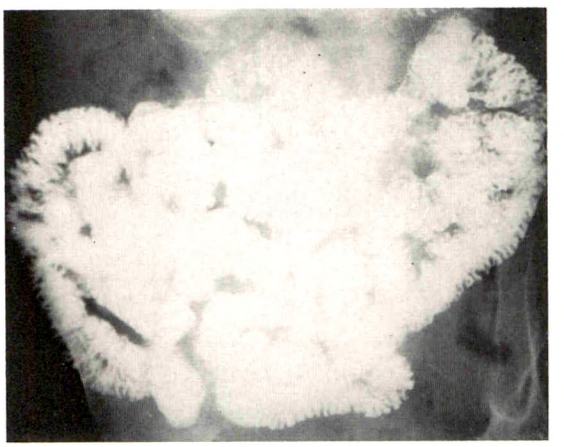


Fig. 3. A 34 year old male. Serum albumin 1.0 gm. per cent. The pattern of the mildly enlarged, redundant ileal folds is reminiscent of normal jejunum.

ANALYSIS

Five patients had normal small intestinal examinations.

In the remaining 15 patients with abnormal small intestinal examinations a characteristic appearance was noted to be comprised of 3 features: (1) enlargement of the intestinal folds of both jejunum and ileum; (2) dilution of the barium column, distally; and (3) absent or minimal dilatation of the bowel (Fig. 1, *A–C*). All 3 features were present at initial evaluation on 9 of the 15 patients with abnormal small intestinal examinations. Two additional patients did not demonstrate dilution on initial evaluation, but manifested all 3 features on a follow-up examination. The last 4 cases with abnormal small bowel examinations did not have the characteristic picture of intestinal lymphangiectasia. All 4 failed to demonstrate dilution; 2 of these cases in addition demonstrated enlargement of folds in the jejunum only.

Enlargement of both jejunal and ileal

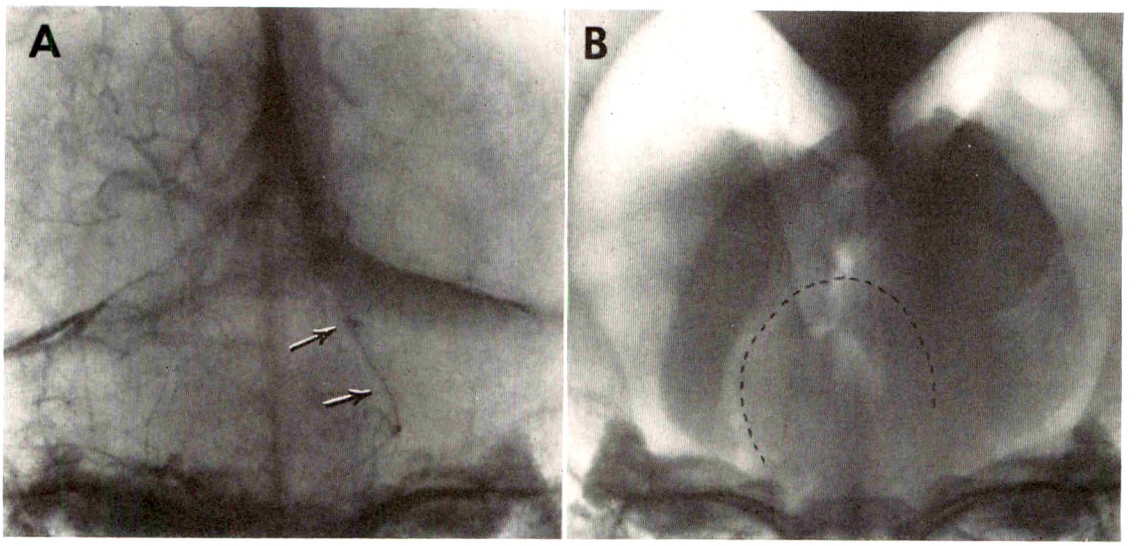


Fig. 6. Tumor of the fourth ventricle. (*A*) Lateral displacement of the precentral cerebellar vein (straight arrows). (*B*) Negative contrast ventriculogram.

some meningiomas of the falx-tentorium angle, thus allowing a diagnosis of extracerebellar growth (Fig. 3*A*). This same vein appears to be displaced superiorly and posteriorly by tumors located in, or originating from the upper part of the vermis (Fig. 3*B*) or from the cerebellar hemispheres in a paramedian position.

Within the anterior (petrosal) draining group we have analyzed the pathologic behavior of 3 vessels: the *lateral anastomotic mesencephalic vein*, the anterior pontomesencephalic vein and the petrosal vein.

The first of these veins has been described in detail in its normal anatomic and angiographic features by Wolf and co-workers.[9] Its presence and identification has a two-fold importance: from an anatomic point of view, because it marks the posterior margin of the cerebral peduncle; from a functional point of view, because it serves as an anastomosis between the supra- and subtentorial nervous structures. However, we were unable to observe, even reviewing our whole series in retrospect, any useful diagnostic clue afforded by this vessel, which even under normal conditions presents great anatomic variability. Possible positive findings could be, at the most, an increase of its caliber in cases where the vein represents the main channel of direct drainage from the neoplasm, or it takes over such function as a collateral channel (Fig. 9, *A* and *B*; and 10, *A* and *B*).

The *anterior pontomesencephalic vein* can undergo alterations from tumors of the brainstem and of the clivus. In the former case, the vein is displaced anteriorly and upwards in a wide, anteriorly-convex sweep (Fig. 11*A*); in the latter instance, on the opposite, it is pushed backwards to describe an arc with a posterior convexity, which presumably follows the deformation of the ventral surface of the pons (Fig. 11*B*). In aqueductal stenosis, the depression of the floor of the third ventricle and the bulging arc of the upper part of the pons cause a depression and a shortening of the interpeduncular segment of the vein, as well as a forward displacement of its pontine segment. This finding, together with the finding of a backwards displacement of the precentral cerebellar vein, produces an increase of the anteroposterior width of the isthmus, calculated as the shortest distance between the lowest point of the interpeduncular segment of a "typical" anterior pontomesencephalic vein and the culliculo-

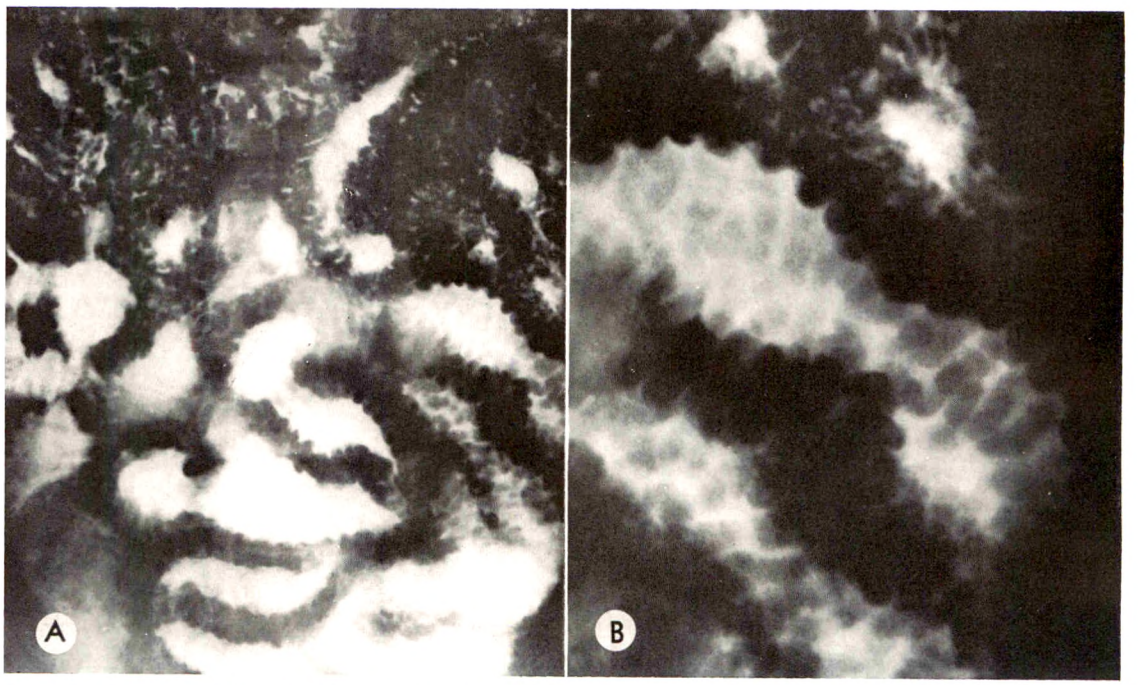


FIG. 4. A 16 year old female with severe diarrhea. Serum albumin 1.4 gm. per cent. (*A*) Marked dilution of the barium column due to increased intestinal secretions. The fragmentation of the barium is the greatest of entire series. (*B*) The left-sided jejunal loops have a "cobblestone" pattern of fold enlargement, better demonstrated on magnified view of this region.

folds was noted in 13 cases and enlargement of only jejunal folds in 2 cases. The enlargement of folds in a single loop was most often uniform, and the pattern of fold enlargement orderly and symmetric. Various patterns were noted, these were often inconstant in an individual case, were dependent on the degree of barium distention, and were difficult to classify.[19] Some patterns seen are noteworthy because of resemblance to those associated with other diseases. The enlarged folds may be straightened, closely set, and parallel to one another, analogous to scleroderma[1] (Fig. 2). If closely set folds are seen in a mildly contracted loop, a "spiculated" appearance may ensue (Fig. 1*A*). Redundancy of enlarged folds in the ileum may give an appearance resembling the normal "feathery" pattern of the jejunum, analogous to occasional cases of amyloidosis[4] (Fig. 3). One case had a diffusely nodular, "cobblestone" pattern of fold enlargement (Fig. 4*B*). One patient had asymmetric enlargement and irregular orientation of jejunal folds producing a disorderly pattern of fold enlargement sometimes seen with Whipple's disease[4] (Fig. 5*A*); in the ileum more orderly, symmetric fold enlargement was present (Fig. 5*B*). Two patients had striking irregular serration to the margin of ileal loops due to deranged, incomplete peristalsis. In neither of these cases was the marginal serration constant during the examination. In 1 of these patients (Fig. 6, *A* and *B*) laparotomy disclosed an enormously thickened mesentery. In 3 cases, rather than the diffusely nodular mucosal pattern described above, a few individual nodules were present in the jejunum (Fig. 7). The individual nodules were all less than 7 mm. in diameter.

Dilution of the barium column was eventually seen in 11 of the 13 cases with diffuse enlargement of intestinal folds. Dilution was not observed in the 2 cases who had enlarged jejunal folds only. At times dilution may be extreme (Fig. 4*A*). Barium

central point of the precentral cerebellar vein, the normal value of which should range between 18 and 23 millimeters.[6]

The group of the *petrosal vein* is of a greater importance both from a surgical (*e.g.*, possibility of hemorrhage during surgical exposure of the fifth cranial nerve) and from a diagnostic point of view, especially of tumors of the cerebellopontine angle. In such cases, since the findings obtainable in the arterial phase of vertebral angiography are not as specific and decisive as those given by fractional gas encephalography, even for the purpose of a surgical decision, a better utilization of the phlebogram as allowed by recent research,[5] in conjunction with the findings of the arterial phase, enhances considerably diagnostic precision,

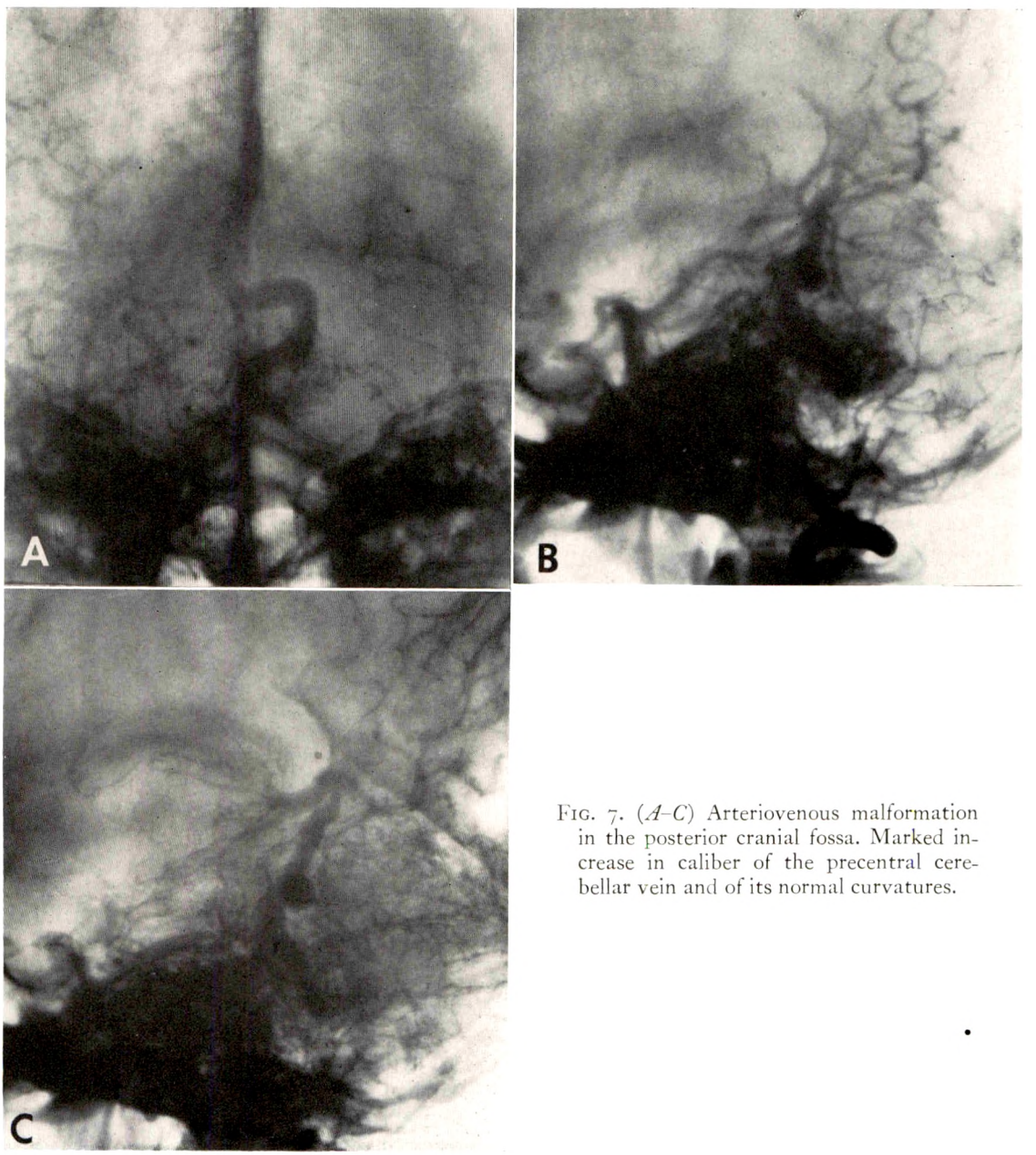


Fig. 7. (*A–C*) Arteriovenous malformation in the posterior cranial fossa. Marked increase in caliber of the precentral cerebellar vein and of its normal curvatures.

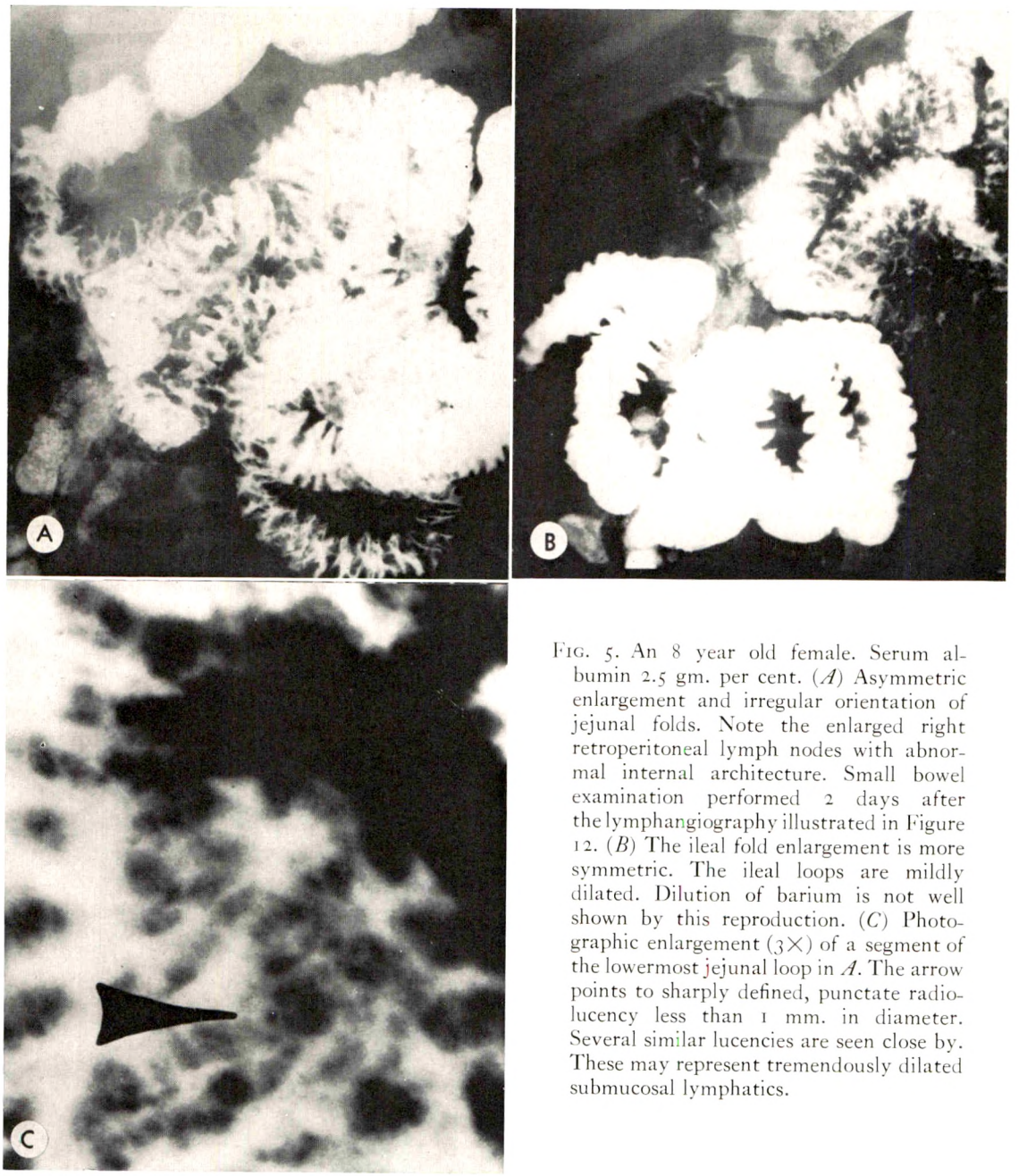


Fig. 5. An 8 year old female. Serum albumin 2.5 gm. per cent. (*A*) Asymmetric enlargement and irregular orientation of jejunal folds. Note the enlarged right retroperitoneal lymph nodes with abnormal internal architecture. Small bowel examination performed 2 days after the lymphangiography illustrated in Figure 12. (*B*) The ileal fold enlargement is more symmetric. The ileal loops are mildly dilated. Dilution of barium is not well shown by this reproduction. (*C*) Photographic enlargement (3×) of a segment of the lowermost jejunal loop in *A*. The arrow points to sharply defined, punctate radiolucency less than 1 mm. in diameter. Several similar lucencies are seen close by. These may represent tremendously dilated submucosal lymphatics.

fragmentation was not a prominent feature of any case.

Dilatation of the jejunum alone was noted in 3 cases and dilatation of the ileum alone in 1 case. Dilatation of both was seen in only 1 patient (Fig. 8, *A* and *B*). In no case was dilatation extreme (as may be seen with nontropical sprue).

Four cases showed central positioning of small bowel loops in the abdomen indicating ascites; 5 patients showed abnormal separation of bowel loops indicative of mesenteric thickening and/or thickening of the bowel wall. All patients had a normal transit time of barium to the cecum ranging from 1 to 6 hours.

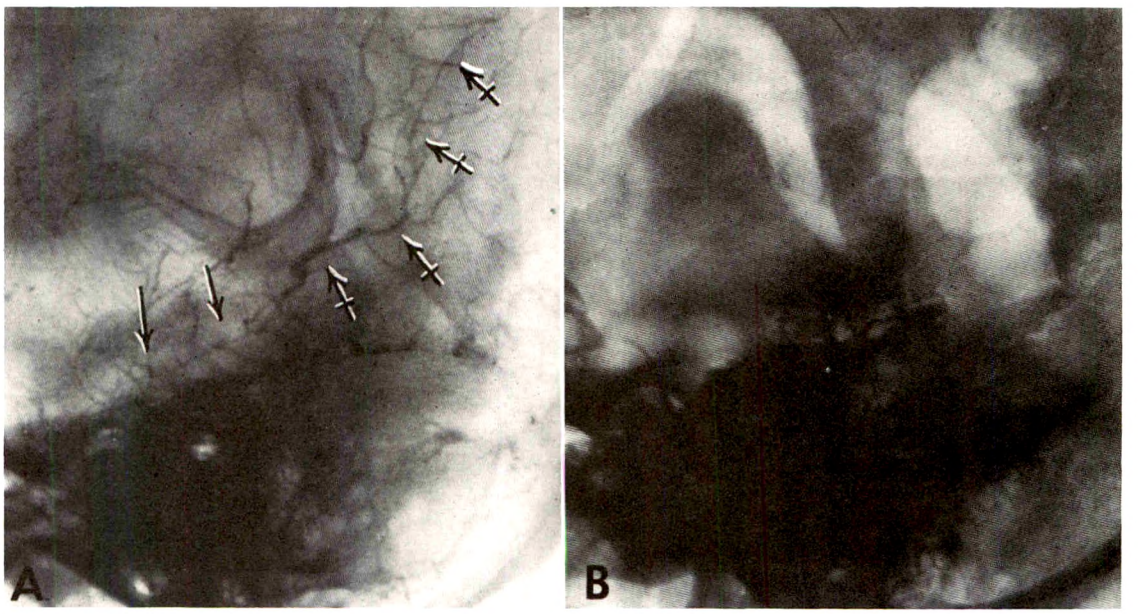


FIG. 8. (*A* and *B*) Cystic occipital glioma. The internal occipital vein (crossed arrows) marks the tumor with a posterior concavity. Depression of the thin posterior mesencephalic vein (straight arrows) is produced by herniation of the temporal lobe.

thus justifying a wider resorting to vertebral angiography.

Tumors of the cerebellopontine angle, in fact, produce characteristic stretching, shifting and compression of the petrosal vein and of its tributaries (Fig. 12, *A* and *B*). Lack of visualization of the petrosal vein together with a good filling of the posteroinferior cerebellar artery holds, instead, the value of an accessory sign, as it can be found also in expanding lesions of the cerebellum and pons.[8]

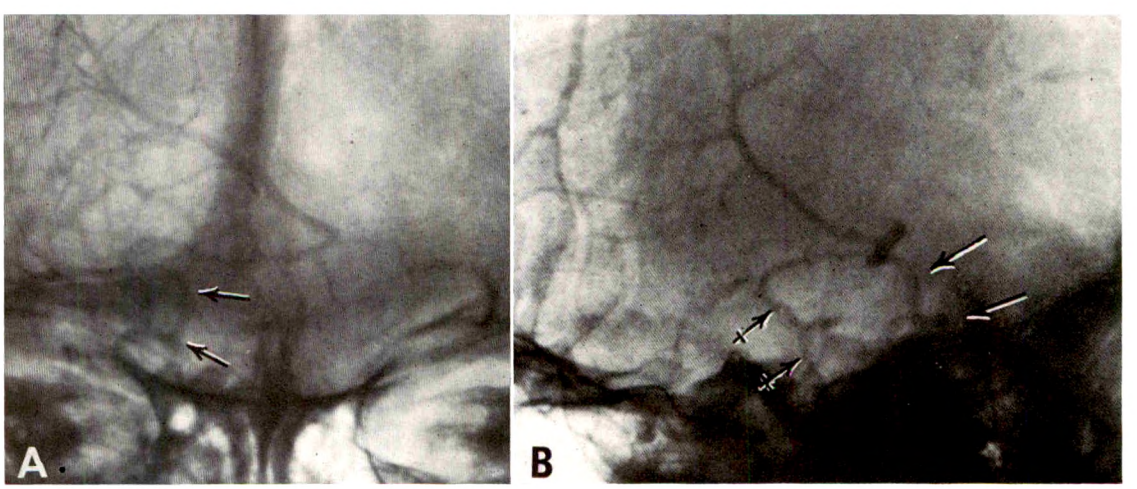


FIG. 9. (*A* and *B*) Glioma of the posterior third of the corpus callosum. The neoplasm has produced obstruction of the great vein of Galen; the internal cerebral vein drains into a large anastomotic lateral mesencephalic vein (straight arrows), the tortuous course of which can be best seen on the lateral angiogram as a direct continuation of the basal vein. The anastomosis of the anterior pontomesencephalic vein (crossed arrows) with the basal vein is also well evident.

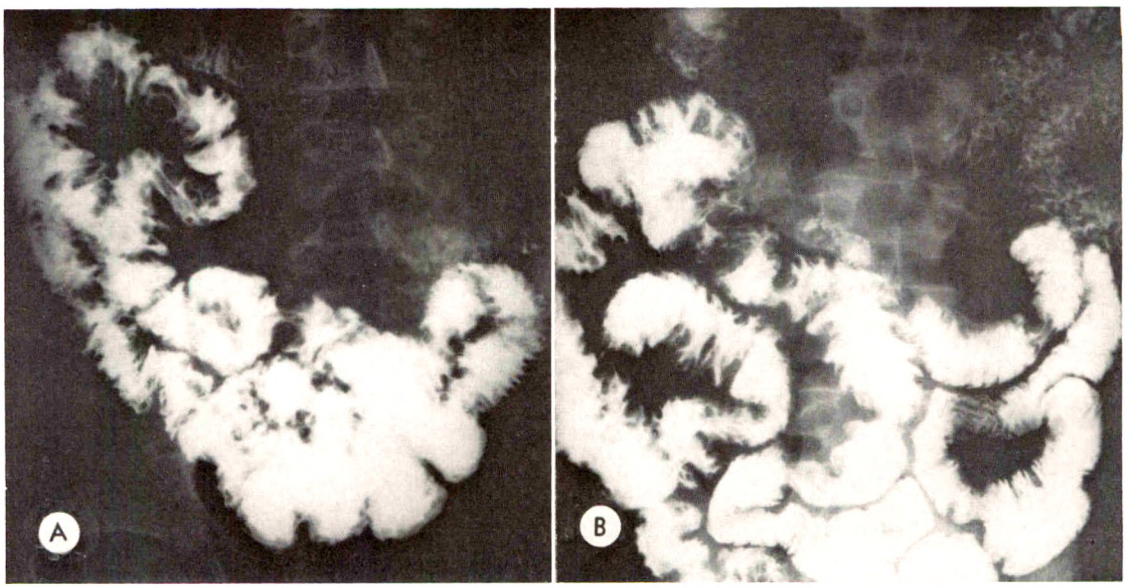


FIG. 6. (*A* and *B*) An 18 year old male. Serum albumin 3.3 gm. per cent. Two roentgenograms from the same examination. Inconstant, irregular serration to margin of ileal loops.

All barium enema examinations performed were normal. Upper gastrointestinal examination disclosed normal esophagus, stomach and duodenal bulb in all instances. In some instances, enlargement of duodenal folds was felt to be present, but values for this determination are not well established. Similarly, in a few instances, the duodenal sweep may have been mildly dilated. Chest roentgenograms often revealed the presence of pleural effusion which may be massive and unilateral.

Six patients, all with initially positive studies, have had follow-up small bowel examinations for various reasons. In 2 cases, large jejunal folds seen initially were increased in size on a subsequent examination. Between examinations, both patients had had intervening ileal resections. In the remaining 4 patients no change was noted in fold enlargement. Dilution of the barium column in the ileum proved the least constant roentgenographic finding on follow-up studies. In 2 patients, as noted above, dilution was not present on the initial study. Two other patients showed varying degrees of dilution on multiple examinations. Although a few patients in this series have had a good clinical response to a low fat diet, none has been restudied roentgenographically.[15]

The level of the serum albumin at the time of the small bowel examination did not correlate well with either the degree of fold enlargement or the presence of increased secretions. The serum albumin levels of the 5 patients with normal examinations were 1.2, 1.7, 1.9, 2.4 and 2.8 gm. per cent. Enlarged folds were noted with albumin levels ranging from 0.8 to



FIG. 7. An 8 year old female. Serum albumin 2.8 gm. per cent. Detail of jejunum. Several nodular defects and greatly enlarged, parallel folds are present.

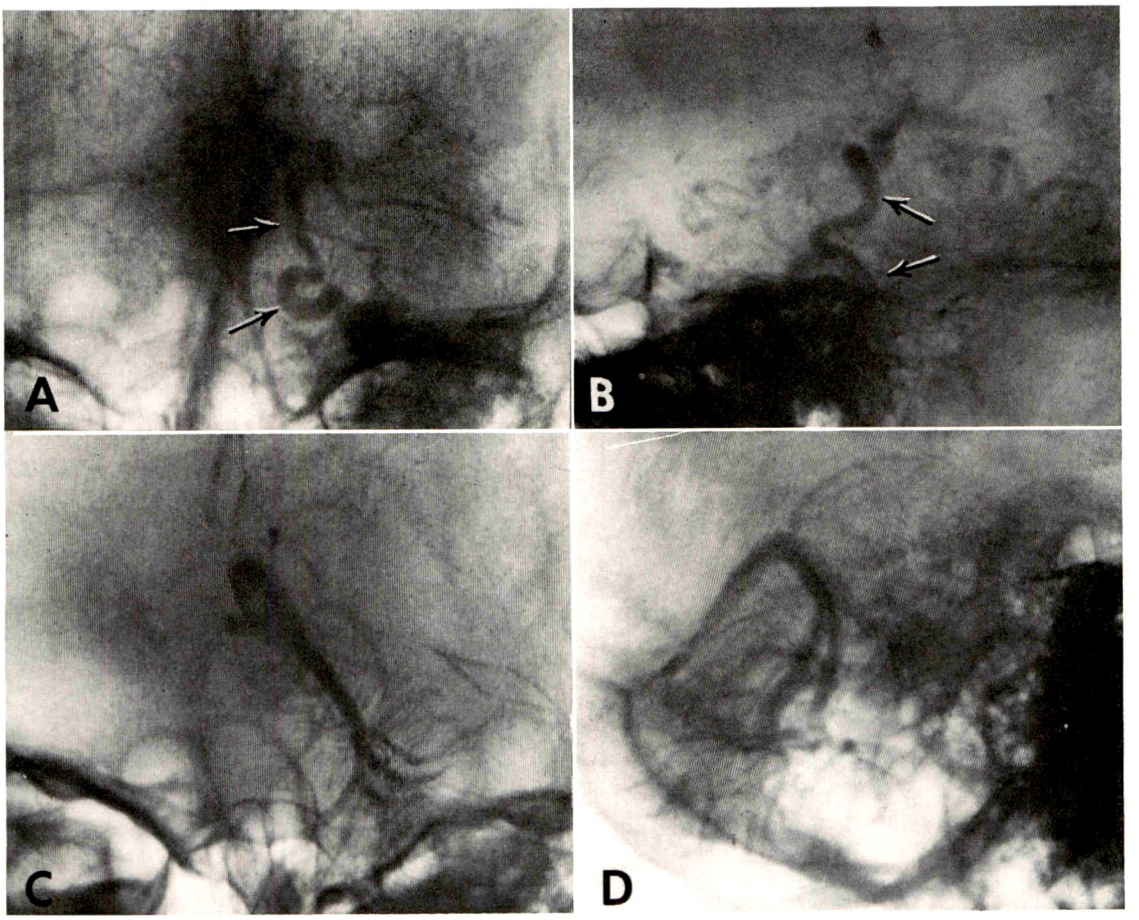


Fig. 10. (*A* and *B*) Supratentorial arteriovenous malformation. The lesion drains partly into the enlarged anastomotic lateral mesencephalic vein (straight arrows), the course of which resembles that of the great anterior cerebellar vein. (*C* and *D*) Cerebellar angioblastoma. A large inferior cerebellar vein drains the lesion into the torcular of Herophylus.

The superficial cerebellar veins of the posterior group have a minor diagnostic importance because of the great variability of their normal course on the surface of the cerebellar lobes; their shifts and deformations are therefore more difficult to evaluate. Here again, they may become larger and more visible at angiography when forming the main channels of direct or collateral venous drainage from the neoplasm (Fig. 10, *C* and *D*).

As far as post-traumatic space-occupying lesions (hematomas) in the posterior fossa are concerned, the angiographic findings are similar to those found with their supratentorial equivalents. However, their detection can present some difficulties when they are of limited size and may make it necessary to resort to oblique views, just as is done for supratentorial hematomas.

CONCLUSION

We believe that our study, limited though it may be to pathologic cases only and not aimed to the detailed description of the normal venous circulation in the posterior cranial fossa, can support our earlier statement;[1,2] *i.e.*, that no single detail of the angiographic examination should be considered decisive for diagnosis and that all clinical, and roentgenologic data already available for the single case

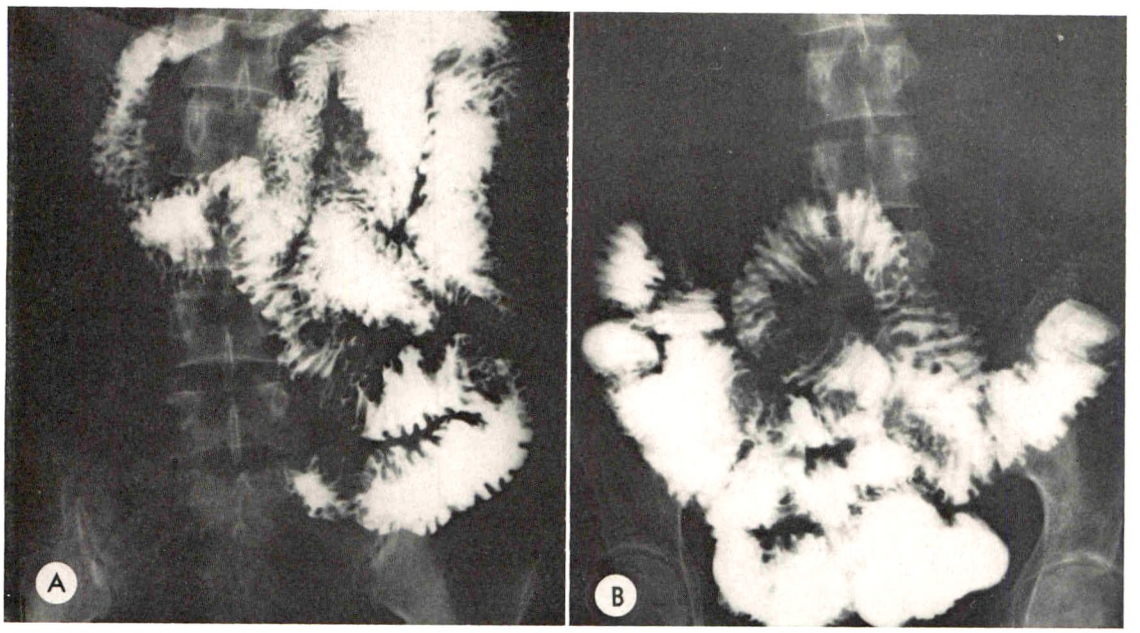


Fig. 8. (*A* and *B*) An 18 year old female. Serum albumin 2.0 gm. per cent. Mild dilatation of both jejunum and ileum, the greatest dilatation observed in this series. There are large folds throughout; no dilution is demonstrated on this examination.

3.3 gm. per cent. The patients showing the greatest amount of barium dilution were those with severe diarrhea.

One patient developed an internal hernia with recurrent, intermittent, intestinal obstruction due to dense adhesions forming after a small bowel resection. The widespread and proliferative nature of the adhesions was probably related to proteinaceous ascitic fluid resulting from the primary disease.

REPORT OF A CASE

This 31 year old white male had the onset of his disease at the age of 18 years with the development of first dependent, and then generalized, edema, always worse on the left side. Hypoproteinemia was discovered and was shown to be due to gastrointestinal protein loss. Over the years, the patient's serum albumin values have ranged from 1.6 to 2.0 gm. per cent and his total serum protein values from 3.5 to 4.5 gm. per cent. Treatment with cortisone, diuretics and various dietary regimens has produced no significant response of either the hypoproteinemia or peripheral edema. In 1960, at the age of 27 years, he developed worsening of peripheral edema. Multiple peroral jejunal biopsies were interpreted as showing areas of normal mucosa interspersed with areas of dilated submucosal lymphatics. Exploratory laparotomy was performed with the hope of finding a localized, resectable lesion responsible for the gastrointestinal protein loss. Preoperative albumin transfusion raised the serum albumin to 3.7 gm. per cent with mobilization of the patient's peripheral edema. Operation disclosed a red, thickened 3 foot length of small bowel just distal to the ligament of Treitz, and a similar less striking area of terminal ileum. Greatly enlarged lymphatics in the bowel wall and in the mesentery were noted. Resection of the proximal jejunum and appendectomy were performed. Pathologic examination showed numerous dilated submucosal and mesenteric lymphatics. Prominent lipofucsin pigmentation of the external muscularis was present. Postoperatively there was no change in the hypoproteinemia or gastrointestinal protein loss.

For 3 years postoperatively the patient had recurrent episodes of massive peripheral edema best treated by bed rest. In addition he experienced almost weekly episodes of severe abdominal pain associated with nausea, vomiting and the appearance of a slightly tender abdominal mass. These episodes regressed spontaneously after a few hours with disappearance of the

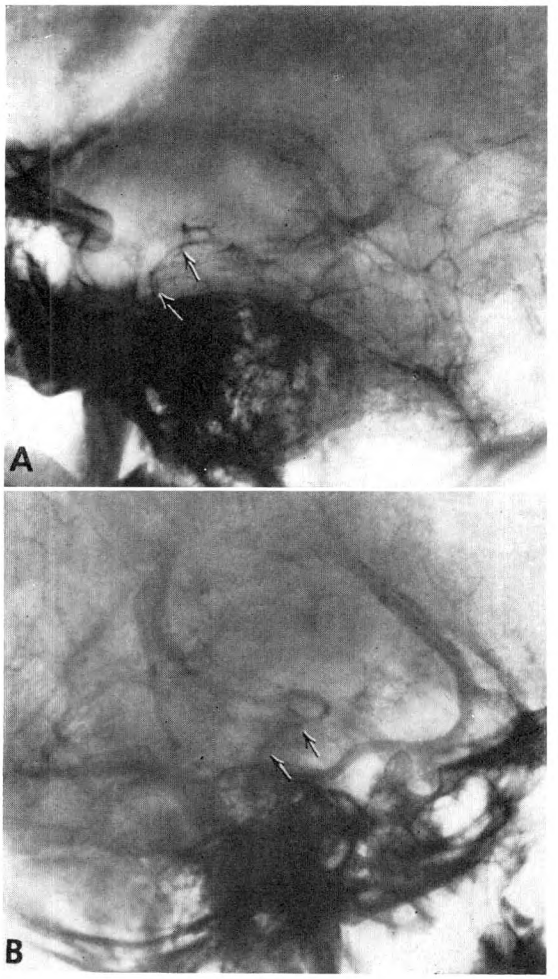


FIG. 11. (*A*) Unverified tumor of the brainstem. Anterior displacement of the anterior pontomesencephalic vein (straight arrows), which presents also a marked anterior concavity. (*B*) Tumor of the clivus. Marked posterior displacement of the anterior pontomesencephalic vein, the normal anterior convexity of which appears to be inverted (straight arrows).

should be used to reach a final preoperative diagnosis. We have selected from our series a few cases which we believe demonstrate our opinion. It seems to us that the venous circulation of the brain in general and of the posterior cranial fossa in particular, being subjects of speculation from the point of view of function, morphology, physiopathology and angiographic variations are conducive to still further investigation.

SUMMARY

Experience with the venous phase of vertebral angiography is reported.

The importance of the angiographic appearance of some of the veins of the posterior cranial fossa for diagnosis is discussed.

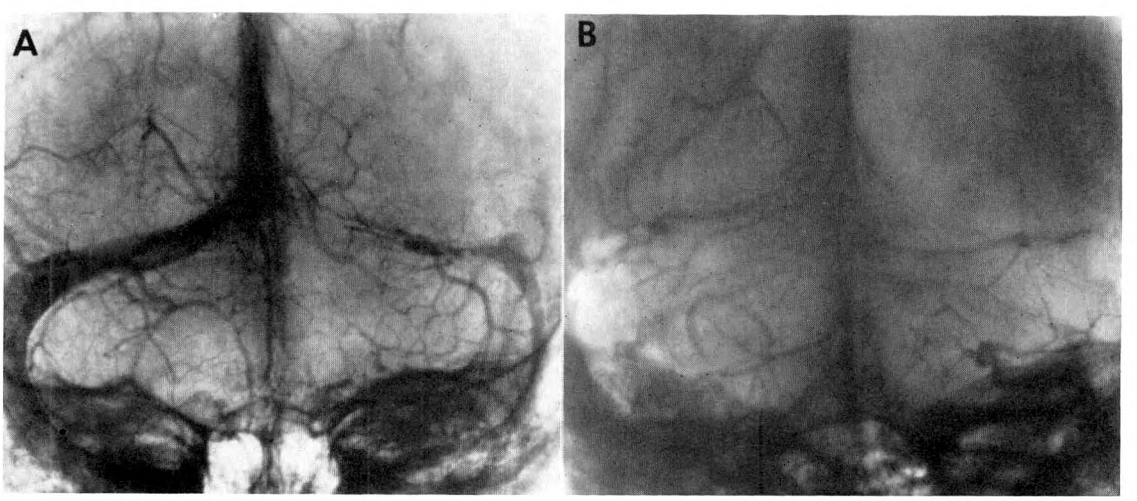


FIG. 12. (*A* and *B*) Two cases of neurinoma of the acoustic nerve in the right cerebellopontine angle. Marked elevation of the petrosal vein. Note the well evident difference with the normal pattern on the opposite side.

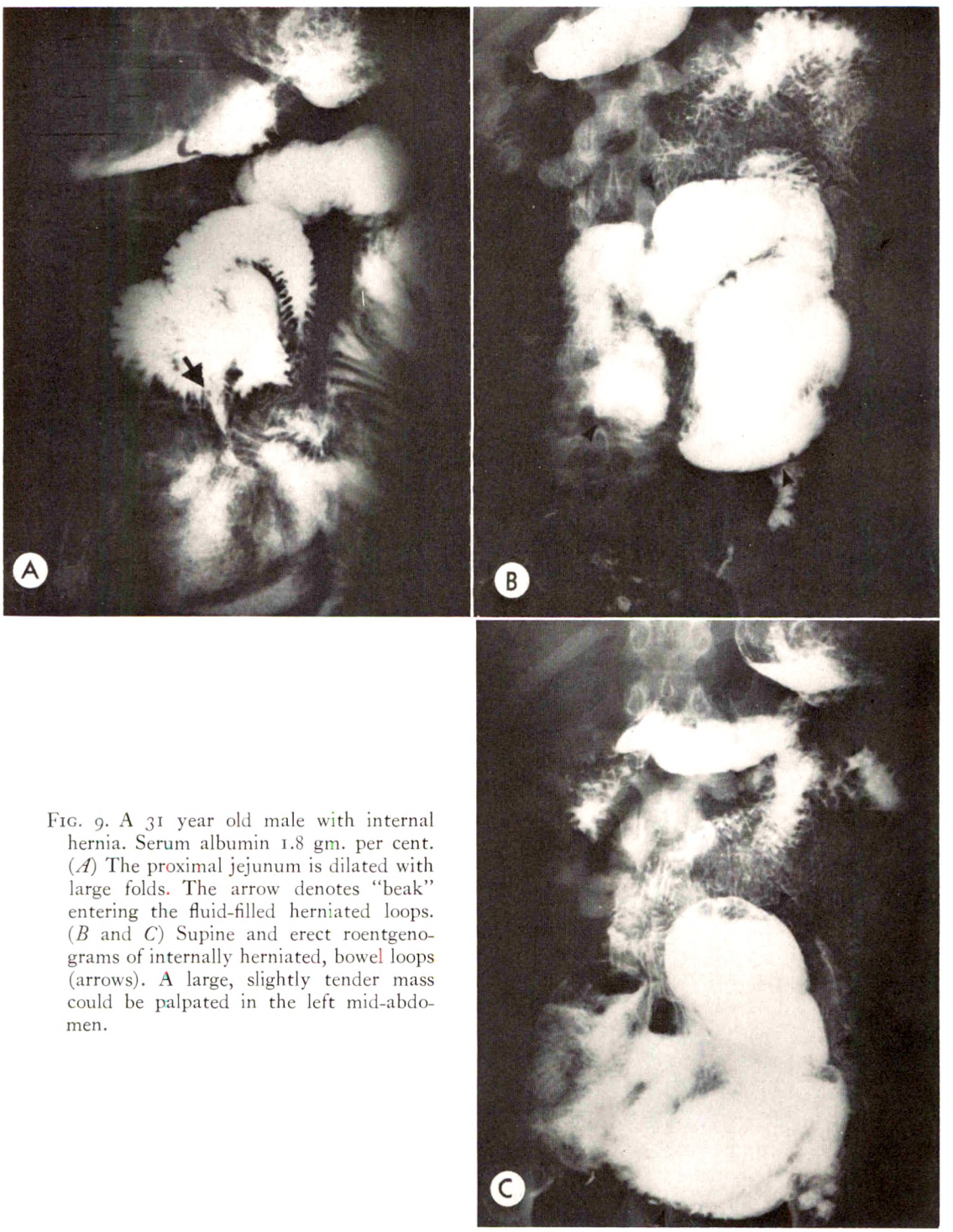


FIG. 9. A 31 year old male with internal hernia. Serum albumin 1.8 gm. per cent. (*A*) The proximal jejunum is dilated with large folds. The arrow denotes "beak" entering the fluid-filled herniated loops. (*B* and *C*) Supine and erect roentgenograms of internally herniated, bowel loops (arrows). A large, slightly tender mass could be palpated in the left mid-abdomen.

abdominal mass. A small bowel examination (Fig. 9, *A–C*) performed during one of these episodes disclosed an internal hernia with proximal intestinal obstruction. In May 1964 the patient again underwent exploratory laparotomy. Approximately 1 liter of cloudy, straw colored ascitic fluid was found. There was thickening of the anterior and posterior parietal

A few demonstrative cases are described and illustrated.

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peritoneum, but no retroperitoneal fibrosis. The entire small bowel was bound up and encased in a firm fibrous capsule. Within this capsule the loops of bowel were adherent to one another with thick adhesions. The most dense scarring was about the ileum where the fibrous tissue had formed small defects through which the small bowel protruded. Proximal to this, the bowel was dilated. Lysis of adhesions was performed. Postoperatively the patient has continued to have recurrent attacks of abdominal pain, unassociated with nausea, vomiting or an abdominal mass.

The ascitic fluid removed at surgery clotted immediately in a glass tube. Electrophoresis of the ascitic fluid showed an albumin of 2.1 gm. per cent and a total protein of 4.4 gm. per cent. On the morning of the day of operation (after intravenous albumin transfusions) the serum electrophoresis disclosed an albumin of 3.6 gm.

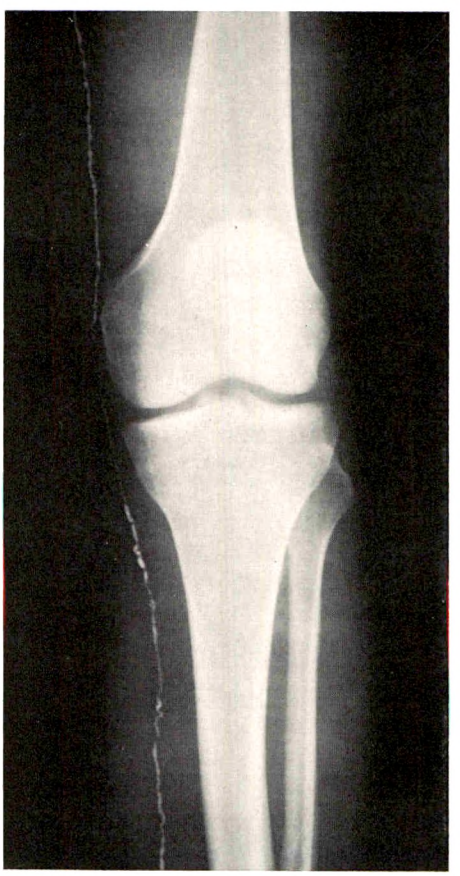


Fig. 10. Hypoplasia of lower extremity lymphatics. Pedal lymphangiogram shows opacification of only a single, tortuous lymphatic vessel in the lower extremity.



Fig. 11. (*A*) Dilated, tortuous thoracic duct. Maximum width, 7 mm.; width at T 3, 6 mm.; normal width at T 3, 1–4 mm.[17]

per cent and a total protein of 5.8 gm. per cent. Cultures of the ascitic fluid were negative for pathogens. Histologic examination of the fibrous capsule enveloping the small bowel disclosed scar tissue and chronic peritonitis. Dense and extensive adhesions should be anticipated after abdominal surgery in patients with intestinal lymphangiectasia.

The results of 4 of the 9 pedal lymphangiographies in this series have been previously reported;[16] 3 of these 4 cases exhibited hypoplasia of the lower extremity lymphatics (Fig. 10). In addition, 1 of these 3 patients showed extensive dermal backflow and probable obstruction of the thoracic duct. The 4th patient had normal abdominal lymphatics, but opacification of only a few small retroperitoneal lymph

ARTERIOVENOUS MALFORMATIONS INVOLVING THE GALENIC SYSTEM*

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THREE categories of midline arteriovenous anomalies have been defined by Litvak *et al.*[4]: (1) aneurysm of the great vein of Galen, which is a dilatation fed directly by anomalous branches of the carotid and/or basilar circulations; (2) a racemose conglomeration of blood vessels with dilated deep venous drainage; and (3) several variations of arteriovenous shunts which are transitional between the aneurysm of the vein of Galen and the racemose angiomas.

While separating midline arteriovenous malformations into these entities is helpful in regard to surgical management, the pathogenesis of these lesions is really based on a single embryologic defect.

It is generally conceded that aneurysm of the vein of Galen is an incorrect term. Despite this, it is still used freely and has led to confusion in the literature as to the nature and frequency of this lesion.[7,8]

The purpose of this paper is to illustrate several cases to justify grouping this entity and its several variations under one term: *i.e.*, arteriovenous malformations involving the Galenic system.

ANATOMY AND EMBRYOLOGY

The vein of Galen is the largest of the deep cerebral veins and although it does not exceed 2 cm. in length, it is the collecting vessel for a large group of veins coming from the deep medially located veins of the diencephalon, basal ganglia and midbrain.

Dilatation of the vein of Galen is obviously a secondary result of the force of arterial blood either directly from an artery or by way of a tributary vein or veins which receive it directly from an artery. The arteriovenous fistula may be immediately adjacent to the vein of Galen or quite remote. This congenital arteriovenous fistula between diencephalic vessels that are embryologically choroidal and drain into the great cerebral vein has already been noted to be much more common than previously thought.[2]

Padget[5] has shown that the primitive arteries and veins are formed on the surface of the neural tube by two separate precapillary plexuses. The pia-arachnoidal arteries are superficial to the pial veins and both are simple endothelial tubes until the 40 mm. stage of the embryo. They cross each other perpendicularly, sometimes in close approximation.

These juxtaposed crossings of the larger arterial and venous endothelial tubes are most conspicuous near the relatively enormous choroidal plexus. Here the posterior choroidal artery crosses and is intimately associated with a comparable vein of equal size. The anterior cerebral artery supplements the supply of the choroid plexus in the embryo prior to the formation of the corpus callosum and is also intimately associated with the veins in this area.

The Galenic venous system is formed by a number of primitive veins of metencephalic, diencephalic and telencephalic origin. The internal cerebral vein receives the superior choroidal vein draining the large choroid plexus. The basal cerebral vein receives telencephalic and diencephalic tributaries and joins the internal cerebral vein to form the great vein of Galen. The mature Galenic system includes the internal cerebral veins, the basal veins of Rosenthal,

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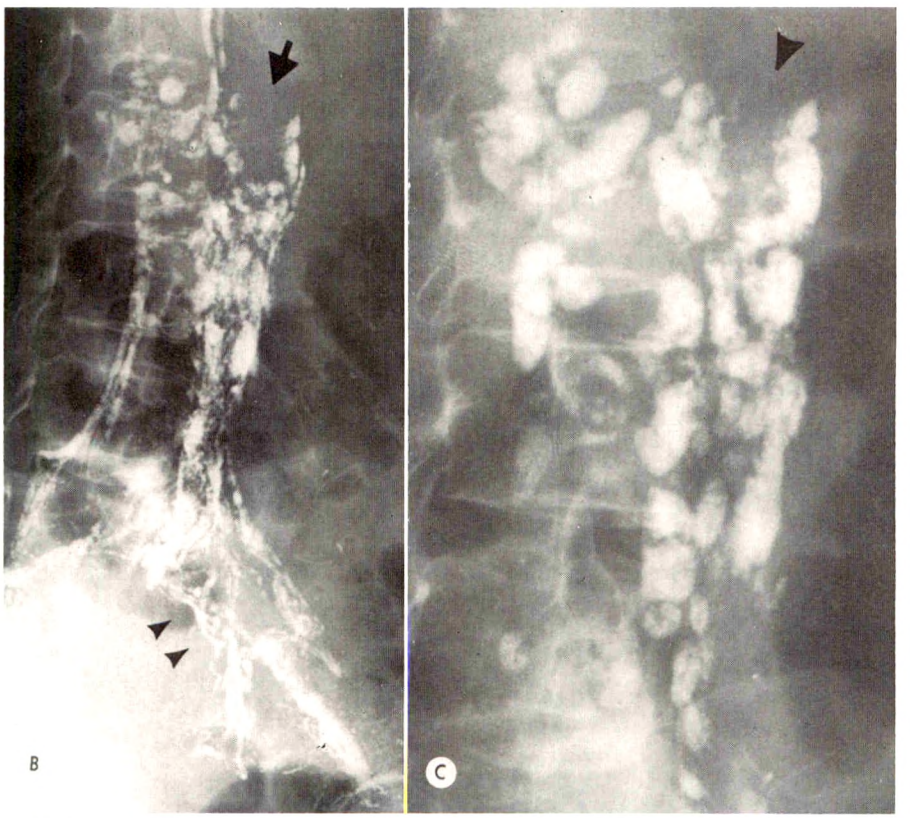


FIG. 11. (*B*) Filling abdominal lymphangiogram. Dilated, varicose iliac lymphatics (2 small arrows) and area of deviation of periaortic lymphatics (single large arrow). (*C*) Twenty-four hour roentgenogram of the abdomen. The area of lymphatic deviation in *B* shows no opacification of the lymph nodes (arrow). The margin of the anterolateral lymph node bounding this area is very irregular. The rest of the small periaortic lymph nodes opacified have numerous, small central filling defects, impossible to reproduce adequately.

nodes. Above the diaphragm, there was a double, tortuous thoracic duct and enlarged supraclavicular lymph nodes.

Two of the 5 patients examined since the previous publication have had normal lymphangiograms. The 3rd, who had a unilateral study only, exhibited tortuous, large lower extremity lymphatics and no passage of oily contrast medium past the distal iliac lymph nodes, with some extravasation of contrast material at that point. The 4th patient, who has been followed for 15 years since the age of 28 years with intestinal lymphangiectasia manifested by peripheral edema, ascites and severe diarrhea, recently developed lymphosarcoma presenting as a breast mass. A lymphangiogram for staging of lymphoma revealed tortuous lower extremity and pelvic lymphatics and a dilated, tortuous thoracic duct (Fig. 11*A*). The periaortic lymph nodes were positive for lymphomatous involvement (Fig. 11, *B* and *C*). The lymphangiography on the 5th patient was performed at the age of 8 years; she has been followed since birth with intestinal lymphangiectasia manifested by peripheral edema, persistent ascites and occasional chylothorax. Only a few tiny lymphatics could be identified in the left foot and these could not be cannulated. Normal lymphatic filling from the right side was obtained to the 2nd lumbar vertebral level (Fig. 12*A*). The cisterna chyli and thoracic duct were never opacified; 24 hour lymphangiograms showed no pulmonary oil

the superior cerebellar veins and the internal occipital veins.

PATHOGENESIS

Arteriovenous malformations may arise when fistulae develop at points where primitive vessels cross during the embryonic stage when they are separated only by a double layer of endothelial cells.[6] The arteriovenous malformation is formed when differentiation of primitive arteriovenous channels into mature arterial, venous and capillary channels fails to take place. Instead the embryonic plexus-like pattern is retained although it is altered secondarily by the effect of blood pressure.

It would appear that the close contact of the primitive Galenic system with the primitive choroidal arteries is an important factor in the production of arteriovenous fistulae in this region. The initial lesion is the congenital fistula between vessels that are embryologically choroidal and drain into the great cerebral vein. The resulting pathologic changes are based on the persistence of a fistula alone or its accompaniment by secondary dilatation elsewhere or by its regression after initiating a secondary arteriovenous malformation. These variations are possible because subsequent venous development is characterized by considerable shifting, added and subtracted anastomoses, and even reversals of current.

The above mentioned types of malformations described by Litvak *et al.*[4] are actually variations of the same initial fistula and clearly result from local developmental failures in the embryo which lead to the persistence of a primitive arteriovenous communication.

ILLUSTRATIVE CASES

Case I (Fig. 1, *A* and *B*) represents a true aneurysm of the vein of Galen.

Case II (Fig. 2, *A* and *B*), Case III (Fig. 3, *A* and *B*), and Case IV (Fig. 4, *A–C*) fall into the racemose category.

One of the transitional types is represented by Case V (Fig. 5, *A* and *B*) in which the arteriovenous malformation is located more remotely from the vein of Galen by

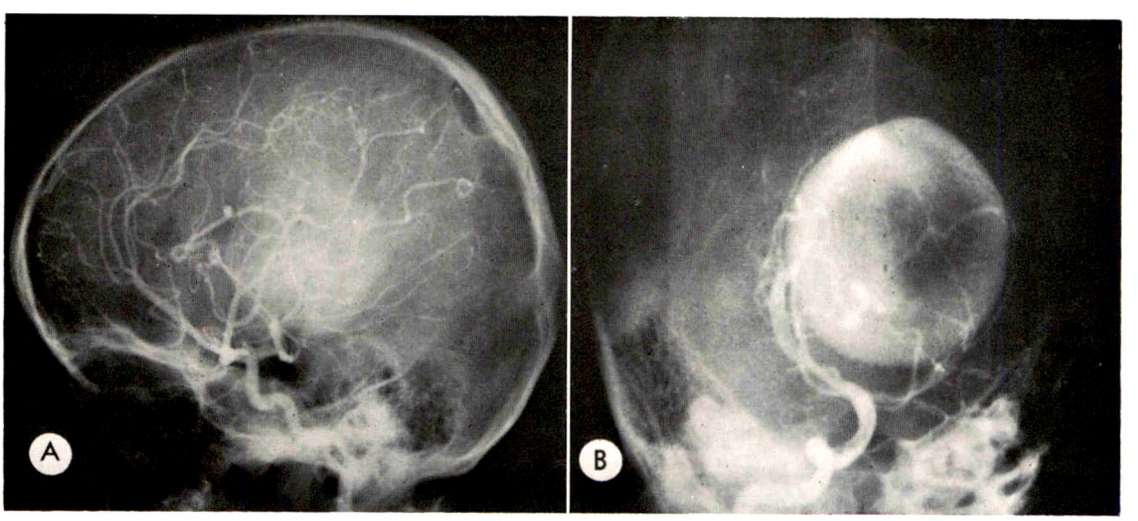


Fig. 1. Case I. (*A*) Lateral carotid angiogram represents the classical malformation in which the posterior cerebral arteries communicate directly with the vein of Galen without an intermediate component. In this case the vein of Galen is the largest part of the malformation. (*B*) This anteroposterior vertebral angiogram represents the classical malformation in which the posterior cerebral arteries communicate directly with the vein of Galen without an intermediate component. In this case the vein of Galen is the largest part of the malformation. Note direct entry of vessels into the giant sized great vein of Galen.

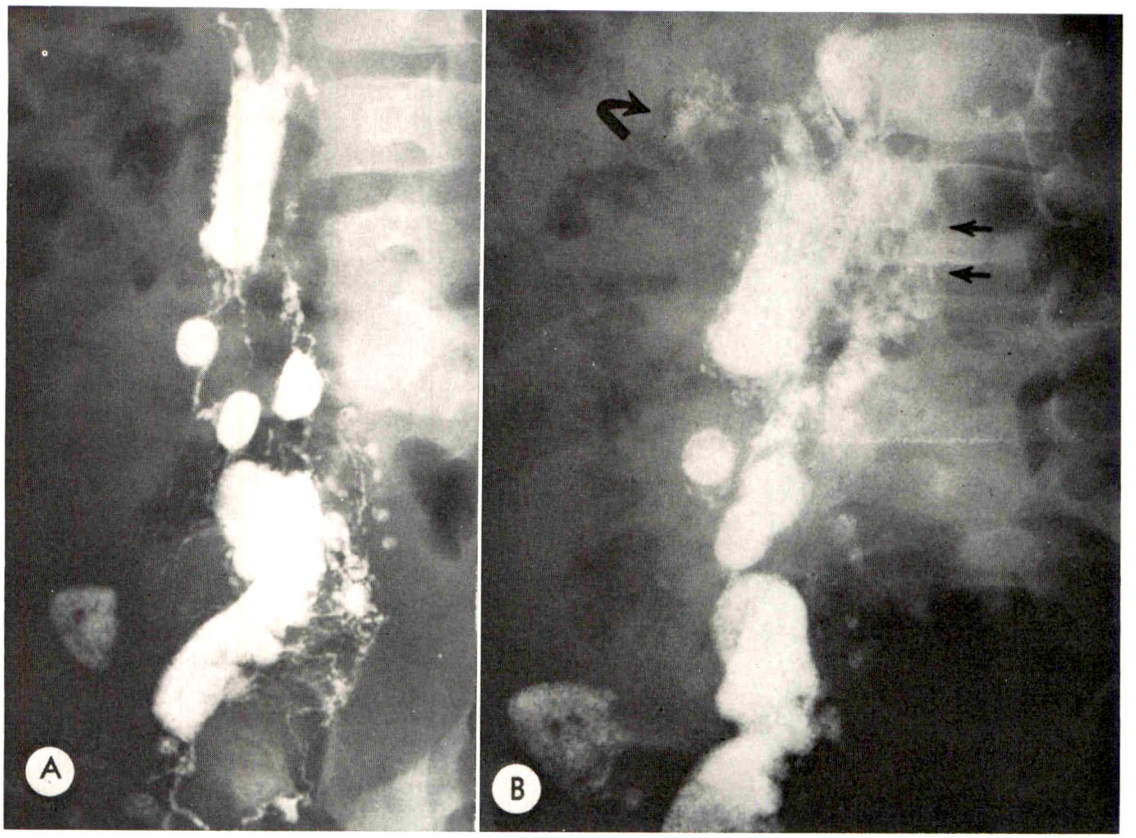


Fig. 12. (*A*) Right-sided lymphangiogram, filling phase. This was the highest level of abdominal filling obtained; normal lymphatics are opacified. (*B*) Twenty-four hour lymphangiogram. Retroperitoneal extravasation of contrast material about the right periaortic lymph nodes (double straight arrows) and mild reflux of the contrast material into mesenteric lymphatics (single curved arrow) are present. Opacified enlarged lymph nodes with abnormal internal architecture are better illustrated in Figure 5*B*.

embolization or opacification of supradiaphragmatic lymph nodes. The 24 hour abdominal roentgenogram revealed extravasation of contrast material about the right periaortic lymph nodes and mild reflux into the mesenteric lymphatics (Fig. 12*B*). All the opacified lymph nodes were enlarged with a "salt and pepper" internal architecture (best seen in Fig. 5*A*). Nine months after the lymphangiography the patient had enlargement of a left supraclavicular lymph node associated with a febrile illness. Biopsy showed nonspecific granuloma and debris; all bacterial cultures of the biopsy specimen were negative.

DISCUSSION

At initial small bowel evaluation 13 of 20 patients manifested diffuse enlargement of the valvulae conniventes with minimal or absent dilatation of the bowel; 9 of these had evidence of increased intestinal secretions. The roentgen differential diagnosis of conditions producing diffuse enlargement of intestinal folds has been considered by Clemett and Marshak.[4] As noted by these authors, in the great majority of cases there should be little confusion of this "large fold pattern" with the "sprue pattern" characteristic of nontropical sprue. Both from a clinical and roentgenographic standpoint, the most frequently considered alternative to intestinal lymphangiectasia is hypoproteinemia due to other causes; *e.g.*, nephrosis or hepatic cirrhosis. Marshak *et al.*[11] have shown that if the serum albumin level is less than 2.5 gm. per cent with such diseases, edema of the bowel may

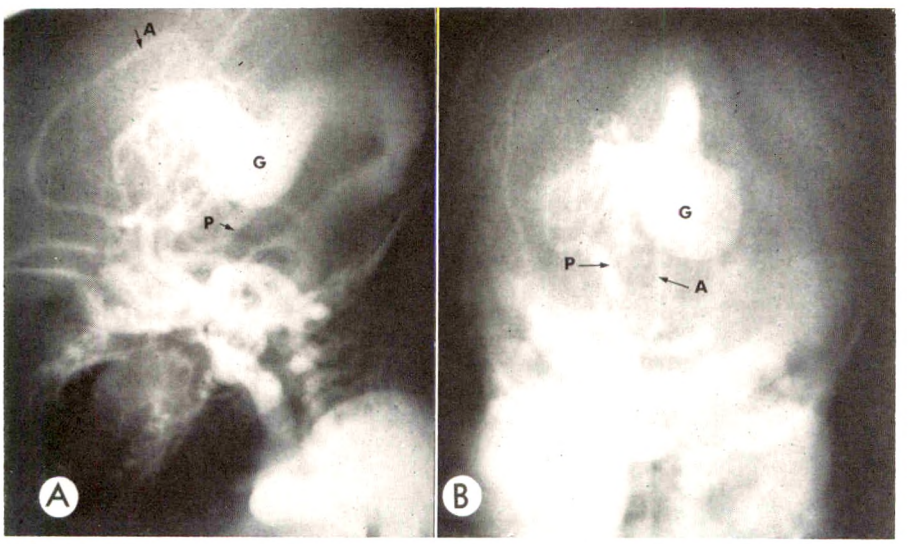


FIG. 2. Case II. (*A*) Lateral arch angiogram. An arteriovenous malformation supplied by the anterior (A) and posterior (P) cerebral arteries and lying lateral and anterior to the vein of Galen (G) into which it drains by way of tributary veins. The vein of Galen (G) is the largest component of the malformation, even though there is an intermediate component. (*B*) Anteroposterior projection of the same study. An arteriovenous malformation supplied by the anterior (A) and posterior (P) cerebral arteries and lying lateral and anterior to the vein of Galen (G) into which it drains by way of tributary veins. The vein of Galen (G) is the largest component of the malformation, even though there is an intermediate component.

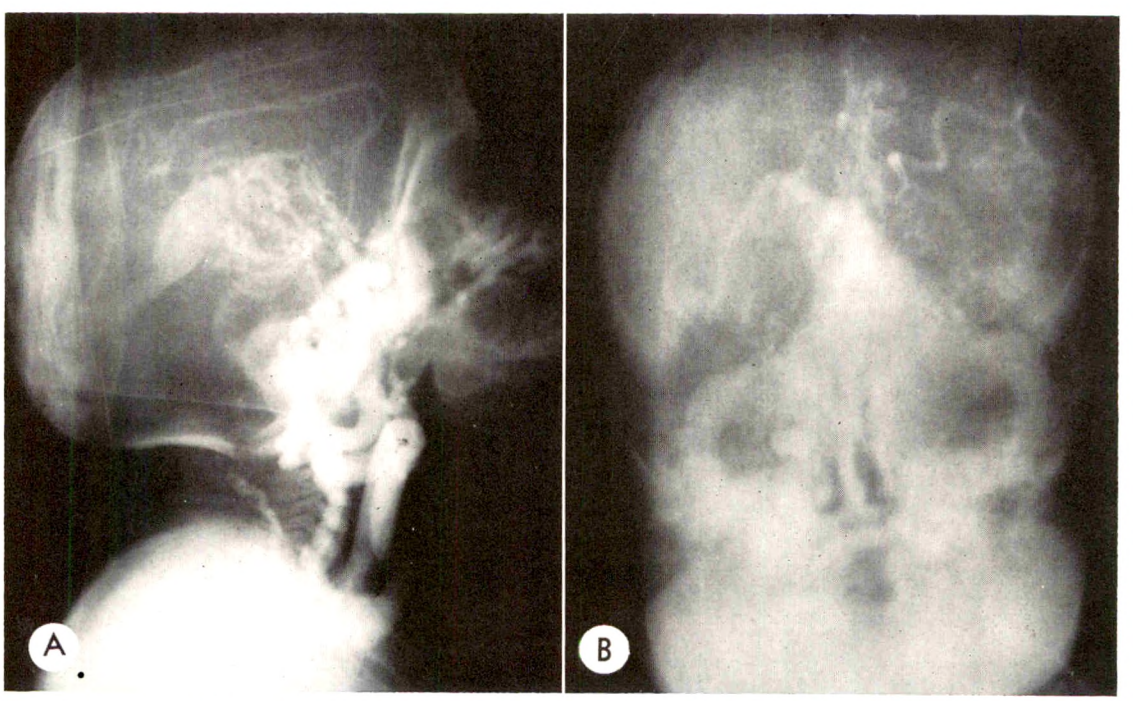


FIG. 3. Case III. (*A*) Lateral arch angiogram. This case demonstrates the arteriovenous malformation to be larger than the vein of Galen which receives the shunted blood by intermediate veins. In addition to the anterior and posterior cerebral arteries, there is a large component of supply from the middle cerebral artery. (*B*) Anteroposterior projection in same case. This demonstrates the arteriovenous malformation to be larger than the vein of Galen which receives the shunted blood by intermediate veins. In addition to the anterior and posterior cerebral arteries, there is a large component from the middle cerebral artery.



FIG. 13. Constrictive pericarditis with protein-losing enteropathy. This is impossible to differentiate from the small bowel appearance of intestinal lymphangiectasia.

be recognized by diffusely thickened intestinal folds. Unless there is evidence of increased secretions, intestinal lymphangiectasia is impossible to differentiate from other causes of hypoproteinemia by the small bowel examination.

The fold enlargement in Whipple's disease often involves only the duodenum and jejunum. Also, in Whipple's disease there may be moderate dilatation of the affected bowel and the pattern of fold enlargement at times is disorganized.[4] Both of these latter features are infrequently observed with intestinal lymphangiectasia. Increased intestinal secretions may be present in Whipple's disease, a condition which has been associated with protein-losing enteropathy.[9] A strikingly symmetric diffuse enlargement of folds ("jejunization") without significant dilatation of the bowel or increased secretions has been recorded in some cases of primary amyloidosis.[7] Occasionally diffuse infiltration of the bowel by lymphoma or leukemia produces enlarged folds. Excessive secretions are not noted and tumor nodules are usually demonstrable. However, 3 or our patients with intestinal lymphangiectasia had several nodules in the jejunum and another had a diffusely nodular pattern of intestinal fold enlargement. Systemic mastocystosis has also recently been shown occasionally to give diffuse enlargement of folds; numerous small nodules may also be noted. Again, increased secretions have not been described.[3] The Zollinger-Ellison syndrome may give enlargement of intestinal folds and increased secretions, but the gastric and duodenal changes should readily exclude its consideration.[25] Clinical criteria usually resolve many of these roentgenographically confusing entities.

Two causes of the "large fold pattern" not considered by Clemett and Marshak[4] should be mentioned. Allergic gastroenteropathy is a syndrome in infants consisting of protein-losing enteropathy, peripheral edema, eosinophilia, iron deficiency anemia and multiple manifestations of allergy. These patients have indistinguishable small bowel patterns from intestinal lymphangiectasia including increased intestinal secretions.[24] Finally, patients with chronic constrictive pericarditis may have small bowel findings identical to intestinal lymphangiectasia (Fig. 13). This is not surprising, as these patients may have a lymphatic disorder with marked gastrointestinal protein loss, peripheral edema, recurrent ascites, and pleural effusions. Small bowel biopsy in several such patients has disclosed findings identical to intestinal lymphangiectasia.[13,14,20] Clinically and roentgenographically constrictive pericarditis should be carefully excluded in any patient considered to have intestinal lymphangiectasia. Corrective cardiac surgery may lead to complete resolution of the gastrointestinal protein loss.[13]

The bizarre appearance to the ileum illustrated by Figure 6, *A* and *B*, has been previously noted by Marshak *et al.*[11] in

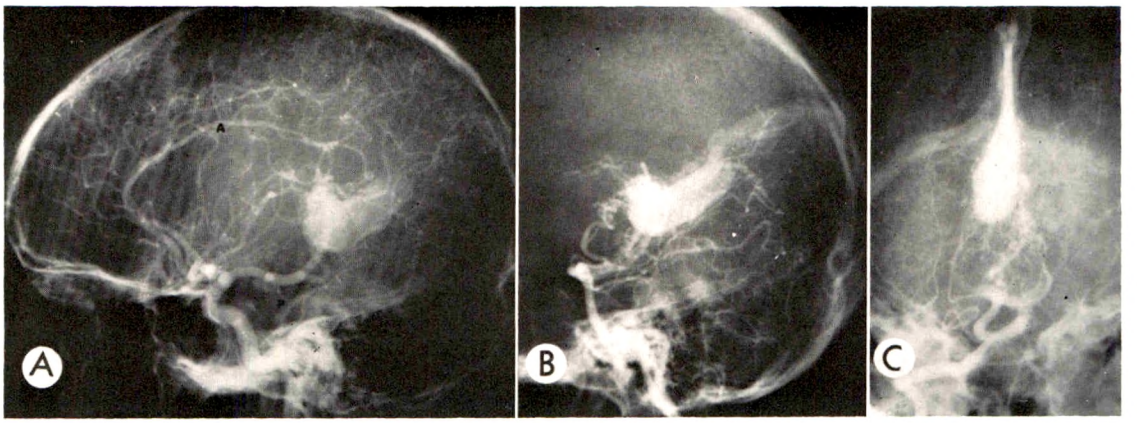


Fig. 4. Case IV. (*A*) Lateral carotid angiogram. This case shows a malformation anterior and lateral to the vein of Galen supplied by the anterior (A) and posterior (P) cerebral arteries. The drainage is by way of choroidal veins to the vein of Galen which is dilated, but not to a global size. (*B*) Lateral and (*C*) anteroposterior vertebral angiograms. This case shows a malformation anterior and lateral to the vein of Galen supplied by the posterior cerebral arteries. The drainage is by way of choroidal veins to the vein of Galen which is dilated, but not to a global size.

the developmental shifting of the primitive vascular system. However, it still maintains its primary drainage into the vein of Galen by way of a dilated superior cerebellar vein.[9]

Case VI (Fig. 6, *A–C*) is another variation in which the dilatation of the vein of Galen is minimal as compared to the intermediate veins, in this case the internal cerebral veins.

Other varieties result from regression of the original dilatation of the vein of Galen by thrombosis of the intermediate veins between the arteriovenous malformation and the vein of Galen.[1,3] This results in a normal appearing or a thrombosed vein of

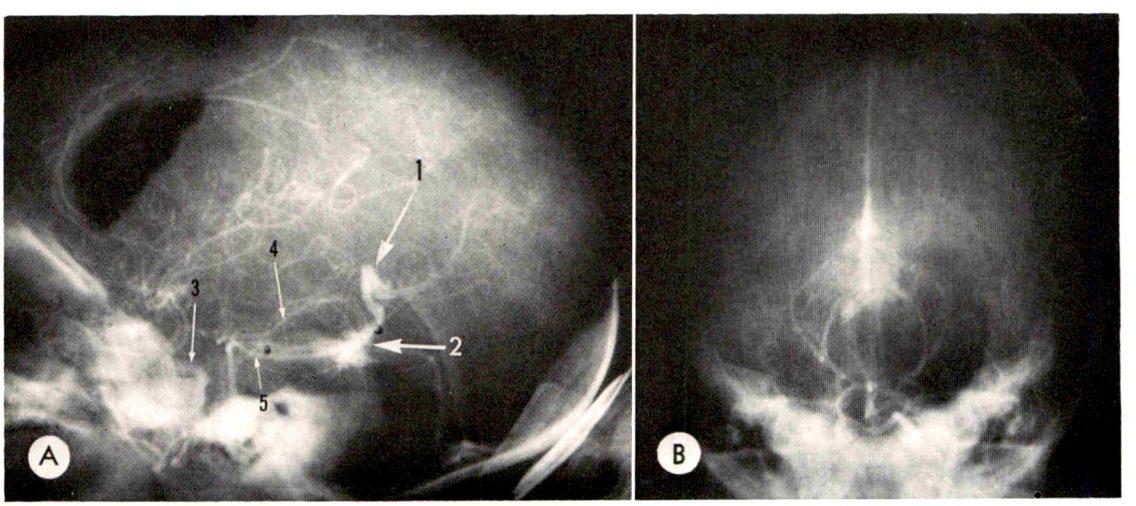


Fig. 5. Case V. (*A*) Lateral arch angiogram. The arteriovenous malformation is actually in the posterior fossa (2) and is supplied by the superior cerebellar arteries (5). Drainage is by way of an enlarged superior cerebellar vein into the vein of Galen (1). (*B*) Anteroposterior roentgenogram of same case. The arteriovenous malformation is actually in the posterior fossa and supplied by the superior cerebellar arteries. Drainage is by way of an enlarged superior cerebellar vein into the vein of Galen. This patient presented with a subarachnoid hemorrhage.

patients with significant hypoalbuminemia. The same authors also illustrate edematous changes in the right colon. All barium enema examinations performed on our patients were normal. In regard to intestinal lymphangiectasia, it remains a moot question whether the edema of the valvulae conniventes and mesentery is secondary to generalized hypoproteinemia and/or a local predilection for interstitial fluid accumulation due to abnormal lymphatic dynamics. Marshak *et al.* imply that hypoalbuminemia alone is responsible.

Other authors[4] have noted tiny, punctate lucencies 1 mm. or less on the small bowel examination in patients with Whipple's disease and intestinal lymphangiectasia. Similar findings were seen in a few of our patients (Fig. 5*C*). It is tempting to consider these a manifestation of enormously enlarged villi secondary to dilated submucosal lymphatics.

The many abnormal lymphangiograms obtained in patients with intestinal lymphangiectasia suggest that the disorder is systemic lymphatic dysplasia. Other evidence supporting this conclusion has been the frequent presence of chylous effusions and asymmetric peripheral edema, even following albumin transfusions.

Abnormal findings in the lower extremities are similar to those seen in patients with primary lymphedema;[6] *i.e.*, either hypoplasia of lymph vessels or dilated, varicose lymphatics.[2,8,16] Either finding may occasionally be associated with dermal backflow. Thoracic duct abnormalities have been seen less frequently. In this series of 9 lymphangiographies, 2 patients had probable obstruction or failure of development of the thoracic duct, and 2 others dilated, tortuous thoracic ducts. The latter finding has been noted in patients with hepatic cirrhosis, congestive heart failure and constrictive pericarditis as a consequence of increased lymph production with resultant increased flow of lymph under increased pressure.[18] Patients with constrictive pericarditis and dilated thoracic ducts have had protein-losing enteropathy and jejunal biopsy findings identical to intestinal lymphangiectasia.[13,20]

Lymphangiographic studies have demonstrated several abnormalities involving abdominal lymphatics. In 1 patient in this series there were normal lymphatics, but marked hypoplasia of lymph nodes.[16] Figure 12*B* is the only example in this series of reflux of contrast material into the mesenteric lymphatics—a finding more dramatically documented by other reports.[2,12,21] In association with mesenteric reflux, others have shown extravasation of contrast material into the proximal small bowel[8,12,21] and even into the peritoneum in a patient with chylous ascites.[8] Demonstration of extravasation of contrast material into the intestine lends support to the possible mechanism of gastrointestinal protein loss in some of these patients; *i.e.*, rupture of dilated lymphatic vessels with leakage of their contents into the bowel. This proposed mechanism is also supported by the finding of lymph in the bowel by intubation in some patients and the lymphopenia present in almost all patients with this disorder.

The pathogenesis of contrast reflux into the mesenteric lymphatics has not been fully defined. When pedal lymphangiography is performed after obstruction of the thoracic duct in the neck or chest, lymph flow usually bypasses the obstruction by means of intrathoracic collaterals and occasionally by lymphaticovenous communication.[17] Noting this, Bookstein *et al.*[2] postulated an anomaly of lymphatic drainage to explain the lymphangiographic mesenteric reflux in his patient with intestinal lymphangiectasia in whom a normal thoracic duct was also demonstrated. Mistilis *et al.*[12] also noted contrast material in the thoracic duct in a patient with lymphangiectasia with lymphangiographic mesenteric reflux. More recently Larson *et al.*[8] have demonstrated lymphangiographic mesenteric reflux in patients with obstruction at the level of the cisterna chyli. One of these patients was a newborn with

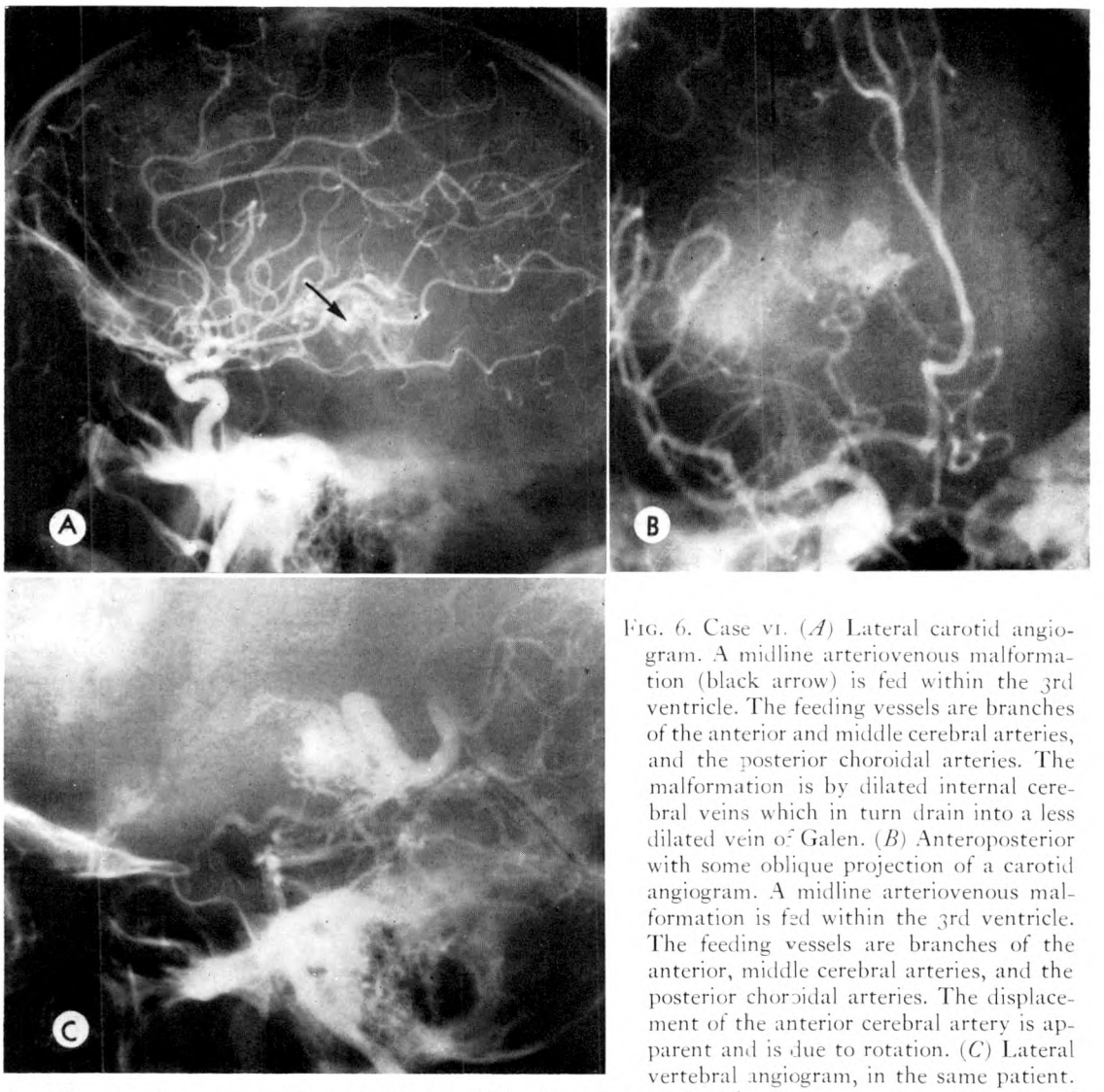


FIG. 6. Case VI. (*A*) Lateral carotid angiogram. A midline arteriovenous malformation (black arrow) is fed within the 3rd ventricle. The feeding vessels are branches of the anterior and middle cerebral arteries, and the posterior choroidal arteries. The malformation is by dilated internal cerebral veins which in turn drain into a less dilated vein of Galen. (*B*) Anteroposterior with some oblique projection of a carotid angiogram. A midline arteriovenous malformation is fed within the 3rd ventricle. The feeding vessels are branches of the anterior, middle cerebral arteries, and the posterior choroidal arteries. The displacement of the anterior cerebral artery is apparent and is due to rotation. (*C*) Lateral vertebral angiogram, in the same patient. A midline arteriovenous malformation is fed within the 3rd ventricle. The feeding vessels are branches of the posterior choroidal arteries and branches of the perforating arteries. The malformation is by dilated internal cerebral veins which in turn drain into a less dilated vein of Galen.

Galen and secondary venous drainage of the malformation.

CONCLUSION

It is suggested that the term arteriovenous malformations involving the Galenic system will more clearly designate the pathogenesis of these lesions and further classification may be based on the clinical entities of congestive heart failure, obstruction of the cerebrospinal fluid pathways or subarachnoid hemorrhage.

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intestinal lymphangiectasia. In our patient with lymphangiographic mesenteric reflux there was no evidence of contrast flow into or beyond the cisterna chyli.

SUMMARY

The roentgenographic features of 20 patients with intestinal lymphangiectasia are presented.

Five had normal small bowel studies.

Of the 15 patients with an abnormal small bowel study at initial evaluation 9 had a characteristic appearance consisting of enlargement of the valvulae conniventes of both jejunum and ileum, increased secretions, and minimal or absent dilatation of the bowel. Four patients had nodular mucosal defects. Two patients had a striking, irregularly serrated margin to the ileum. One patient developed an internal hernia secondary to dense adhesions found after a partial jejunal resection.

A roentgenographic differential diagnosis of the characteristic appearance of the disease is discussed.

Pedal lymphangiographic findings of 9 patients were abnormal in 7, supporting the concept that this disease is a systemic lymphatic dysplasia. Lower extremity abnormalities were similar to those associated with primary lymphedema. In the abdomen 1 patient had hypoplasia of lymph nodes, another showed moderate contrast material reflux into mesenteric lymphatics associated with possible obstruction of the cisterna chyli and enlarged lymph nodes. Two patients had enlarged, tortuous thoracic ducts, and 2 patients had probable obstruction or absence of this structure.

Recently, a patient followed for 15 years with intestinal lymphangiectasia has developed disseminated lymphosarcoma.

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THE NEURORADIOLOGY OF HEMANGIOBLASTOMAS OF THE CEREBELLUM*

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CEREBELLAR hemangioblastomas constitute 7.3 per cent of the primary tumors of the posterior fossa in adults.[10] Neuroradiology plays a specific and definitive role in the diagnosis. Most cases are investigated with a combination of angiography and ventricular studies. Angiography with subtraction studies should enable one to make an accurate diagnosis.

Based on the radiologic appearances of 8 selected cases and a review of the features in previously described series,[2,6,11,13,14] a new and simplified classification will be presented.

REPORT OF CASES

CASE I. A 36 year old male was admitted for the evaluation of headaches of 2–3 months' duration. Neurologic examination revealed a tendency to veer to the right on walking with cerebellar signs on the right side. The hematocrit was normal. Right brachial angiography demonstrated depression of the right posterior inferior cerebellar artery and lateral displacement to the left of the vermial and cerebellar branches (Fig. 1, *A–C*). The basilar artery was anteriorly displaced. The sweep of the pericallosal artery was increased suggesting ventricular dilatation. In the intermediate and the venous phases of angiography, 2 discrete dense vascular nodules were seen inferiorly in the right cerebellar hemisphere. Each measured approximately 10×10×10 mm. in size. Both lesions had translucent centers. Subtraction studies aided in the visualization of the lesions, especially in the frontal view. Pantopaque ventriculography (Fig. 1, *D* and *E*) demonstrated displacement of the aqueduct and the fourth ventricle anteriorly and to the left. A suboccipital craniectomy and upper cervical laminectomy were carried out. A large cyst with a small strawberry-sized vascular tumor was excised from the right cerebellar hemisphere. Histologic examination revealed an abnormal increase in the small blood vessels with plumper than normal cells. The diagnosis was that of a capillary hemangioblastoma.

CASE II. W. K., a 50 year old male, was admitted because of gait unsteadiness, right arm clumsiness and slurred speech. Neurologic examination demonstrated right-sided cerebellar signs with ataxia.

The family history disclosed that one brother died of polycystic kidneys. The patient was not polycythemic. Left brachial angiography (Fig. 2*A*) demonstrated a moderate sized posterior meningeal artery derived from the left vertebral artery. A faint nodular stain measuring 12×12×8 mm. was seen in the right cerebellar hemisphere from the arterial through the venous phases of angiography. The stain was only clearly defined on subtraction studies. On the right brachial angiogram (Fig. 2*B*) the choroid point of the right posterior inferior cerebellar artery was displaced anteriorly and inferiorly. The stain was again seen in the right cerebellar hemisphere superiorly, and subtraction studies assisted in its identification (Fig. 2, *C* and *D*). Pneumoencephalography (Fig. 2, *E* and *F*) demonstrated displacement of the fourth ventricle to the left with upward tilting of the roof. The right cerebellopontine angle did not fill with air. The lateral ventricles were enlarged.

At operation (right suboccipital craniectomy) a large cyst containing clear colored fluid was found in the right cerebellar hemisphere. A tumor nodule was present in the anteromedial aspect of the cyst. Histologic examination revealed a hemangioblastoma.

The patient was hypertensive with a blood pressure of 210/110 mm. Hg. Following further clinical studies the right adrenal gland was explored and a right adrenalectomy carried out. However, on histologic analysis the adrenal gland was unremarkable. The patient remains hypertensive.

CASE III. F. H., a 50 year old male, was admitted because of gait unsteadiness and headaches of 5–6 months' duration. Lumbar

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SUBMUCOSAL LYMPHATIC CYSTS OF THE GASTROINTESTINAL TRACT: A RARE CAUSE OF SUBMUCOSAL MASS LESION*

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ROCHESTER, MINNESOTA

INTRAMURAL lesions of the gastrointestinal tract are relatively uncommon but may occur at any site throughout its length. The lesions may be derived from any one of the mesodermal elements that compose the gastrointestinal tract wall or from the ectodermally derived nerves in the wall. Malignant neoplasms that are metastatic to the bowel wall from distant organs or from other parts of the gastrointestinal tract also may occur. Intramural nodules of aberrant pancreas have been seen in the duodenum. Closed duplication of part of the bowel, intramural hematoma, and varices or, rarely, isolated varix may all present as intramural mass lesions.

When the barium filled gastrointestinal tract is examined roentgenologically, any of the previously mentioned entities may be seen as a submucosal filling defect that is relatively radiolucent in contrast to the surrounding barium. However, some of these lesions appear more sharply circumscribed than others and have distinctive roentgenographic features that enable the radiologist to suggest a tissue diagnosis with some degree of accuracy.

This applies in particular to lipomas, isolated varix, and submucosal lymphatic cysts. The purpose of this communication is to describe the roentgenographic features of the submucosal lymphatic cyst and to emphasize the inclusion of this rare benign cyst in the differential diagnosis of a submucosal lesion that displays such characteristics.

MATERIAL AND METHOD

From 1959 through 1968, 1,437,767 patients, on whom 453,708 roentgenographic examinations of the gastrointestinal tract were performed, were seen at the Mayo Clinic. Of that number, 9 had lymphatic cysts excised from the gastrointestinal tract. Three of the 9 patients had had relevant roentgenographic findings. Because the roentgenographic characteristics of submucosal lymphatic cyst are similar to those reported in the case of submucosal lipoma,[4,5,10] we compared the data of our series with the films and fluoroscopic reports on 9 patients who had had gastric lipomas excised in an effort to delineate any differences.

RESULTS

In 7 of the 9 patients, the cysts were single and submucosal. In the other 2 patients, multiple cysts were scattered throughout the serosa of the small intestine and the omentum, peritoneum, and broad ligaments.

Of the 7 patients with single submucosal cysts, 4 were men and 3 were women. Three of the cysts were located in the duodenum, 2 in the stomach, 1 in the jejunum, and 1 in the descending colon. Five of the lesions ranged from 2 cm. to 4 cm. in diameter, and the other 2 were 0.5 cm. and 0.6 cm., respectively. Three of the lesions were seen on barium examination of the gastrointestinal tract and were diagnosed roentgenographically as lipoma or leiomyoma. The other 4 cysts were coincidental findings at operation.

The outstanding roentgenographic features in the 3 cases examined were those of sharply defined, smooth, curvilinear filling

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punctures revealed elevated protein levels of 112 and 98 mg. per cent. Minimal left-sided cerebellar signs were present with a tendency to fall to the left when sitting. His gait was wide-based. The hematocrit was normal.

Right brachial angiography revealed downward displacement of the posterior inferior cerebellar artery. Commencing in the intermediate phase and present through to the venous phase, a discrete round nodular density was seen in the superior cerebellar vermis (Fig. 3*A*). It measured 6×10×10 mm. and had a translucent center. A small draining vein was present. Considerable ventricular dilatation was seen, as evidenced by the wide sweep of the thalamostriate vein in the frontal projection. A left brachial angiogram demonstrated a well defined posterior meningeal branch of the vertebral artery (Fig. 3*B*). Pantopaque ventriculography demonstrated considerable anterior displacement of the aqueduct of Sylvius and of the fourth ventricle.

A suboccipital craniectomy was performed with removal of a midline cerebellar tumor nodule lying within a large cyst. The histologic report was that of a hemangioblastoma.

Case iv. K.S., a 64 year old male, was admitted because of a 3 week history of frontal headaches and gait unsteadiness. Neurologic examination was negative except for a staggering gait. Cerebrospinal fluid examination was unremarkable. The hematocrit was normal. Simultaneous bilateral brachial angiography showed a posterior meningeal branch of the vertebral artery, no visualization of the pos-

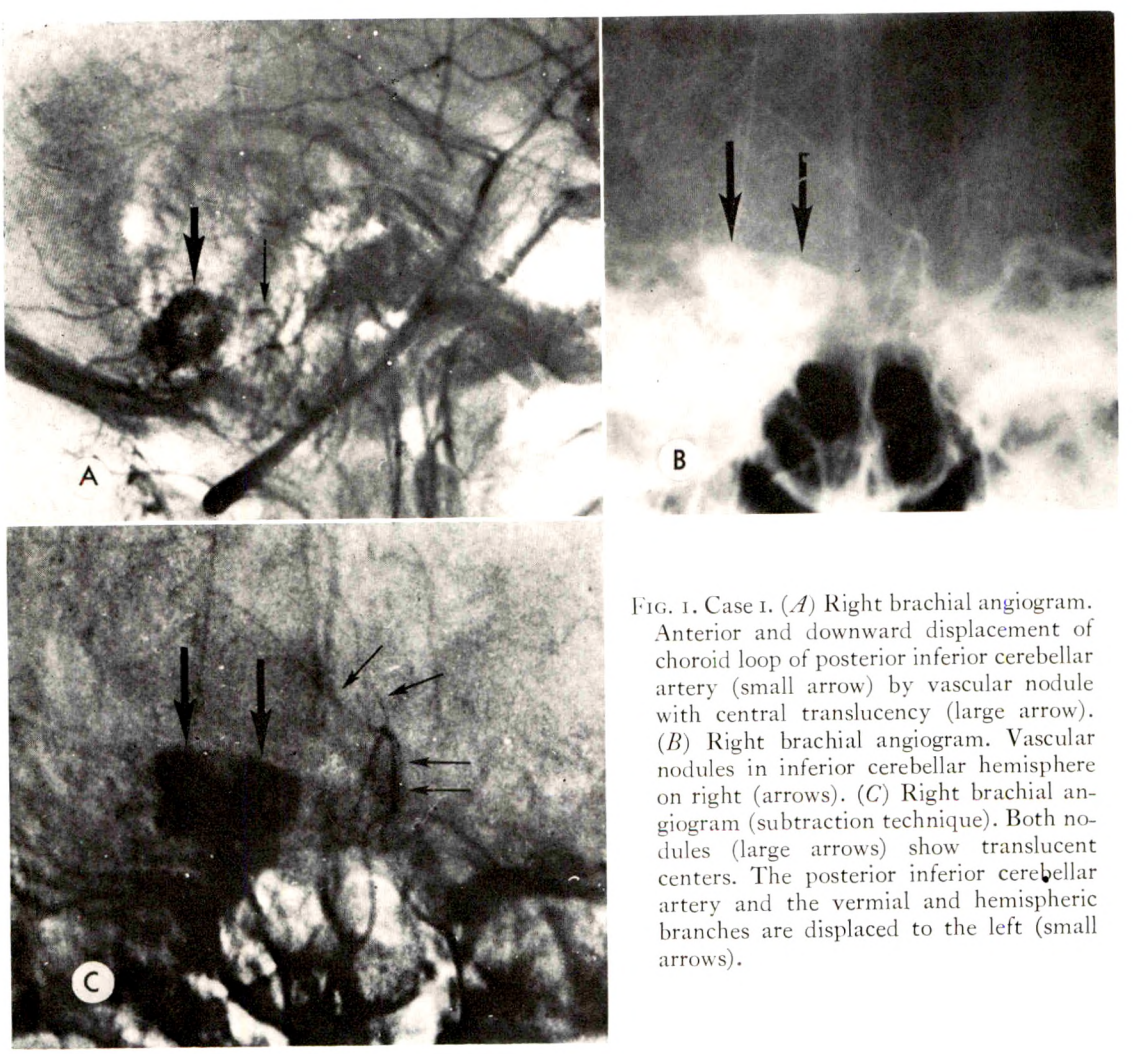


Fig. 1. Case 1. (*A*) Right brachial angiogram. Anterior and downward displacement of choroid loop of posterior inferior cerebellar artery (small arrow) by vascular nodule with central translucency (large arrow). (*B*) Right brachial angiogram. Vascular nodules in inferior cerebellar hemisphere on right (arrows). (*C*) Right brachial angiogram (subtraction technique). Both nodules (large arrows) show translucent centers. The posterior inferior cerebellar artery and the vermial and hemispheric branches are displaced to the left (small arrows).

defects which at fluoroscopy were seen to change shape readily in response to compression and peristaltic activity. The overlying mucosa was not ulcerated. A brief résumé of the 3 cases is given.

REPORT OF CASES

Case I. A 79 year old man was examined because of vague dyspepsia. A sharply circumscribed, easily compressible, gastric filling defect was found; this was believed to be a lipoma. Because of the possible sarcomatous potential of the lesion, exploration was undertaken, and a 3.5 cm. submucosal cyst lined by flattened endothelial cells and filled with a thin serous fluid was found (Fig. 1). The overlying mucosa showed mild chronic gastritis.

Case II. A 60 year old man had epigastric discomfort and melena of short duration. A sharply defined, rounded filling defect found in the second part of the duodenum was believed to be a lipoma or a leiomyoma. At operation, a 2.5 cm. endothelial lined lymphatic cyst containing a slightly viscid colorless fluid was found. The cyst was covered by normal mucosa (Fig. 2).



Fig. 1. Case I. A sharply defined, easily compressible, filling defect of submucosal origin on lesser curvature of stomach of a 79 year old man. The lesion was believed to be a lipoma because of its pliability. At operation, a 3.5 cm. submucosal lymphatic cyst lined by flattened endothelial cells and filled with a thin serous fluid was found.



Fig. 2. Case II. Sharply defined defect in second part of duodenum was believed to be a lipoma or a leiomyoma. At operation, a 2.5 cm. endothelial lined lymphatic cyst containing a slightly viscous colorless fluid was found. The lesion was covered by normal mucosa.

Case III. A 62 year old man had a colon examination during a routine medical evaluation. He had no gastrointestinal symptoms. The sharply defined filling defect in the upper descending colon was believed to be a leiomyoma. A 2 cm. thin-walled lymphatic cyst lined by flattened endothelium was excised (Fig. 3*A*).

Comment. Of the 9 patients who had gastric lipomas excised, 5 had sharply defined smooth lesions that were indistinguishable from the lymphatic cyst and 4 had poorly defined irregular lesions easily distinguishable from the lymphatic cyst.

DISCUSSION

A lymphatic cyst is a thin-walled cyst that may be unilocular or multilocular, is lined by flattened endothelium, and contains a thin serous or milky fluid (Fig. 3*B*). The cause of these cysts is uncertain. They are benign and in our experience are asymptomatic.

The lymphatic cyst should not be confused with generalized intestinal lymph-

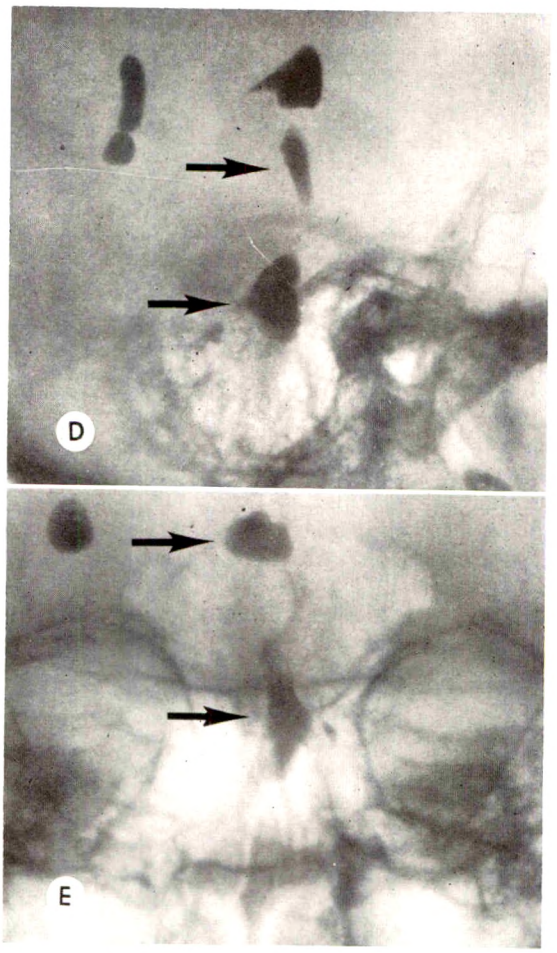


FIG. 1. (*D*) Pantopaque ventriculogram (Polaroid print). Anterior displacement of aqueduct (upper arrow) and fourth ventricle (lower arrow). (*E*) Pantopaque ventriculogram. Slight lateral tilting of posterior third ventricle (upper arrow). Lateral displacement of fourth ventricle to left (lower arrow).

terior inferior cerebellar arteries and a discrete vascular nodular density in the right superior cerebellar hemisphere. The lesion which had slight central translucencies measured 10×9×4 mm. A suboccipital craniectomy and upper cervical laminectomy were carried out. A cyst was found in the right cerebellar hemisphere containing approximately 35 cc. of clear xanthochromic fluid. A beefy red nodule was attached to the wall of the cyst. Histologic analysis of the nodule revealed a hemangioblastoma.

CASE V. L. N., a 33 year old female, was admitted for the evaluation of nausea, vomiting and vertigo of 8 months' duration. Neurologic examination revealed some decrease in balance and difficulty in walking heel-to-toe with the eyes open. The hematocrit was normal. An intravenous pyelogram was normal. Left brachial angiography was carried out and demonstrated a tangle of vessels measuring 20×15×13 mm. in the inferior vermis fed by a slightly dilated posterior inferior cerebellar artery. The choroid point was displaced anteriorly. An immediate shunt of contrast material into an inferior vermial vein was seen (Fig. 4, *A–C*). The remainder of the vessels of the posterior fossa in the arterial and venous phases were unremarkable.

A suboccipital craniotomy and C-1 laminectomy were carried out. The tumor mass which was solid with no associated cyst was easily

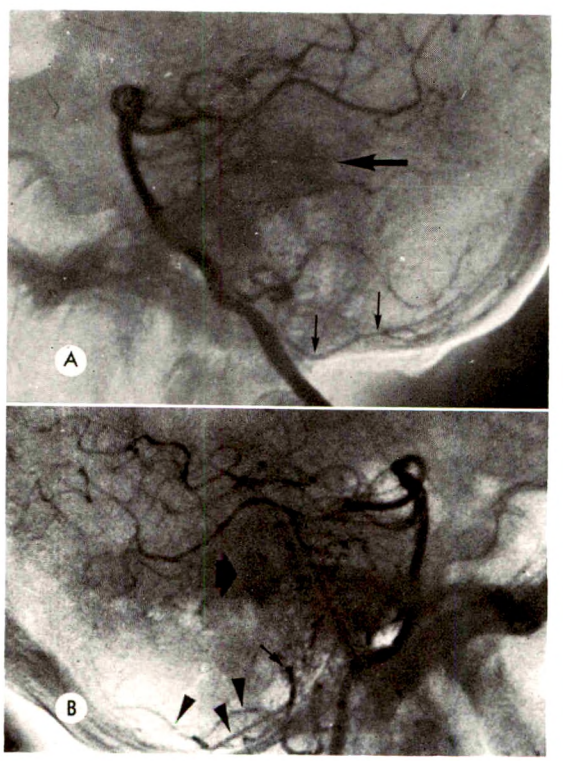


FIG. 2. Case II. (*A*) Left brachial angiogram. Posterior meningeal artery (small arrows) derived from left vertebral artery. Faint stain seen in superior cerebellum (large arrow). (*B*) Right brachial angiogram (with compression of right carotid artery). Anterior displacement of choroid point of posterior inferior cerebellar artery (small arrow) with downward displacement of vermial and hemispheric branches (arrowheads). Nodular stain seen in superior cerebellar hemisphere (large arrow). Superior cerebellar arteries are poorly visualized.

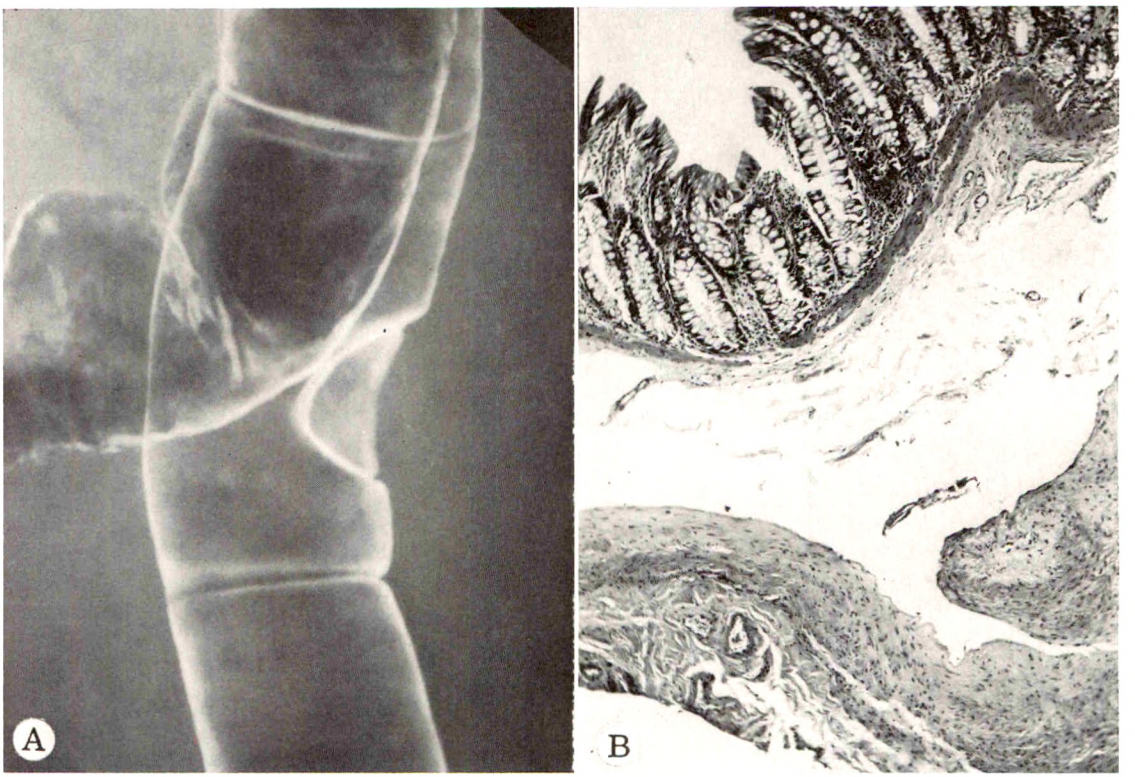


FIG. 3. Case III. (*A*) Sharply defined filling defect in descending colon just below the splenic flexure was believed to be a leiomyoma. At operation, a 2 cm. thin-walled lymphatic cyst was excised. (*B*) Submucosal lymphatic cyst (collapsed) lined by flattened epithelium and with intact overlying mucosa. (Hematoxylin and eosin; ×50.)

angiectasia. In this condition, there is cystic dilatation of submucosal lymphatic vessels which usually involves the small intestine only but which also may be found confined to the colon.[3,7,13]

A previous review[1] reported 9 cases of lymphatic cysts of the abdomen seen at the Mayo Clinic during the 36 years between 1911 and 1947. The lesions were mesenteric in 6 cases and retroperitoneal in 2. In 1 case, the lesion was of undetermined origin. There were no intramural lymphatic cysts. Our review of the English language literature revealed only 1 reference to a similar lesion,[2] except for a recent report from our institution of 1 of the cases in the present series.[8]

It has been stated that because of the low radiodensity of fat, radiolucency is diagnostic of lipoma.[11] Others have suggested that this applies only to large lipomas and that, since small ones are more commonly seen, radiolucency is, in fact, rare in lipomas.[5] However, the perception of an image is governed by the pattern of illumination projected on the retina and is dependent on the characteristics of the border of the retinal image and not on its area.[12]

If a relatively lucent area is demarcated by a sharp border, the impression of lucency is greater than if it is bounded by a fuzzy ill-defined margin. This is true whether the area is small or large. Therefore, radiolucency in a submucosal gastrointestinal lesion probably is a function of the sharpness of the demarcation from the surrounding tissue. In our series, each of the lymphatic cysts examined roentgenographically had a sharply defined border and appeared radiolucent, whereas just over half of the lipomas we studied had

peeled away from normal vermis tissue. Histologic analysis demonstrated a capillary hemangioblastoma.

Case vi.* J.M., a 40 year old female, was admitted complaining of a generalized head-

* Courtesy of Dr. A. Moon, Department of Radiology, Rhode Island Hospital, Providence, Rhode Island.

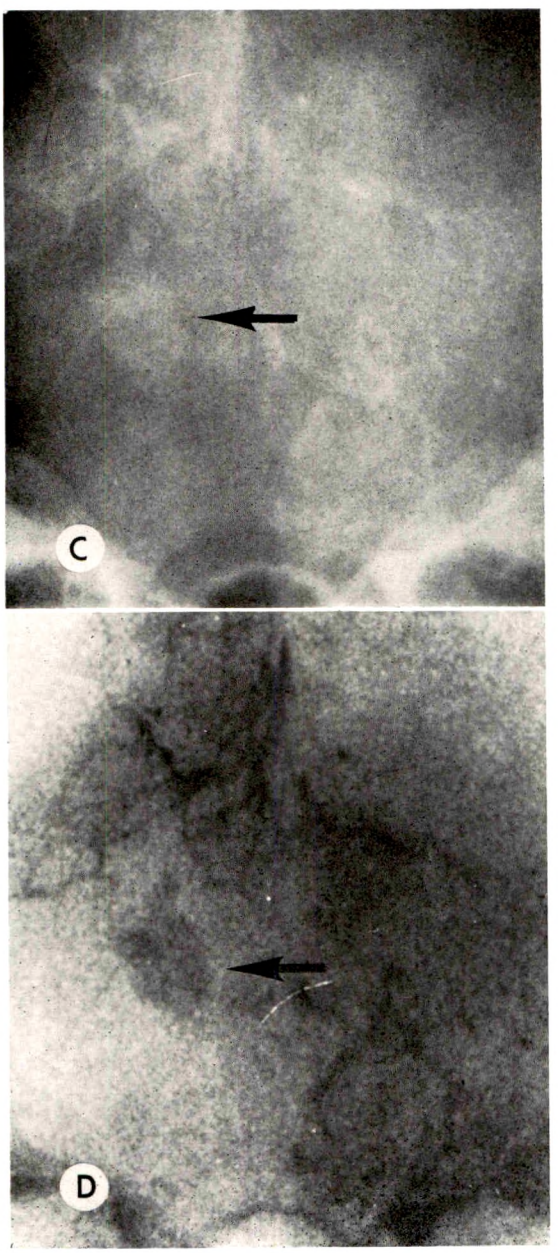


Fig. 2. (*C* and *D*) Right brachial angiograms (routine and subtraction roentgenograms). Vascular nodule in right superior cerebellar hemisphere (arrows).

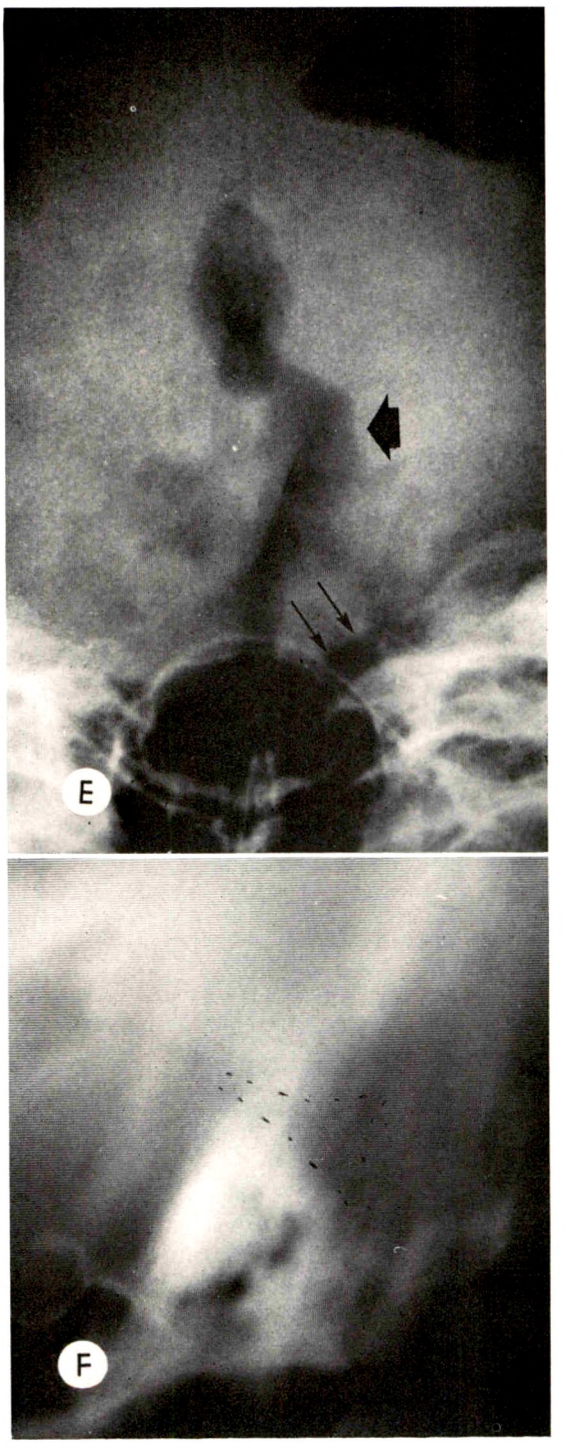


Fig. 2. (*E* and *F*) Pneumoencephalograms. Fourth ventricle displaced and tilted to left (arrow). Air seen in left cerebellopontine angle (small arrows) but not in right. Fourth ventricle not displaced in sagittal plane in lateral projection (retouched).

this appearance. The lymphatic cysts also changed shape with peristalsis and compression. However, this characteristic is also applicable to lipomas because of their fluid consistency[5] at body temperature. The mucosa overlying a lipoma frequently ulcerates,[5] whereas in our series of 7 submucosal lymphatic cysts, the mucosa was intact in each case but did show mild inflammatory changes in 1 instance.

When a filling defect of submucosal origin changes shape on compression, the likely possibilities are a lipoma or a lymphatic cyst. If the lesion is sharply circumscribed and the overlying mucosa is intact, a lymphatic cyst must be a definite consideration in the differential diagnosis. However, the differential diagnosis also includes an isolated submucosal varix[6] and a fat-containing dermoid cyst.[9] Leiomyoma, leiomyosarcoma, and liposarcoma can usually be eliminated from the differential diagnosis because they do not change shape readily and the mucosa overlying these lesions is frequently ulcerated.

SUMMARY

Seven patients with single, submucosal, lymphatic cysts of the gastrointestinal tract were seen at the Mayo Clinic in a 10 year period. In 3 of these cases, the evidence was seen on roentgenographic examination. In the other 4 cases, the lesions were noted incidentally at operation. A comparison of the fluoroscopic and roentgenographic findings in these 3 cases was made with those in 9 cases of proved gastric lipomas.

Each of the lymphatic cysts presented as a sharply defined, rounded, easily compressible submucosal lesion, whereas just over half of the lipomas had this characteristic appearance.

The finding of these roentgenographic features should suggest inclusion of lymphatic cyst in the differential diagnosis.

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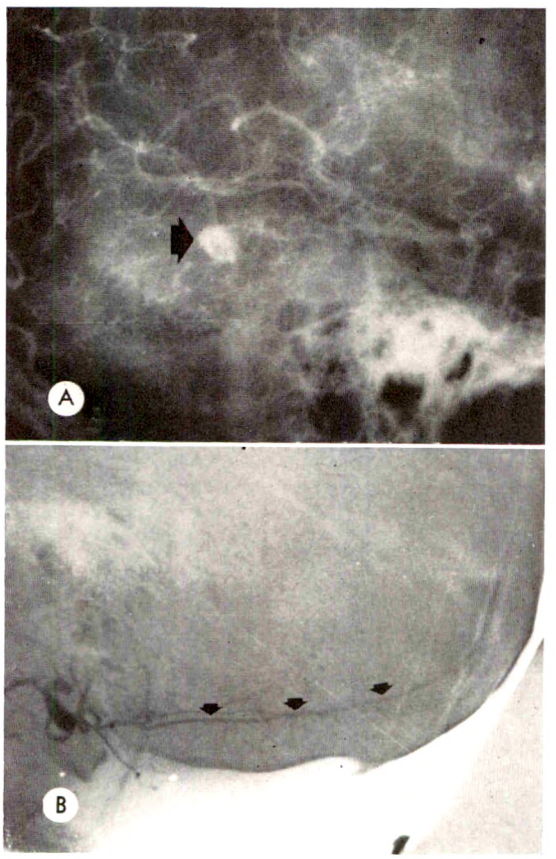


FIG. 3. Case III. (*A*) Right brachial angiogram. Nodular density with translucent center in superior vermis of cerebellum (arrow). (*B*) Left brachial angiogram. Well defined posterior meningeal artery (arrows).

ache progressively worsening for 4 months. The headache was associated with blurred vision and staggering gait. On examination, she had bilateral papilledema, ataxia, and a decreased ankle jerk in the left lower extremity. The hematocrit was normal. Left carotid angiograms (Fig. 5, *A* and *B*) demonstrated reflux of contrast material down the basilar artery to opacify the right superior cerebellar artery which was dilated and was seen to opacify a large fairly homogeneous vascular tumor measuring 35×25×45 mm. in the superior vermis. The tumor was best seen in the late arterial through venous phases. It had an irregular translucent center. The rostral portion of the internal cerebral vein was elevated (Fig. 5*C*). Right brachial angiography (Fig. 5, *D* and *E*) again identified the tumor lying in the superior vermis of the cerebellum. In the venous phase the tumor was seen to elevate the rostral portion of the basal vein of Rosenthal (Fig. 5*F*). The ventricles were dilated.

A suboccipital craniotomy was carried out with the removal of a cerebellar hemangioblastoma which had partially herniated upwards through the tentorial incisura.

CASE VII.† A 57 year old male was admitted for the evaluation of occipital headaches and a staggering gait of 4 weeks' duration. The hematocrit was normal. On neurologic examination abnormal cerebellar signs indicating a right-sided lesion were present. Brachial angiography demonstrated forward displacement of the choroid point of the posterior inferior cerebellar artery. A faint nodule measuring approximately 5×5 mm. was seen in the superior vermis. Pantopaque ventriculography demonstrated large lateral and third ventricles. The proximal aqueduct was dilated and displaced anteriorly and to the left. The fourth ventricle did not fill.

A suboccipital laminectomy was carried out with removal of a right cerebellar cyst containing a mural nodule. Histologic examination revealed a hemangioblastoma.

CASE VIII.† A 59 year old female was admitted complaining of incoordination of her right limbs and falling to the right for a period of 3–4 months. The family history was of considerable importance. Her paternal grandfather had surgical enucleation of 1 eye and her father died of a brain tumor. She had 6 brothers; 1 died of a brain tumor and 1 had 2 daughters, 1 of whom died of a brain tumor and the other had the diagnosis of Lindau von Hippel disease made in 1964. She had 3 sisters, 1 had the diagnosis of Lindau von Hippel disease and had a nephrectomy in 1967. The patient had a left nephrectomy in 1962 for the removal of a renal hemangioblastoma. The patient's son has had an eye enucleated.

The patient demonstrated right-sided cerebellar signs. Selective vertebral angiography demonstrated a large vascular tangle of vessels with a central translucency in the right cerebellar hemisphere (Fig. 6, *A* and *B*). Dilated feeding arteries and draining veins were seen. A second smaller relatively homogeneous nodule with a central translucency was present in the left cerebellar hemisphere.

† Courtesy of Dr. E. Ferris, Department of Radiology, University Hospital, Boston, Massachusetts.

ABDOMINAL LYMPHOGRAPHY: A STUDY WITH SPECIAL REFERENCE TO THE INDIVIDUAL VARIATIONS IN DOSAGE OF CONTRAST MEDIUM*

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OSLO, NORWAY

LYMPHOGRAPHY has been used extensively in diagnostic radiology during recent years. Numerous papers have been published concerning the technique, the yield of the examination, and later, the complications. When studying the literature on the subject, we found that the recommended amount of contrast medium varied considerably.

The purpose of the present investigation was to determine the amount of contrast medium required in a department where the procedure has become a routine study. We observed that there was a great variation in the amount of contrast medium needed to visualize the lymph nodes in the abdomen. For this reason we tried to determine if there was any correlation between the size of the patient, the age, the status of the lymph nodes, and the optimal dose. We also tried to evaluate any possible correlation between the dose given and the roentgenographically demonstrable changes in the lungs the day following the injection. Finally we aimed to find if there was any correlation between the dose and any rise in temperature following the examination.

MATERIAL AND METHOD

One hundred consecutive patients with proven malignant lymphoma, who had undergone lymphography, were included in this study; 69 of the patients were suffering from lymphogranulomatosis, 14 had lymphosarcoma or reticulosarcoma, and the remaining 17 had malignant lymphoma without more distinct classification of the disease. We included technically satisfactory studies only, demonstrated by visible contrast material in the thoracic duct and without evidence of leakage at the site of injection.

The age distribution of the patients is shown in Figure 1. The oldest patient was 81, the youngest 8 years old. The same Figure also shows the distribution of normal (60), and abnormal (40) lymphographic findings in the abdomen at the different age groups.

Thirty-five of the patients were females, 65 were males. Thirteen of the females and 27 of the males had abnormal lymphograms. Six patients had drainage of the thoracic duct performed in connection with the study.

TECHNIQUE

The lymphographies were performed using a modified technique of Kinmonth *et al.*[10] After cannulation of a lymphatic vessel on the dorsum of each foot, lipiodol ultrafluid (ethiodol) was injected at a rate of approximately 6 ml. per foot per hour. The flow of the contrast material then was followed at intervals by the use of an image intensifier. The injection was stopped as soon as contrast medium became visible in the thoracic duct. By then we had also made ajustments if there was any asymmetric filling on the two sides.

Roentgenograms were taken in different projections of the abdomen and chest immediately after completion of the injection and also on the following day.

The amount of contrast material used was recorded in every case with an accuracy of 0.5 ml.

Interpretation of the lymphograms was

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A suboccipital craniotomy was carried out on the right side with removal of a fairly solid vascular tumor with little associated cyst formation.

DISCUSSION

In 1940, Dyke and Davidoff[2] described the pneumoencephalographic appearances of cerebellar hemangioblastomas. They described displacement of the aqueduct, compression and anterior displacement of the fourth ventricle and, in 1 case where the lesion obstructed the fourth ventricle exits, dilatation and upward displacement of the fourth ventricle. The pontine cisterns

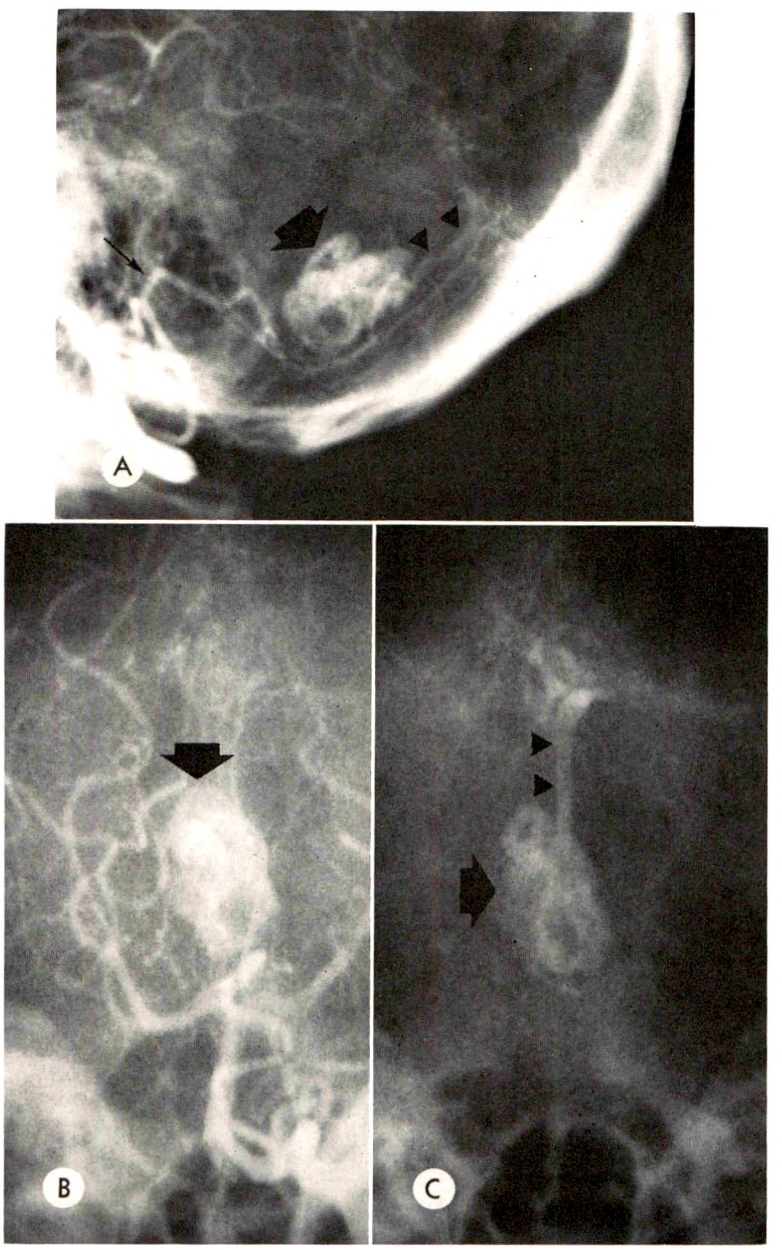


Fig. 4. Case v. (*A–C*) Left brachial angiograms. Choroid point of posterior inferior cerebellar artery displaced anteriorly (small arrow). Tangled mesh of vessels in inferior vermis (large arrow) with draining inferior vermial vein (arrowheads).

done according to the usual criteria, evaluating the increase in size and changes in the pattern of the lymph nodes. Doubtful changes were recorded as negative. Among the positive lymphograms, some showed severe changes with pronounced enlargement of many of the lymph nodes. These were considered a separate group (++), assuming that one here might need more contrast material in order to get satisfactory filling of the lymph nodes (Fig. 2).

The roentgenographic pulmonary changes caused by the injected contrast material seen the day following the study were divided in 3 groups: (0) no visible changes; (+) slight to moderate changes; and (++) pronounced changes.

The temperature was recorded. Fifteen patients were excluded either because of incomplete recording or because the study was performed during a febrile period. A rise in temperature of 0.5°C. or more the morning following the injection was considered as a febrile reaction.

The height of the patient was recorded in all cases in order to be correlated with the amount of contrast material used.

RESULTS

AMOUNT OF CONTRAST MEDIUM NEEDED; ITS RELATION TO THE LYMPHOGRAPHIC CHANGES, HEIGHT AND AGE OF THE PATIENT

The amount of lipiodol ultrafluid needed for satisfactory visualization of the retroperitoneal lymph nodes ranged from 4.5 ml. to 28 ml. per patient (Fig. 3).

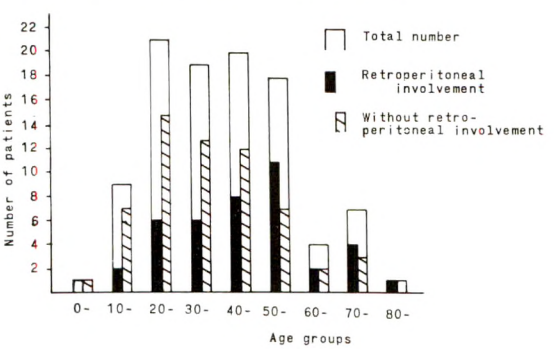


Fig. 1. Lymphography in 100 patients with malignant lymphoma. Distribution of normal and pathologic lymphograms in the different age groups.

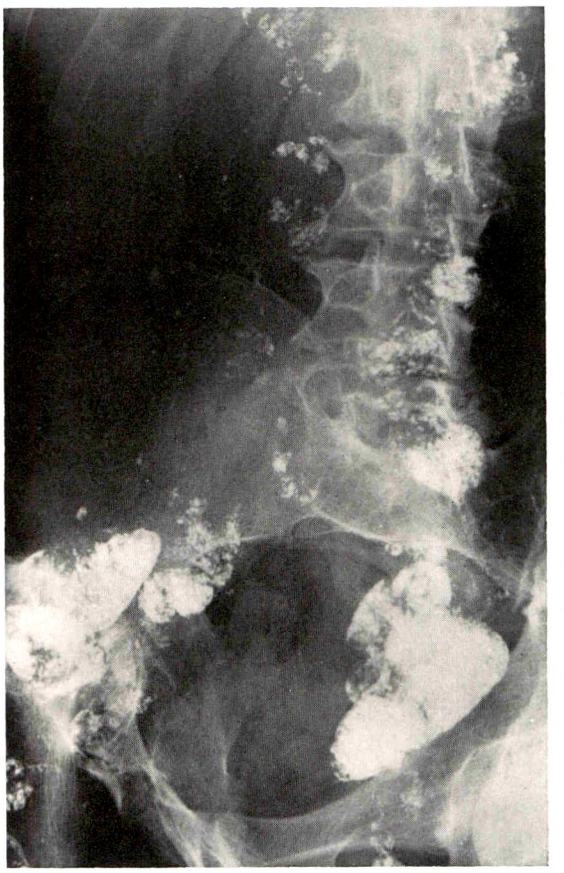


Fig. 2. Oblique lymphogram showing greatly enlarged retroperitoneal lymph nodes caused by lymphomatous involvement. Graded as ++.

Figure 3 also illustrates the relation between status of the lymph nodes and the amount of contrast medium needed. When the lymphograms were considered normal or showed moderate pathologic changes, the mean volume of contrast material used was 16 ml.; 18 ml. was the average dose in patients where several of the lymph nodes were greatly enlarged.

In order to evaluate the relation between the height of the patients and the required amount of contrast material, we calculated ml. of contrast medium per m. of height. As demonstrated in Figure 4, the results showed a great variation, ranging from 4 ml. to 14 ml., with an average of 9.5 ml. per m. height. The value was found to be slightly higher in patients with abnormal lymphograms than in the normals.

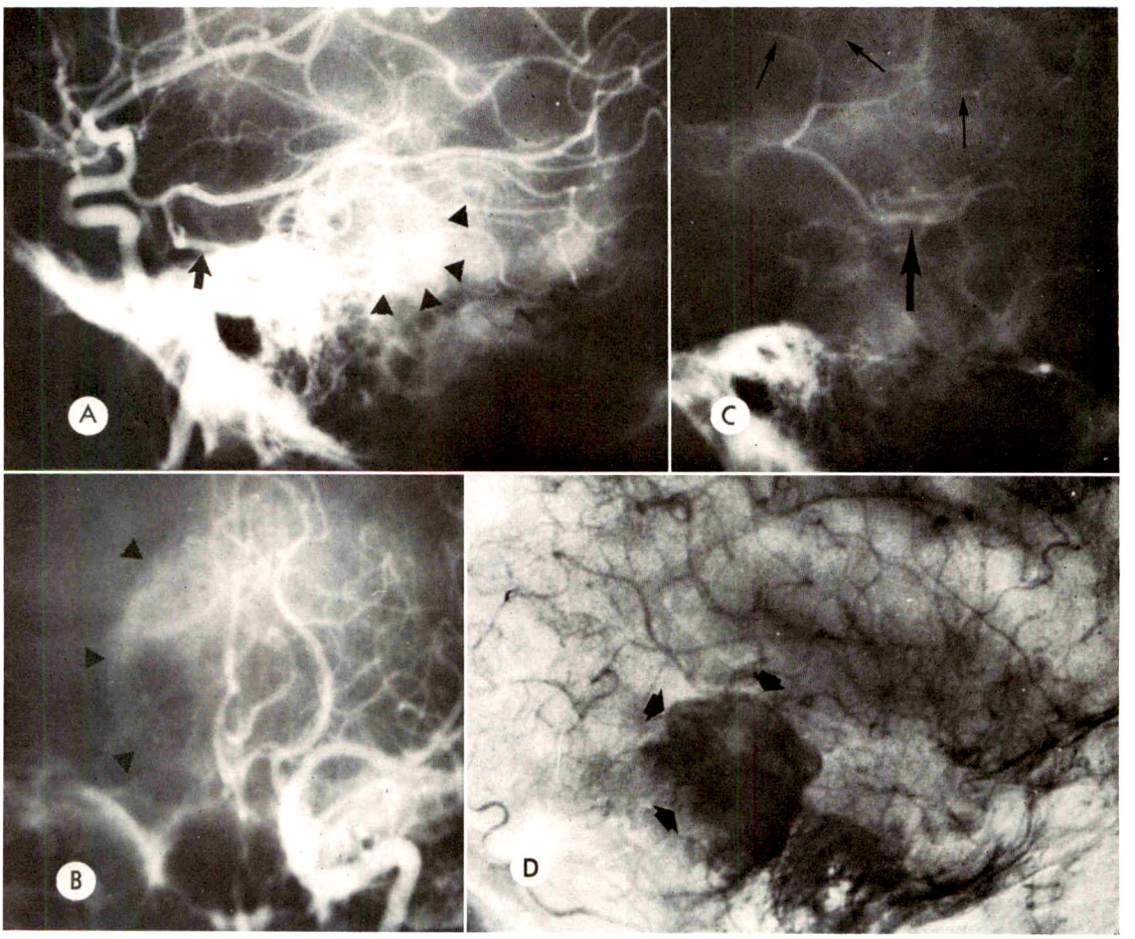


FIG. 5. Case VI. (*A* and *B*) Left carotid angiogram. Reflux of contrast material down basilar artery to opacify dilated right superior cerebellar artery (small arrow) and vascular tumor in superior cerebellum (arrowheads). (*C*) Left carotid angiogram (venous phase). Elevation of rostral portion of internal cerebral vein (arrow) due to upward herniation of tumor through tentorial incisura. Note stretching of subependymal veins of lateral ventricle (small arrows) indicative of lateral ventricle dilatation. (*D*) Right brachial angiogram. Vascular tumor with central translucencies well seen (arrows).

were narrow, and lateral ventricular dilatation which was often asymmetric was also seen.

Radner[13] in 1951 and Sjögren[14] in 1953, discussed the value of angiography in the diagnosis of cerebellar hemangioblastomas and found that the hypervascularized part of the growth was angiographically seen in the majority of cases. Sjögren mentioned that the mural nodule in a cyst tumor was the only part of the tumor seen. The cystic component was determined by vessel displacement.

Ollson[11] in 1953, assessed the appearances on vertebral angiography. He described 6 cases and mentioned that in 3 cases a small nonhomogeneous network of entangled vessels of varying size was seen, in 1 case a homogeneous stain and in 1 case tortuous vessels in ring formation were noted. In 1 case, 2 separate angiomas were found apparently with a similar pattern to the first type described. Taveras and Wood[16] in 1964, described 3 different types of vascular patterns: (a) a mural nodule within a cyst sometimes associated with draining veins; (b) a circular arrangement of vessels surrounding a clear cystic space; and (c) a large mass with many abnormal vessels and distinct draining veins.

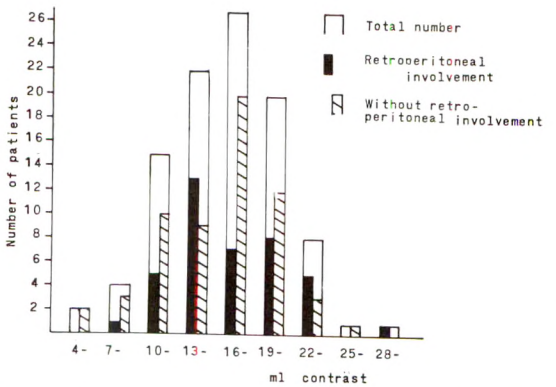


FIG. 3. Lymphography in 100 patients with malignant lymphoma showing the amount (ml.) of contrast medium used. In all patients the injection was stopped when the contrast material became visible in the thoracic duct.

In order to evaluate if there was any relation between the age of the patients and the amount of contrast material needed, we divided our patient material into decade groups. In the groups between 20 and 80 years, the average dose showed only slight variation, ranging from 16 ml. to 17.5 ml. The patients with an age below 20 years needed an average dose of 15.5 ml. except for 3 patients aged below 15, who needed only 6 ml.

PULMONARY CHANGES CAUSED BY OIL EMBOLISM; THEIR RELATION TO THE CONTRAST MEDIUM VOLUME AND THE STATE OF THE RETROPERITONEAL LYMPH NODES

Chest roentgenograms obtained the day following the injection showed pulmonary changes, referable to oil embolism, in 86 patients. In 16 cases these changes were graded as ++ (Fig. 5*B*). Five of the patients with normal chest roentgenograms had drainage of the thoracic duct performed during the injection of the contrast medium. The dose of contrast material given was not significantly larger in patients with chest roentgenograms graded as ++ than in patients with normal chest roentgenograms.

RELATION BETWEEN ELEVATION OF TEMPERATURE, PULMONARY CHANGES AND LYMPH NODE APPEARANCES

Of 85 patients with a satisfactory recording of temperature, 55 patients had unchanged temperature the day following injection of the contrast medium. (The patients with thoracic duct cannulation are not included.) In 9 patients only, the rise of temperature was 1°C. or more. In the 30 patients with an increase of temperature of at least 0.5°C., the temperature reaction lasted for 1 day only in 24 of the cases. Four of the patients had a raised temperature for 3 days or more.

There was no significant correlation between elevated temperature and the groups with and without roentgenographic evidence of retroperitoneal lymph node involvement. The number of patients with a raised temperature was slightly higher in the group with roentgenographically demonstrable pulmonary oil emboli than in the other (Table 1).

DISCUSSION

An ideal lymphogram is obtained when the retroperitoneal lymph nodes are satisfactorily filled and the amount of contrast material escaping to the lungs is kept as low as possible. In the early period of lymphography, there was a tendency to use larger doses than is customary today. Doses up to 30 ml. or even more were recommended.[5] Serious pulmonary complications were noted,[1,3,4,8,9,13,15] and during later years there has been a tendency to reduce the doses. Some authors[2,4] recommend a stan-

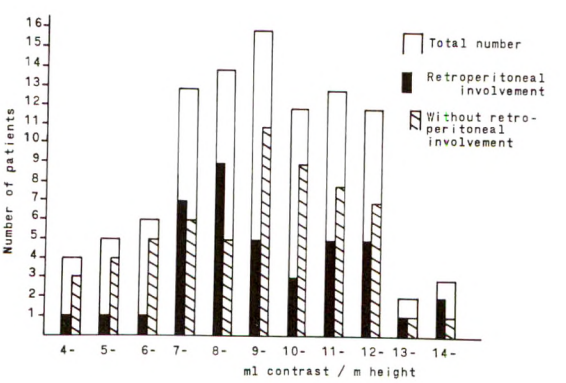


FIG. 4. Lymphography in 100 patients with malignant lymphoma showing the amount of contrast medium used per height (ml./m.) of the patient.

Krayenbühl and Yaşargil[6] in 1968 described the angiographic findings in 62 infratentorial hemangioblastomas. They described 3 different types: (a) expanding cystic tumor without a vascularized nodule; (b) solid vascular tumor of varying size with an enlarged feeding artery and occasionally with draining veins; and (c) a large vascular mass with large abnormal vessels.

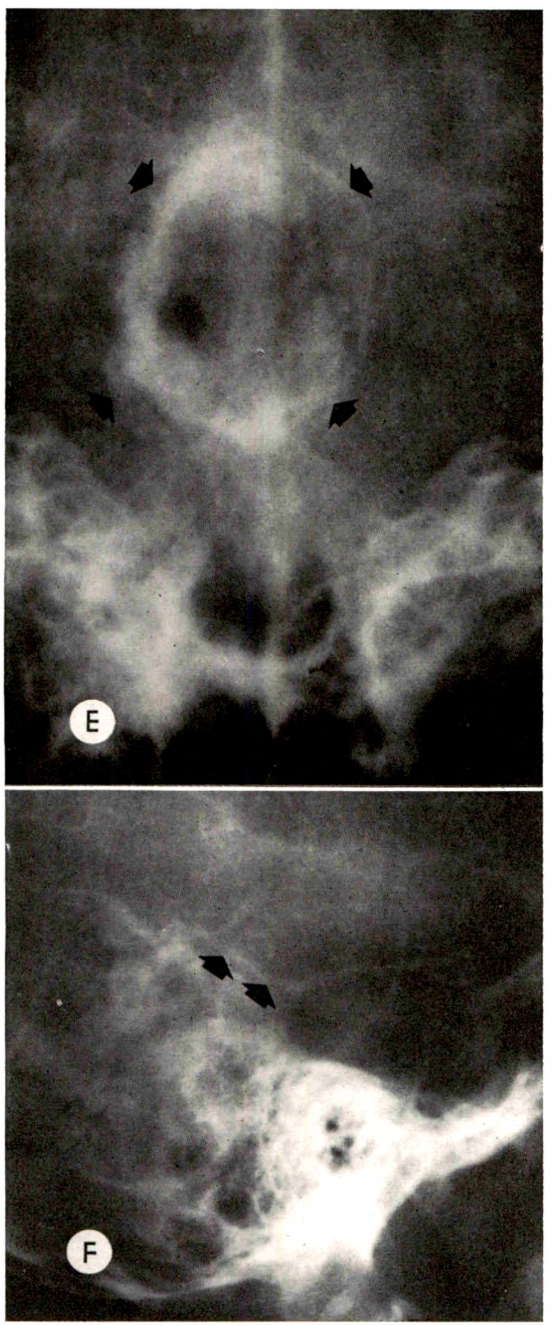


Fig. 5. (*E* and *F*) Right brachial angiograms (venous phase). (*E*) Large midline tumor with central translucencies (arrows). (*F*) Elevation of rostral portion of basal vein of Rosenthal (arrows).

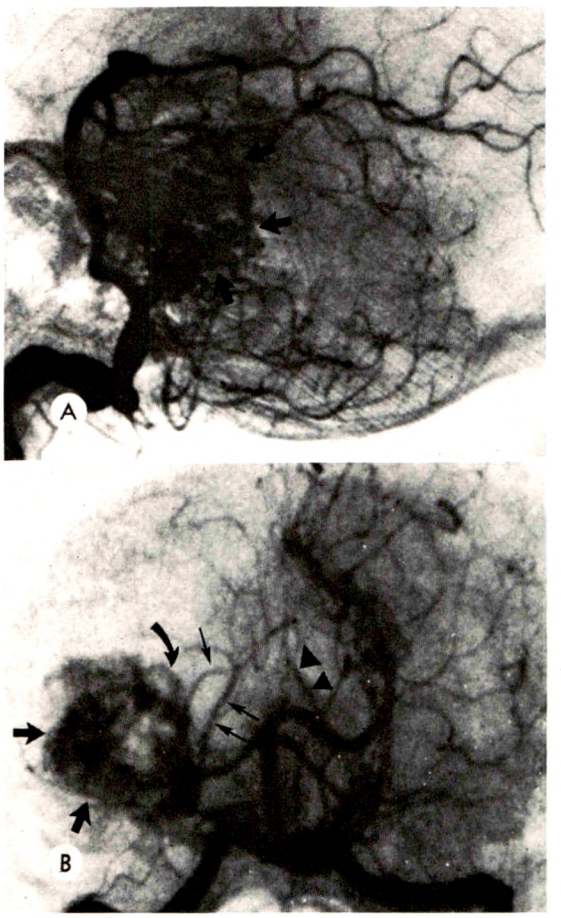


Fig. 6. Case VIII. (*A*) Left vertebral angiogram. Vascular tangle of vessels in superior lobe of cerebellum with some central translucencies (arrows). (*B*) Left vertebral angiogram (frontal projection). Vascular tangle of vessels (large arrows) supplied by right superior cerebellar artery (small arrows). Vermial branch of right posterior inferior cerebellar artery displaced to left (arrowheads). Early draining vein seen on superomedial border of lesion (curved arrow) confirmed on later venous angiograms.

An analysis of our 8 cases is seen in Table 1. In 6 of the 8 cases, the lesions consisted of nodules of varying size and density. In the remaining 2 cases (Cases V and VIII) the lesion consisted of a non-

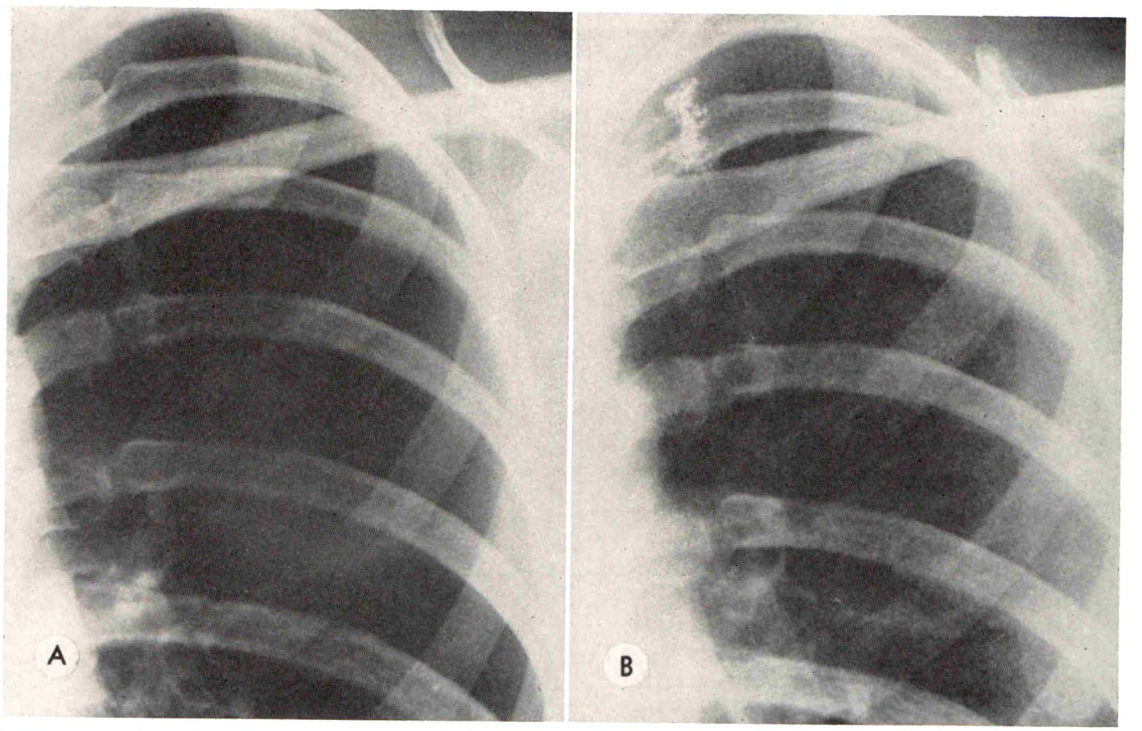


Fig. 5. (*A*) Normal chest roentgenogram before injection of contrast medium. (*B*) Generalized, small densities in the lungs the day following the injection.

dard dose given in adult patients. Realizing the great individual variations, others recommend a more individual dosage considering mainly the age and height of the patients.[7,16,17] Some prefer roentgenograms of the abdomen taken during injection of the contrast medium, and others recommend monitoring by use of an image intensifier to evaluate when a satisfactory dose has been given.[3,6,13]

Dosage of Contrast Medium

Figure 3 illustrates the large individual variations in amount of contrast medium needed in our series. When correlated to the state of the lymph nodes, we found a slightly increased volume in patients with severely affected lymph nodes. Compared with the individuals where the lymphograms were normal or moderately pathologic, the difference in average volume, however, was only 2 ml.

We expected, as has also been stated by several authors, that the height of the patients would influence the contrast material volume.[7,16] As shown in Figure 4, however, there was a great variation in the needed amount of contrast material per meter. Therefore, the height of the patients can not be used as a safe guide in predicting the dose. Gerteis and Greuel[7] stated that older patients needed less contrast material

Table I

Correlation between pulmonary changes referable to the injected contrast material and the rise of temperature

	Temperature		
	Not Raised	Raised	Total
No evidence of pulmonary oil embolism	6	2 (25%)	8
Roentgenologic evidence of pulmonary oil embolism	49	28 (36%)	77

TABLE I
SUMMARY OF CASES

Case No.	No. of Lesions	Dense Nodule	Tangle of Vessels	Translucent Center	Associated Vessel Displacement	Posterior Meningeal Artery	Value of Subtraction	Enlarged Feeding Arteries	Draining Veins	Ventricular Displacement	Operative Findings
I	2	+ +		+ +	Downward and lateral displacement of P.I.C.A.* and its branches	−	++			Fourth ventricle and aqueduct displaced anteriorly and to the left	Cyst with small vascular tumor in right cerebellar hemisphere
II	1	+			Choroid point of P.I.C.A. displaced anteriorly	+	++			Fourth ventricle displaced to the left	Large cyst with small nodule in cerebellar hemisphere
III	1	+		+	Downward displacement of P.I.C.A.	+			+	Anterior displacement of fourth ventricle and aqueduct	Cyst with nodule in superior aspect of cerebellum in the midline
IV	1	+		+	P.I.C.A. not seen on bilateral angiograms	+				No studies	Small cyst with nodule in cerebellar hemisphere
V	1		+	+	Choroid point of P.I.C.A. displaced anteriorly	−		Vermial branch of P.I.C.A.	+	No studies	Solid tumor in inferior vermis
VI	1	+		+	Rostral portion of internal cerebral vein elevated; basal vein of Rosenthal displaced upwards	−	+	Right superior cerebellar artery		Displacement of aqueduct forward and to the left	Superior vermis tumor with cyst
VII	1	+			Choroid point of P.I.C.A. displaced anteriorly	−	+			Displacement of aqueduct forward and to the left	Large cyst with small nodule in right cerebellar hemisphere
VIII	2	 +	+	+ +	Displacement of superior cerebellar veins; lateral displacement of vermial branches of P.I.C.A.	−	+	Right superior cerebellar artery	+	No studies	Solid tumor with small cystic component

* P.I.C.A. = posterior inferior cerebellar artery.

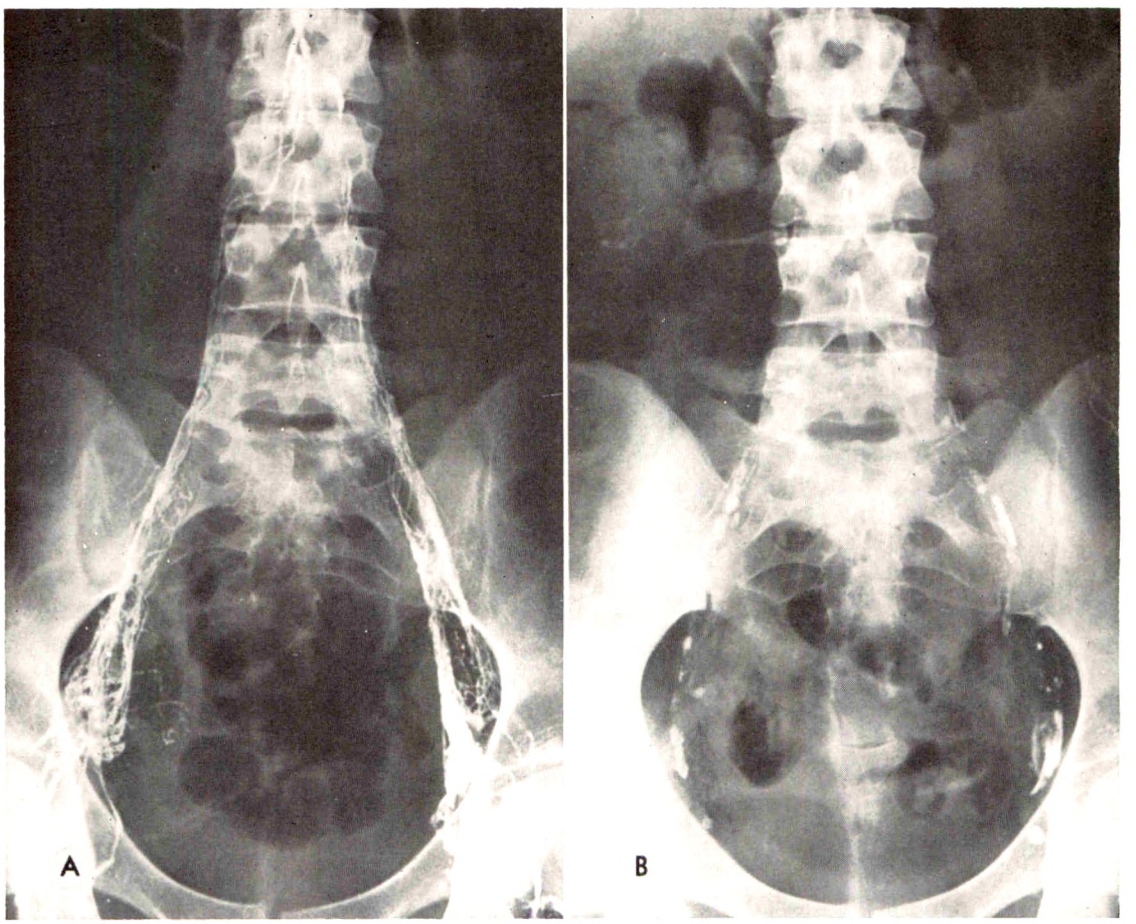


Fig. 6. Small lymph nodes in a patient who had received chemotherapy for 4 years prior to the study. In this patient contrast medium was seen in the thoracic duct after injection of 7.5 ml. of contrast medium only. (*A*) Lymphogram obtained the day of injection. (*B*) Lymphogram obtained the following day.

than younger ones. They explain that this is due to involutionary changes of the lymphatic system, causing a reduction in the number and size of the lymph nodes. Our study did not support this. We did not find any reduction in the volume of contrast medium needed in older patients compared to younger age groups. The 3 children in our series are of special interest. In all of them a contrast medium volume of only 4 ml. per m. height was needed, while in the other age groups the mean volume was 9 ml. per m. height. Of special interest also is a patient who had received chemotherapy for 4 years. In this patient, a satisfactory lymphogram, with contrast material visible in the thoracic duct, was obtained with a dose of 4 ml. per m. height, or a total of 7.5 ml. In this case the lymph nodes were small and gave an impression of atrophy, possibly due to the long-lasting chemotherapy (Fig. 6, *A* and *B*). This may be of practical importance. In the future one may expect to get an increasing number of similar patients as the result of improved therapy. None of our patients had received radiotherapy prior to the examination. Such therapy also may reduce the size of the lymph nodes, and care must then be taken to restrict the dose as stressed by Macdonald.[12]

Pulmonary changes caused by oil droplets

The oily contrast medium, entering the pulmonary circulation via the thoracic duct

homogeneous network of tangled vessels fed by a dilated artery. In Cases I and VIII the lesions were multiple. In Cases III, V and VIII dilated draining veins were present.

In 6 of the 8 cases translucent areas were present in the center of the lesions. Large cystic components to the tumor were found in 6 cases. In Case V, where angiography demonstrated a solid tumor with little associated vessel displacement, no cyst was found at surgery. The presence of a cyst was suggested from the angiograms by a greater displacement of the vessels than would be suggested by the size of the opacified lesion. The displacement of the posterior inferior cerebellar artery in 5 cases and absence of visualization of the artery in another were associated with significant cystic components to the lesion. Local anterior displacement of the choroid loop of the posterior inferior cerebellar artery was seen in the 1 case without the cyst. Abnormalities of the posterior inferior cerebellar artery were thus seen in 7 cases, signifying the vessel's importance in angiography of the posterior fossa.

The posterior meningeal branch of the vertebral artery was visualized in 3 cases. This vessel which may be seen in 20–30 per cent of normal patients is enlarged in dural arteriovenous malformations of the posterior fossa or in tumors invading the falx cerebelli or adjacent dura and in cases of hemangioblastomas.[4,9] It is difficult to conclude from the 3 cases of this study in which the arteries were identified that the vessels were enlarged in response to the tumor, as the arteries did not appear specifically to extend to the site of the hemangioblastoma nodules.

The value of subtraction studies was apparent in all cases in which it was obtained. Subtraction was particularly valuable in Case I, where in the frontal view the lesion was partially "lost" in the petrous bone, and in Case II, where the lesion was not seen until subtraction studies were obtained.

Air and pantopaque ventriculography helped in the anatomic elucidation of the site of the lesions, but was otherwise nonspecific.

Cerebellar hemangioblastomas can be associated with hypernephroma and polycythemia.[7] In the Lindau von Hippel syndrome one may see retinal hemangiomas, pancreatic and renal cysts as well as renal and adrenal tumors. For these reasons, a roentgenogram of the abdomen should always be obtained following cerebral angiography to assess gross renal function and morphology. Hemangioblastomas with angiographic abnormalities have also been described in the supratentorial region[5,6,8] and in the spinal cord.[1,3,15]

SUMMARY AND CONCLUSIONS

A series of cases of cerebellar hemangioblastomas investigated by angiography and in some cases encephalography and ventriculography is presented. The encephalographic appearances are nonspecific and only indicated the presence of a mass lesion in the cerebellum.

The angiographic appearances are specific and the following classification is suggested: *A. Dense nodule*: 1. homogeneous, 2. central translucency; *B. Tangle of vessels*: 1. homogeneous, 2. central translucency.

Enlarged feeding arteries and draining veins may be seen with either type. Cysts are often associated and cause appropriate vessel displacement. Cysts without angiographically visible vascular lesions may occur. The posterior meningeal artery may be enlarged in response to the vascular tumor. The lesions may be multiple.

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and eventually other lymphaticovenous communications, is trapped in the lungs, causing small emboli. This will produce changes usually detectable on chest roentgenograms taken the day after the injection. The more oil reaching the lungs, the more pronounced the changes will be, as demonstrated on the roentgenograms. It has been shown experimentally in dogs that 50 per cent of the visible oil in the lungs disappeared within 2 or 3 days. After 3 weeks only 10 per cent was left.[15] The oil emboli reduce the diffusion capacity for carbon monoxide from 10 to 60 per cent, usually approximately 25 per cent.[6] Maximum reduction came about 2 hours after injection. The diffusion capacity was still reduced after 24 hours, but was back to normal values after 48 hours. In patients who have normal pulmonary function, this reduction will cause no harm and usually they will have no symptoms. The more the pulmonary function is reduced prior to the injection, the more serious this added reduction will be to the patient. Even fatal cases have been reported,[9] and at least some of these have probably been caused by too large a volume of contrast material.

In all patients in the present series, the injection was stopped when the contrast material had reached the same anatomic level—the thoracic duct. We therefore thought that the amount of oil entering the lungs should be approximately the same in all patients. This, however, was not the case. In our material, 9 patients did not have detectable pulmonary changes caused by oil emboli. This number included 2 children, where the injected volume was so small that one could not expect any visible changes.

In 16 patients the chest roentgenograms revealed pronounced changes caused by the contrast material. Fifteen of them had normal lymphograms. Among the 7 adult patients without visible oil droplets in the lungs, 6 had abnormal lymphograms with lymphomatous involvement of the retroperitoneal lymph nodes. It therefore seemed that pathologic lymph nodes retained more of the contrast material which was in the lymphatics when the injection was stopped, and thereby in a way protected the lungs. We have not seen this mentioned in the literature previously.

TEMPERATURE REACTION

A moderately increased temperature in the evening the day of injection was often seen. This seemed to do no harm to the patient. We recorded the temperature the days following the injection in order to determine if this perhaps could reveal those patients who got more pronounced reaction to the examination. Thirty-five per cent of the patients got some fever reaction, in most of them this lasted for 1 day only. Four patients had a raised temperature for 3 days or more. In these patients there was no evidence of pneumonia or local infection. Of 6 patients who had their thoracic duct drained during the injection, 5 did not show any pulmonary oil droplets or pyrexia. The remaining 1 showed evidence of oil embolism, despite cannulation of the thoracic duct. In this case there was elevation of temperature lasting for 1 day. The incidence of rise in temperature was slightly higher in the group of patients with roentgenographically demonstrable pulmonary oil embolism than in the others (Table 1). These observations could point towards the lungs as the releasing site of fever, possibly some sort of chemical pneumonitis.

We did not find any correlation between raised temperature and the state of the retroperitoneal lymph nodes.

SUMMARY AND CONCLUSION

An ideal lymphogram is obtained when the retroperitoneal lymph nodes are satisfactorily filled up to the level of the chylous cistern, and when the contrast material volume escaping to the lungs is kept at a minimum.

The needed amount of contrast medium showed great variation in our series of 100 patients. Three patients aged below 15 years needed very small amounts. This was also the case in a patient who had previously received chemotherapy for several

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years. In the remaining 96 patients the dose varied from 9.5 ml. to 28 ml. In these, the contrast medium volume did not show any relation either to the age or height of the patients. In cases with widespread infiltration of the retroperitoneal lymph nodes, the average dose of contrast medium needed was found to be slightly larger than in patients with normal or moderately abnormal lymphograms.

Widespread infiltration of the lymph nodes in a way seemed to protect the lungs from oil embolism. This is probably due to a greater storing capacity of the lymph nodes of the contrast material still in the lymphatics when the injection is stopped.

Temperature reaction of some duration was unusual in our material. Five patients, who had their thoracic ducts cannulated and where no oil was seen in the lungs, did not show any temperature reaction. Another patient also drained, but nevertheless with visible oil in the lungs, had fever reaction. This could suggest that the pyrexia may have some relation to the oil trapped in the lungs.

Considering the great variation in the different patients, it seems impossible to predict the desired dose of contrast medium. The only way to obtain a correct estimate is by following the contrast column with fluoroscopy at intervals, preferably by the use of an image intensifier. The injection should be stopped as soon as contrast material is seen in the thoracic duct; then the pulmonary changes caused by emboli will usually be moderate, and in our material we did not observe any serious pulmonary complications.

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INTRACRANIAL SOLITARY CHONDROMA OF DURAL ORIGIN*

By ENRIQUE PALACIOS, M.D.†
KANSAS CITY, KANSAS

INTRACRANIAL cartilaginous tumors are uncommon. Scattered reports would indicate that these tumors can be classified into 4 groups: (1) there are those tumors which arise from the base of the skull, especially in the spheno-occipital region;[4,7,8,11] (2) this group comprises lesions originating from the paranasal sinuses and extending into the cranial cavity;[10] (3) in the third group are those cartilaginous lesions arising from the choroid plexuses,[5] the genesis of which is poorly understood; and (4) this group includes tumors which have a dural attachment.[1,2,5,13]

This paper documents a case of the latter group, an intracranial chondroma of dural origin which revealed unusual radiographic and surgical features.

Alpers[1] reviewed the literature and found 7 cartilaginous lesions located over the cerebral hemispheres and added 1 of his own. Six of these cases were pure chondromas and 2 were osteochondromas. Two of the chondromas showed small calcification on the plain roentgenograms, while the osteochondromas appeared almost entirely as calcified masses. In one instance, there was hyperostosis of the adjacent bone which surgically revealed no tumor invasion. Five of these 8 cases were located in the parietal region, 2 in the frontal and 1 in the frontoparietal region. All the lesions were encapsulated and had a firm dural attachment. Verbrugghen and Learmonth[13] have reported a chondroma of the right parietal region, attached to the falx cerebri. The patient had a history of jacksonian convulsions. Forsythe *et al.*[5] described a case of a 51 year old woman whose plain roentgenograms of the skull demonstrated a large calcified mass in the posterior parietal area. The patient had a history of periodic headaches of 10 years' duration, occasional nausea, tinnitus, postural vertigo, and a slight degree of tremor. The diagnostic dilemma was whether this mass was a meningioma or a calcified hematoma. Surgically, a large osteochondroma with dural attachment was removed with complete clinical recovery.

REPORT OF A CASE

A 30 year old, right-handed, white female was admitted to the University of Kansas Medical Center for evaluation of a seizure disorder of 1 year's duration. The patient had had 5 nocturnal grand mal seizures without any significant postictal state. She was seen by a private physician 1 month prior to admission, at which time a roentgenographic study of the skull revealed an area of hyperostosis in the left parasagittal region. An electroencephalogram was done and was interpreted as being abnormal. The only disability that the patient could recall was some speech difficulty.

The past history, family history, and review of systems were noncontributory. Except for obesity, the general physical examination was

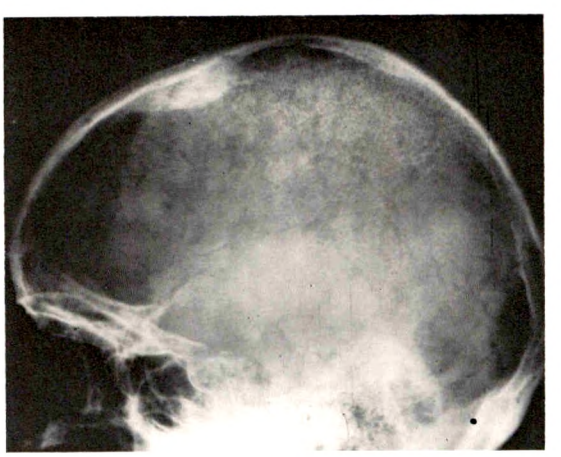


FIG. 1. Skull roentgenogram demonstrating hyperostosis of the left frontoparietal region.

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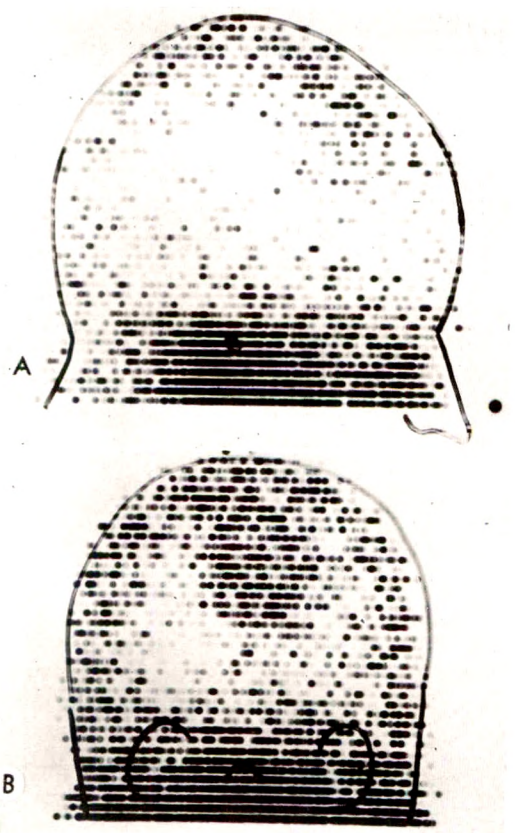


Fig. 2. (*A* and *B*) Radioactive scans showing an abnormal uptake in the left frontoparietal region crossing the midline.

unremarkable. The neurologic examination was within normal limits. There was no evidence of papilledema.

Routine laboratory examinations were unremarkable. An electroencephalogram showed focal slow waves in the left frontoparietal area. Skull roentgenograms demonstrated an area of hyperostosis in the left frontoparietal region (Fig. 1). Radioactive brain scans revealed an abnormal uptake and some activity was seen to cross the midline (Fig. 2, *A* and *B*). A left carotid arteriogram demonstrated a large, avascular, parasagittal mass just to the left of the midline. There was marked displacement of the midline structures to the right. Pathologic vessels or tumor stain were not identified. Meningeal arteries did not appear to supply the tumor (Fig. 3, *A* and *B*). Despite the lack of demonstrable supply by meningeal vessels, the preoperative diagnosis was a meningioma.

A bifrontal craniotomy was performed. When the bone flap from the left parasagittal area was elevated, a tumor could be palpated, just in front of the coronal suture. It was exceedingly hard and appeared to be attached to the bone itself. The center of the surface of the bone flap looked like the hyperostosis of a meningioma, yet it was avascular. The dura had been involved by the pedicle of the tumor. The dural incision revealed a 10 cm. in greatest diameter, yellowish-gray, glistening, hard tumor which could be separated and removed by blunt dissection without sectioning the sagittal sinus. The mass lay in the angle between the superior sagittal sinus, the falx cerebri and the convexity dura. The tumor was not attached to the brain, and there were no vessels penetrating it from the brain. It had compressed the left frontal lobe over to the right. The lesion was completely

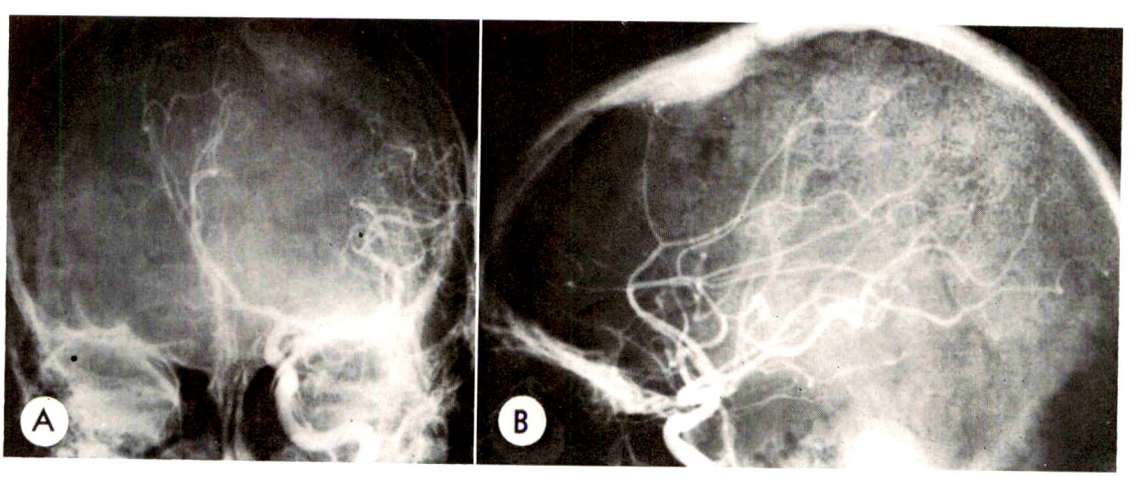


Fig. 3. (*A*) Anteroposterior and (*B*) lateral roentgenograms of carotid arteriography, revealing a large avascular tumor in the left frontoparietal region, which crosses the midline. No meningeal arterial supply to the tumor is detected.

EDITORIAL

THE SEVENTY-FIRST ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Seventy-first Annual Meeting of the American Roentgen Ray Society was held at the Deauville Hotel in Miami Beach, Florida, September 29–October 2, 1970. This is the second time that the Society has met in this famous resort city.

President James C. Cook officially called the meeting to order at 8:30 A.M., Tuesday, September 29th and, as his last function, installed, with the assistance of Dr. William B. Seaman, of New York City, Chairman of the Executive Council, President-Elect John F. Roach as the new President of the Society. As part of this function, he presented to Dr. Roach the symbolic Pfahler gavel, fashioned from a mastodon's tooth recovered from Alaskan glacial ice, and the working Erskine gavel made from memorabilia of Röntgen, Snook and Coolidge. Concluding the ceremony, outgoing President Cook pinned on Dr. John F. Roach the gold emblem of the Society engraved "President of the American Roentgen Ray Society."

President Roach, as his first function, remitted to Dr. James C. Cook the Past Presidents' Book, inscribed with his name and containing a list of all Past Presidents, together with beautiful color photographs of the historic Pfahler and Erskine gavels. Immediately thereafter, he gave the "Presidential Address," which is published *in extenso* as the leading article in this issue of the JOURNAL.

In his Presidential Address, Dr. Roach, after emphasizing the Society's sponsorship of its official organ, THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE, discussed the 6 routes which the American Roentgen Ray Society takes to fulfill its purpose crystallized in Article II of the Constitution, which states that "the object shall be the advancement of medicine through the science of radiology." These 6 routes are: (1) the Scientific Sessions, covering many aspects of diagnosis, therapy, nuclear medicine and physics; (2) the "Refresher Courses"–incorrectly named, said President Roach, "because not only will you be refreshed, but you will also be introduced to new and stimulating information and ideas;" (3) the Scientific Exhibits; (4) the Technical Exhibits; (5) the Caldwell Lecture; and (6) the discussions in small informal groups sitting around the lunch table, the hotel room, the swimming pool or some other similar place.

President Roach concluded: "The pearls will soon drop from all six sides. Don't let them pass you by. May you have an enjoyable and educational experience in their gathering."

The First Scientific Session began at 8:45 A.M. Tuesday with President John F. Roach in the chair. Thirteen papers were read at this session on various timely and most instructive subjects. These included: 1 paper of the roentgenologic demonstration of tiny amounts of intraperitoneal gas; 2 papers on gallbladder topics: 4 papers on vascular and renovascular conditions; 5 papers of oto-orbito-neurologic interest and Paper 13, which was the *Award Paper of the American Society of Neuroradiology*. This last paper was co-authored by Drs. Stephen A. Kieffer, John M. Wolff, Walter B. Prentice and Merle K. Loken, Minneapolis, Minnesota, and its title was: "Scinticisternography, in Individuals Without Known Neurological Disease."

encapsulated, avascular and looked and felt like a big lobulated cartilaginous mass (Fig. 4). The area of hyperostosis in the bone flap was drilled to the diploe, revealing no evidence of bone invasion.

After removal of the tumor, the incisions were closed. A dural graft was placed in the area of the tumor attachment. The neurosurgeons were confident that all of the tumor was removed and that there was no damage to the adjacent brain. The final histopathologic diagnosis was chondroma (Fig. 5). Postoperatively the patient recovered completely.

DISCUSSION

The bones of the base of the skull develop from cartilaginous tissues; thus, intracranial chondromas and osteochondromas occurring from the base of the skull or paranasal sinuses are not surprising. On the other hand, the cranial vault develops from membranous tissues, and chondromas arising from these noncartilaginous structures require some explanation of their origin. Metaplastic meningeal fibroblasts have the potential for changing into chondroblasts. Some meningiomas may duplicate all the structures derived from the primitive meninx which is already in an advanced stage of differentiation. They may contain bone, traceable to the periosteal component of the "skeletoneural intertissue."[6] In a study of 121 meningiomas, Lapresle *et al.*[9] classify meningiomas by their histologic appearance and consider osseous and chondromatous tissues as secondary components of one of their groups.

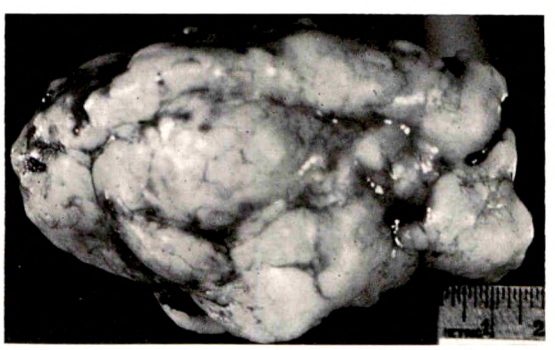


Fig. 4. Gross specimen of the encapsulated, avascular, cartilaginous mass.



Fig. 5. Microscopic section of the lesion demonstrating pure chondromatous tissue.

The surgical and radiographic information from this case would indicate that this chondroma originated from the dura mater. The tumor probably developed at a very slow rate for many years, pressing against the left cerebral hemisphere, producing minimal symptoms. These cartilaginous lesions are avascular, compress the brain, but do not invade it. They are similar to cartilaginous lesions in other parts of the body.

Despite the lack of meningeal arterial supply to the tumor as shown angiographically, the preoperative diagnosis was meningioma. This tumor produced hyperostosis of the inner table of the skull with roentgenographic characteristics indistinguishable from that of a parasagittal meningioma. The association of hyperostosis and meningiomas (25 per cent) is well known. Cushing[3] believed that meningiomas invade adjacent bone, producing osteoblastic activity and formation of new bone. Penfield[12] described the presence of meningiomatous cells within the involved bone, believing that the new bone was reactive in nature and not an integral part of the tumor; however, in cases of "en plaque" meningiomas one may find considerable hyperostosis without invasion of the bone by tumor. In the case recorded here, the hyperostotic area revealed no evidence of tumor cells.

Normal cartilage could be produced with the formation of chondromas or osteochondromas attached to the dura mater. This explanation gains support when it is

On Tuesday at 8:30 P.M. the Caldwell Lecture was given by Russell H. Morgan, M.D., Professor of Radiology, Johns Hopkins School of Medicine; and Radiologist-in-Chief, Johns Hopkins Hospital, Baltimore, Maryland. The title of his Lecture was "The Emergence of Radiology As a Major Influence on American Medicine." Doctor Morgan, who is one of the greatest scientists Radiology has ever produced, gave a brilliant Lecture, masterfully conceived and authoritatively documented.

Dr. Morgan was introduced by his illustrious friend and disciple Dr. Robert D. Moseley, Professor of Radiology, and Chairman, Department of Radiology, University of Chicago, Chicago, Illinois.

At the conclusion of the Lecture, President Roach awarded to Dr. Morgan the Caldwell Gold Medal, the highest recognition of the Society "for meritorious scientific achievement."

The Second Scientific Session began Wednesday, September 30, at 8:30 A.M. with Dr. Glenn E. Sheline, San Francisco, California, First Vice-President of the Society in the Chair. At this session again 13 papers were delivered on various subjects. Among them were: 1 paper on the vector principle in the differential diagnosis of abdominal masses in the left upper quadrant; 1 paper on inefficient use of x rays in diagnostic radiology, another on beam quality measurements in diagnostic roentgenology; 1 paper on innovations in angiography; 1 paper on a new biplane angiographic system; and 7 papers on practical clinical radiologic topics. Paper 26 was the *Award Paper of the Society for Pediatric Radiology* titled: "Plain Skull Roentgenograms in Head Trauma of Children," by Fred Roberts, M.D., and Charles E. Shopfner, M.D., Kansas City, Missouri. In this paper the authors stated that roentgen study of the skull is a common procedure in the evaluation of head trauma in children mainly because of possible "medicolegal" implications, although detection of a fracture had no significance in the clinical management when compared to a control group of children who had head injuries but no fracture. In 1969, 570 patients were seen for head injury, by the authors, of whom 49, or 8.6 per cent, had fractures.

Wednesday at 1:30 P.M., reflecting the intimate relationship that exists between the American College of Radiology and the American Roentgen Ray Society, the College presented its traditional "Report from the American College of Radiology." Dr. Robert W. McConnell, Chairman of the Board of Chancellors of the College, acted as the Moderator. The "Report" this year comprised the following topics, which proved of extraordinarily great interest: (1) Robert W. McConnell: General introduction and comments upon productivity of Washington office; (2) Robert G. Fraser: Problems of medical specialists in Quebec with Provincial Medicare Program; (3) Stanley M. Wyman: College accreditation program under which offices and hospital departments of radiology are surveyed; (4) James P. Steele: Recent developments in technologist affairs; and (5) Clifford Wells: The College matching program and the Summer preceptorship program.

At the conclusion of the report Ted F. Leigh introduced the most recent cine film produced by the College for use in teaching medical students, residents and others, "Roentgen Anatomy of the Normal Chest," which was shown for the first time at this meeting and created a most favorable impression on the large audience present.

The Third Scientific Session began Thursday morning at 8:30 A.M. with Dr. William B. Seaman, New York, New York, Chairman of the Executive Council, presiding. At this session likewise 13 papers were delivered. Of these, 5 papers were devoted to the latest devices and methods used in the roentgenologic examination of intra-abdominal organs, 1 paper was on the carpals in congenital disorders, another on Waldenström's primary macroglobulinemia and the remaining 6 papers were on carefully selected subjects of interest to therapeutic radiologists. These papers dealt with such topics as the use of hyperbaric

recalled that dural calcification and ossification are commonly found in elderly individuals and are considered physiologic.

SUMMARY

The rare occurrence of an intracranial parasagittal chondroma of dural origin is reported.

The clinical, radiographic and surgical features are illustrated.

The possible etiology of this intracranial cartilaginous tumor is reviewed and similarities to meningiomas are noted.

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I would like to acknowledge the suggestions made in the preparation of this paper by Arch W. Templeton, M.D., Chairman, Department of Radiology, and by John Kepes, M.D., Department of Neuropathology, University of Kansas Medical Center.

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oxygen, unusual radiation response of various inoperable radioresistant tumors, second look surgery after radiation therapy for advanced cancer of the ovary, intra-arterial irradiation of tumors and betatron electron beam dosimetry. Paper 39, which was the *Award Paper of the American Radium Society*, constituted the climax of this extremely informative part of the program. The title of Paper 39 was "Radiotherapy of the Primary Lymphoma of the Orbit" and the authors were Drs. Stanley F. Foster, Charles S. Wilson, and Patricia K. Tretter of New York City. In this paper the authors reviewed the records of 33 cases treated in the past 30 years: 30 by orthovoltage radiotherapy, and 3 by surgery alone. The results indicated that good local control can be obtained and that in a significant number of cases the disease does not generalize. Even in those patients who developed generalized disease the eye symptoms were controlled. Complications of treatment were few utilizing orthovoltage technique which facilitates adequate shielding of the lens and cornea. This valuable paper will be published *in extenso* in the February 1971 issue of the JOURNAL.

On Thursday at 6:30 P.M. a Cocktail Party was held on a beautiful evening, among exquisite surroundings "at the Poolside." This was followed at 7:30 P.M. in the Casanova Room by the Annual Banquet, which is always the highlight of the Social Activities of the Society. President Roach, after introducing the newly elected officers and their wives, presented the greatly appreciated Awards to the winners of outstanding Scientific Exhibits. After this brief ceremony "without speeches" (except the scintillating aphorismal radiologic anecdotes of Dr. Lopez), there was a most entertaining floor show and a pleasant evening of dancing.

On Friday morning, the Fourth and last Scientific Session began again at 8:30 A.M., and was presided over by Dr. Hymer L. Friedell, Cleveland, Ohio, Second Vice-President of the Society. The remaining 13 papers which were read at this Session consisted of: 4 papers on topics of the gastrointestinal tract; 1 on optimum doses in excretory urography; 2 on pulmonary conditions; 2 on mammography; 1 on the adult symphysis pubis and 3 on subjects of nuclear medicine. The placing of such miscellaneous papers on the same program was amply justified by the large attendance which was still present when the meeting was adjourned until the Seventy-second Annual Meeting, which will be held in Boston, Massachusetts, September 28–October 1, 1971.

The Section on Instruction was again under the directorship of Dr. Harold O. Peterson, who invited a distinguished Faculty of 48 Instructors to give formal courses of greatest contemporary interest, but at the same time allowing for informal discussions from those attending. There were 13 periods of 90 minutes each on Tuesday, Wednesday and Thursday, and 10 periods on Friday, or 49 periods altogether, 14 of which were double periods of the same course. The arrangement of the courses was excellent and most of them were completely "sold-out."

The Scientific Exhibits this year were most outstanding and for the first time in the Society's history overflowed the available facilities. Dr. Richard G. Lester and his Committee express their sincerest regret to the many authors whose projected exhibits could not be accommodated because of space limitation. A detailed description of the displayed exhibits is given in the following pages of this issue of the JOURNAL. The Society is greatly indebted to Dr. Lester and his able Committee for their untiring effort in assembling these splendid exhibits.

The Technical Exhibits, arranged under the expert guidance of Mr. Clifford Sherratt, Honorary Member, and Dr. Ernst F. Lang, Manager of the Annual Meeting, likewise were magnificent and of greatest instructive value. An account of these exhibits with a detailed list of the exhibiting firms is also given in this issue of the JOURNAL.

The Chairman of the Committee on

FALSE POSITIVE BRAIN SCAN ASSOCIATED WITH FEBRILE CONVULSIONS*

By M. L. NUSYNOWITZ, LTC, MC, USA, *and* R. W. CLARK, MAJ, MC, USA
EL PASO, TEXAS

RADIOPERTECHNETATE brain scintillation scanning, because of its high diagnostic accuracy, low radiation dosage, and benignity, has become a screening method for the evaluation of cerebral lesions. Its sensitivity to approximately 85 per cent of documented neoplasms and a high percentage of non-neoplastic lesions is well appreciated,[1] and the rate of false negative results is acceptable in view of the simplicity of the technique. The presence of a false positive result is relatively uncommon with this procedure; we recently had a patient in whom the radiopertechnetate brain scan led to extensive diagnostic studies with negative findings.

REPORT OF A CASE

R. J., William Beaumont General Hospital 34-0104-26-08, a 14 month old white girl, was admitted on March 25, 1968 for evaluation and treatment of a focal seizure. For 3 weeks prior to admission she had been treated with antibiotics and symptomatic measures for bilateral otitis media. On the day of admission she developed lethargy, irritability, and fever to 103.4° F. While being examined, the child developed convulsions consisting of oculogyric movements, tonic movements of the right arm and leg, and incontinence; these were immediately controlled with appropriate medications. Positive physical findings included infection of the left tympanic membrane and evidence of oropharyngeal inflammation. The neurologic examination revealed obtundation, bilateral pupillary constriction, and symmetrically depressed deep tendon reflexes. The patient was maintained on anticonvulsant therapy without further difficulty.

Laboratory studies revealed that the following were normal: white blood cell and differential counts, blood glucose, serum calcium, phosphate, and electrolytes, urinalysis, cerebrospinal fluid examination, and chest and skull roentgenograms. Cultures of the nose, throat, urine, and cerebrospinal fluid revealed no pathogenic organisms. The electroencephalogram was interpreted as showing diffuse slow wave activity, slightly more pronounced on the left. Echoencephalography was normal. Brain scanning performed on March 29, 2 days after the patient became afebrile, using 2 mc of radiopertechnetate administered intravenously and an Anger camera as the imaging device, showed definite abnormality in the temporal region in the midline (Fig. 1). Repeat brain scanning 11 days later showed identical results. As a consequence of the electroencephalogram and brain scan, pneumoencephalography was performed with completely negative results. The patient has since been followed for 1 year, during which time she has had absolutely no difficulty. Repeat echoencephalography and brain scanning 10 months after the initial episode were normal (Fig. 2).

DISCUSSION

In retrospect, we feel that the abnormality initially noted on the brain scan represents concentration of radiopertechnetate by the choroid plexus. It is estimated that between 1 and 5 per cent of patients undergoing radiopertechnetate brain scanning will demonstrate markedly increased activity in the region of the choroid plexus which may lead to a false positive interpretation.[1,2] The prior administration of perchlorate will presumably block the choroidal concentration of radiopertechnetate and eliminate false positive scans on this basis. Unfortunately, we did not administer perchlorate during our studies. However, the patient's clinical course and normal repeat brain scan study (again without perchlorate) supports our contention that choroid plexus concentration of the radiopertechnetate was indeed responsible for the initial abnormal study. We sub-

* From the Radioisotope-Endocrine and Research and Development Services, William Beaumont General Hospital, El Paso, Texas.

Local Arrangements was Dr. David Kirsh, with charming Mrs. David Kirsh acting as Chairman of the Ladies' Committee. The attractive social activities prepared for this year's Annual Meeting were beautifully narrated in a little brochure titled "American Roentgen Ray Society—Ladies' Program 1970" by Ruth B. Altman, poetically inspired wife of Dr. Donald H. Altman. The detailed program included: Tuesday, a visit to Coconut-Grove, which is Miami's art colony, luncheon at the Country Store, and a cruise to VillaVizcaya, Miami's famous Italian "Villa," named after the Spanish ship, Caravel "Vizcaya," and built in 1912–1917 by James Deering, co-founder of International Harvester, employing nearly 1,000 artisans; Wednesday, a visit to fantastic Seaquarium, located on Key Biscayne, with all kinds of sea-life, performing seals and dolphins, and iguanas from the Lost Islands, seen from Monorail or Aquafoil; and Thursday, a tour of the Parrot Jungle, with its exotic parrots, macaws, cockatoos, graceful flamingos and other dazzling tropical birds. And "there were many more things to do and see." The Society is deeply appreciative to both Hospitality Committees for these unusual arrangements.

On Monday, September 28, the Annual Golf Tournament for members of the Society and their guests was held at the Country Club of Miami. Competition was for the Willis F. Manges Trophy (low net) and the Exhibitors' Trophy (low gross). The play, which was on one of the most scenic courses of the country, on a beautiful day under sunny skies was followed in the evening by cocktails and the traditional Annual Golf Dinner. Dr. Ralph M. Caulk, Past President of the Society and ardent golf enthusiast, with the aid of Dr. Carl Balli and the Local Golf Committee, made all the necessary arrangements. The Tennis Tournament was held on the same day at the North Shore Tennis Center with Dr. Andre Capi in charge.

The Committee on Press Relations, with Dr. Richard E. Litt as Local Chairman, ably assisted by Mr. Charles Honaker, Director of Public Relations of the American College of Radiology, conducted the press and public relation activities with finesse and great efficiency.

The Society expresses its sincerest thanks to the Miami Convention Bureau and the Management and Staff of the Deauville Hotel, for the efficient registration and the courteous service rendered under the difficult circumstances of our time.

To President Roach, the Society extends its warmest congratulations for the grandest Annual Meeting in the Society's history. "The pearls dropped from all six sides" and there was "an enjoyable and educational experience in their gathering."

Traian Leucutia, M.D.

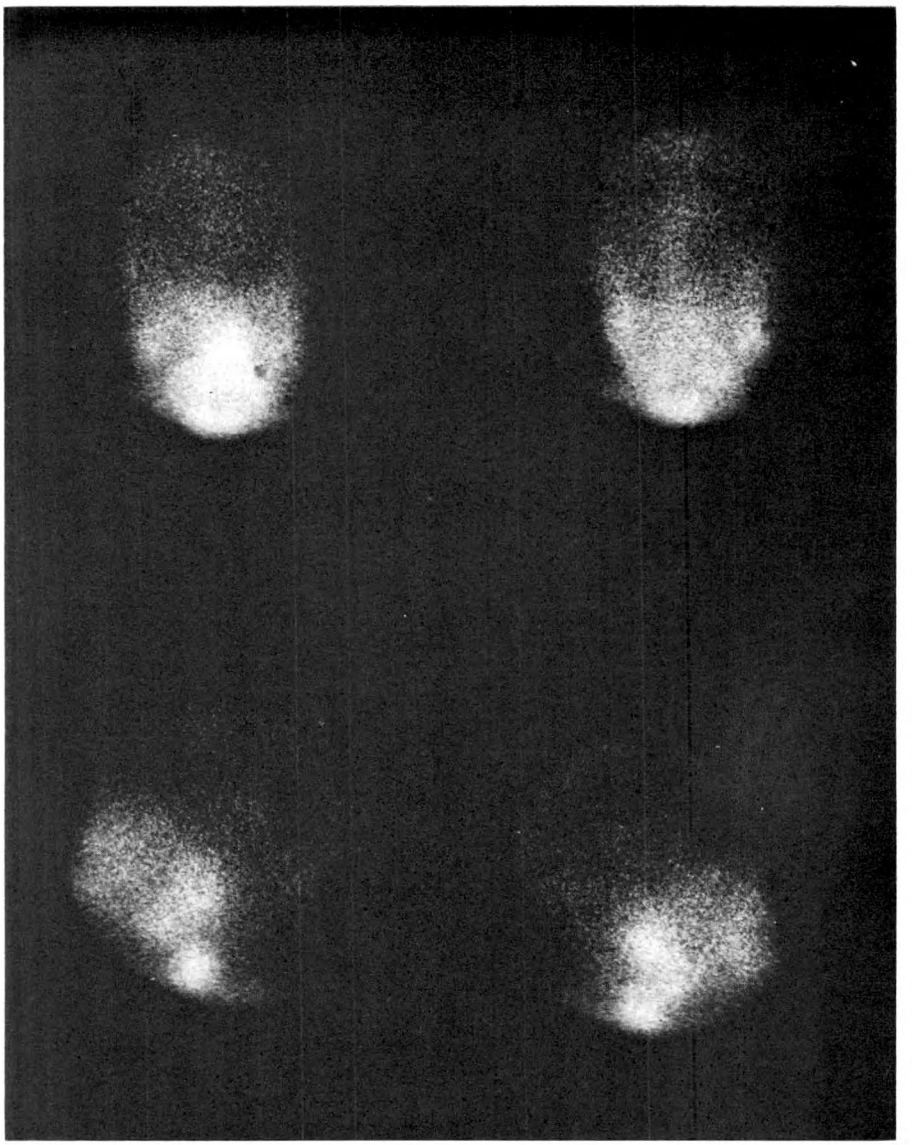


FIG. 1. Anterior, posterior, left and right lateral brain scintiphotographs showing abnormal accumulation radiopertechnetate in the region of the choroid plexus.

scribe to the recommendation of Witcofski *et al.*[2] that perchlorate should either be used routinely or prior to confirmatory repeat studies. Finally, it is interesting to speculate on the role of the febrile and/or convulsive state in altering the physiology of the choroid plexus relative to the accumulation of radiopertechnetate.

SUMMARY

A 14 month old girl had an abnorma brain scan associated with a febrile cor vulsion. We consider this a false positiv result since further diagnostic evaluatio and a brain scan performed 10 months late were normal. The brain scan abnormalit was located in the region of the choroi

THE SCIENTIFIC EXHIBITS

THE SCIENTIFIC EXHIBITS of the Seventy-first Annual Meeting of the American Roentgen Ray Society held at the Deauville Hotel, Miami Beach, Florida, September 29-October 2, 1970, were numerically the largest and most instructive in the Society's history. They were located in the decorous Peacock-Alley, overflowing into part of Napoleon Room 3, and—as in former years—many of them complemented the Scientific Essays on the same subject presented at the Scientific Sessions.

Dr. Richard G. Lester, Chairman, and Drs. Raymond E. Parks and James F. Martin, Members of the Scientific Exhibits Committee, who spared no time or effort in assembling these splendid exhibits, have been amply compensated by the great interest of the unusually large number of visitors who spent considerable time in studying these exhibits.

The secret Committee on Awards must have had a very difficult task in evaluating each exhibit and selecting those most meritorious for the customary Awards. One Gold Medal, 1 Silver Medal, 1 Bronze Medal and 10 Certificates of Merit were awarded.

The following is a list and a brief description of each Scientific Exhibit displayed at this year's Annual Meeting.

GOLD MEDAL

Azygous Arch and Its Pleural Reflections—Roentgen Anatomic Correlations. E. Robert Heitzman, M.D., Joseph V. Scrivani, M.D., and Joseph Martino, M.D., State University of New York, Upstate Medical Center, Syracuse, New York.

The posterior portion of the right mediastinal pleura is draped over the arch of the azygous vein and extends cephalad and caudad in intimate relationship to the esophagus, right paratracheal and subcarinal lymph nodes, the vagus nerves and other vital structures.

The interfaces produced between these pleural planes and right lung are altered by lymphadenopathy, esophageal tumors, dilatation of the azygous vein, enlarged left atrium and some other conditions.

The pathologic anatomy was correlated with roentgen appearances and the principle of the esophago-azygous recess was re-emphasized.

The exhibit was authoritatively documented by appropriate roentgenograms, color photographs, and pertinent anatomic cuts of gross specimens and many very beautiful drawings in various colors, depicting the very close correlation that existed between the pathologic, anatomic and roentgenographic approach.

SILVER MEDAL

Intrauterine Fetal Transfusions. James W. Lecky, M.D., William W. Olmsted, M.D., and Richard A. Bashore, M.D., UCLA School of Medicine, Los Angeles, California.

Erythroblastosis fetalis is a disease of the fetus and newborn caused by incompatibility between the fetal and maternal blood. Rh+ fetal red blood cells (RBC) enter the maternal circulation via the placenta, resulting in the formation of maternal antibodies. The antibodies, in turn, cross the placenta and cause the destruction of fetal RBC. The disease may vary from mild anemia to anemia severe enough to cause *in utero* death.

The Exhibitors performed 241 intrauterine fetal transfusions (IUFT) in 120 patients with erythroblastosis fetalis.

The indications for transfusion and an explanation of the Liley Titer Curve were presented.

Color photographs and roentgenograms of the technique were shown in conjunction

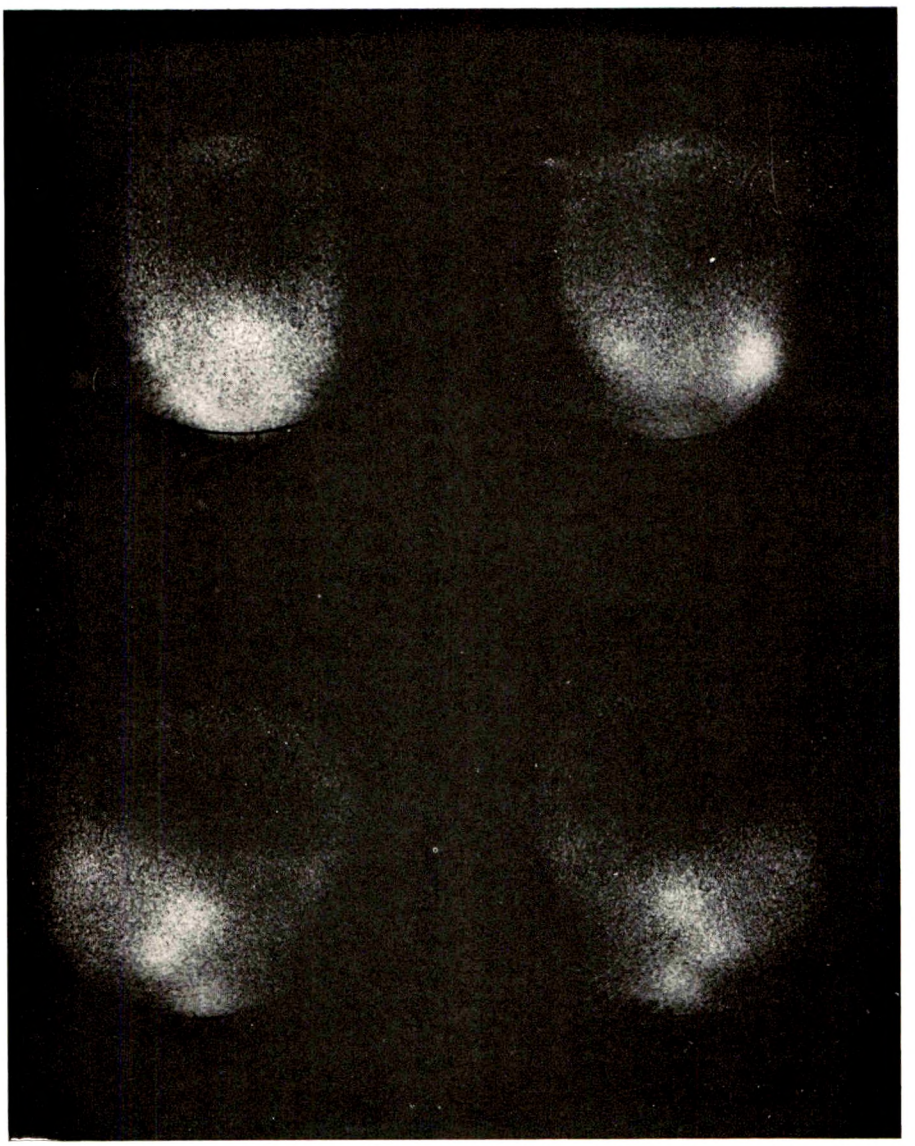


Fig. 2. Same views 10 months later showing normal results.

plexus and, presumably, perchlorate pretreatment would have prevented the false positive result due to accumulation of radiopertechnetate.

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2. Witcofski, R. L., Janeway, R., Maynard, C. D., Bearden, E. K., and Schultz, J. L. Visualization of choroid plexus on technetium 99m brain scan. *A.M.A. Arch. Neurol. & Psychiat.*, 1967, *16*, 286–289.

with a physical display of the catheter-needle system.

Errors in intrafetal position were discussed.

Bronze Medal

A Unique Radiographic Demonstration of Lumbar Vertebral Anatomy. Howard J. Barnhard, M.D., and Doyne Dodd, with the Technical Assistance of Jack Rush, R.T., and Jeannette Baskin, R.T., and Calvin Jackson, University of Arkansas Medical Center, Little Rock, Arkansas.

This exhibit was designed to demonstrate normal vertebral anatomy, using a unique approach. For presentation purposes a "building block" format was used. To the plain lumbar vertebral body were "added" various vertebral appendages, until the vertebra was completed. Subtraction films below each new roentgenogram clearly demonstrated which structure was added. The contribution of each structure to the roentgenographic image was shown in anteroposterior, lateral and both oblique views.

The approach was shown with both an isolated lumbar vertebra and another series with an intact vertebra above and below.

The opinion was expressed that the teaching value alone justifies the approach. However, new information gained is another advantage as shown by the following examples:

(1) LAO subtraction reveals that the pedicle contributes somewhat more than just the sharply defined oval density seen on the plain roentgenogram.

(2) In the oblique views the addition of the transverse process is seen to add considerable density to the anterior margin of the oval shadows that forms the "eye" of the Scotty dog. Thus the "eye" consists of both pedicle and transverse process.

Certificates of Merit

1. *The Roentgen Diagnosis of the Ischemic Gut.* Denis C. Adler, M.D., and M. Halpern, M.D., Los Angeles County, University of Southern California Medical Center, Los Angeles, California.

The routine roentgen manifestations of the ischemic bowel were presented using plain film roentgenograms and barium examination.

Reversible vascular occlusion, segmental infarction, ischemic colitis and bowel gangrene were demonstrated.

The correlation of these changes with arteriographic findings was discussed and the role of arteriography in the diagnosis of ischemic colon disease was evaluated.

2. *Roentgen Appearance of Pulmonary Vascularity in the Diagnosis of Heart Disease.* James T. T. Chen, M.D., Michael Paul Capp, M.D., Irwin S. Johnsrude, M.D., Jack K. Goodrich, M.D., and Richard G. Lester, M.D., Duke University Medical Center, Durham, North Carolina.

Changes in the pulmonary vasculature faithfully reflect the underlying cardiac disease. Careful analysis of the pulmonary vascularity is one of the most important processes toward accurate roentgen diagnosis of the circulatory system.

The purpose of this exhibit was to illustrate the specific patterns of pulmonary vascular alteration in different cardiac conditions, congenital and acquired.

For example, leftward streaming of the pulmonary blood flow is the hallmark of valvular pulmonary stenosis and upward streaming is diagnostic of severe mitral stenosis. The roentgenograms were supplemented by angiocardiograms and lung scans whenever necessary.

There was a scientific essay on the same subject.

3. *Quantitation Transmission Radiography As an Index of Regional Ventilation.* Ronald G. Evens, M.D., Mustafa Adatepe, M.D., Rex Hill, M.S., Joanne Markham, B.S., and E. James Potchen, M.D., Mallinckrodt Institute of Radiology, Washington University, St. Louis, Missouri.

A new technique using quantitative transmission radiography affords a clinical

CEREBROSPINAL FLUID (CSF) SCANNING: CISTERNOGRAPHY*

By A. EVERETTE JAMES, JR., M.D.,† FRANK H. DELAND, FRED J. HODGES, III, *and* HENRY N. WAGNER, JR.,
with the assistance of WENDY A. NORTH, M.I.R.
BALTIMORE, MARYLAND

ASSESSMENT of cerebrospinal fluid dynamics by means of radioactive materials introduced into the subarachnoid space is increasing. Because of the improvement in imaging techniques and the development of new radiopharmaceuticals, the quality of the information is improving and specific structures can now be identified. The anatomic information present in the radioisotopic image and the patterns of cerebrospinal fluid (CSF) flow provide a basis for assessment of abnormalities of CSF movement and resorption.

GENERAL PRINCIPLES

The distribution of cerebrospinal fluid is graphically depicted by scanning or scintigraphy after the introduction of an appropriate radiopharmaceutical into the subarachnoid space.[2,4,9,10,14] This space normally contains approximately 120–160 ml. of cerebrospinal fluid; some 22–40 ml. are within the ventricular system.[23,24,27] Within the skull, the different regions of the subarachnoid space vary greatly in size, being small or absent over the summits of the cerebral convolutions and large where the brain does not follow closely the contour of the skull.[5,8,21,26,35] Areas of the subarachnoid space which contain more radiopharmaceutical during cisternography are readily seen in the images.[13]

Although it has been thought in the past that cerebrospinal fluid is produced only in the ventricular choroid plexuses, CSF is probably produced throughout the subarachnoid space.[3] The greatest volume is probably produced in the third and lateral ventricles. Movement of cerebrospinal fluid is well demonstrated at cisternography.[9,10] There is experimentally a demonstrable net flow of cerebrospinal fluid from the ventricles to the remainder of the subarachnoid space. The rapid movement of radiopharmaceuticals from the ventricles to the region over the cerebral convexity, when injected intraventricularly, documents this net flow. CSF movement is probably from areas of greatest CSF production to areas of greatest resorption.[3,28,33] After injection intrathecally in the lumbar region, the movement of radiopharmaceuticals into the cranial area suggests that currents within the CSF do occur as more than random movement of the radiopharmaceutical is seen.

Since cerebrospinal fluid is in osmotic balance with blood, diffusion of injected material out of the subarachnoid space through the subarachnoid membrane occurs readily for most substances. Large protein molecules diffuse slowly out of the CSF space, whereas small molecules and ions are readily exchanged across the arachnoid membrane.[33] For this reason, human serum albumin (molecular weight, 70,000) has been labeled with radionuclides (Table 1) for ventriculography and cisternography. The albumin molecule does not diffuse readily through the subarachnoid membrane and is, during the period of study, confined to the subarachnoid compartment.[25,33] Molecules smaller than albumin such as Yb^{169} diethylenetriaminepentaacetic acid (molecular weight 600) will also remain temporarily localized to the subarachnoid space during cisternography. Because CSF movement may be assessed

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This study was supported by U.S.P.H.S. Grant G.M. 10548.
† Advanced Academic Fellow of James Picker Foundation on Recommendation of the Committee on Radiology, NAS-NRC.

assessment of regional pulmonary ventilation in man. This has been useful as an adjunct to perfusion lung scintiscanning by identifying the change in thoracic density during respiration, allowing distinction of some patients with altered regional perfusion due to inhomogeneous ventilation from those with normal ventilation and diminished perfusion due to pulmonary embolism.

The method proposed is far simpler than either radioactive gas or particle inhalation techniques.

In addition, the computer processing may well permit data storage and computer interpretation.

The early recognition of alterations in ventilation in a large screened population may assist in the early diagnosis of disordered regional pulmonary function as has been seen in bronchogenic carcinoma and pulmonary emphysema.

The method was exhibited and clinical examples of its usefulness were shown.

There was a scientific essay on the same subject.

4. *Positive Contrast Orbitography.* William N. Hanafee, M.D., Robert S. Hepler, M.D., and Carl G. Coin, M.D., UCLA Center for the Health Sciences, Los Angeles, California.

Positive contrast orbitography, with image intensification control for needle placement, has proven exceedingly helpful in localizing infiltrating lesions of the orbit and tumors about the orbital apex. This fact was amply demonstrated in the exhibit.

Fluoroscopy confirms satisfactory placement of the contrast material as well as aids in identifying increased resistance at tumor locations.

There was a scientific essay on the same subject.

5. *The Roentgenologic Evaluation of Post Tracheostomy Lesions.* A. Everette James, Jr., M.D., Alfred L. Weber, M.D., Jack W. Bowerman, M.D., Murray L. Janower, M.D., Alexander S. MacMillan, Jr., M.D., and Hermes C. Grillo, M.D., Johns Hopkins Hospital, Baltimore, Maryland.

Roentgenologic methods are excellent for the evaluation of post-tracheostomy complications. Both the abnormal anatomy and altered physiology can be graphically depicted. The advent of the balloon cuffed tracheostomy tube to insure an airtight system for mechanically assisted respiration has led to an increase in the number of post-tracheostomy complications and has given rise to tracheal abnormalities peculiar to this type of tracheostomy tube.

Recent developments in surgical techniques of tracheal reconstruction have made it possible to definitely repair these abnormalities. This was demonstrated in the exhibit.

6. *Yb^{169} DTPA: A Versatile Radiopharmaceutical.* A. Everette James, Jr., M.D., Fazle Hosain, Ph.D., Frank H. DeLand, M.D., and Henry N. Wagner, Jr., M.D., Johns Hopkins Medical Institutions, Baltimore, Maryland.

Ytterbium 169 Diethylenetriaminepentaacetic Acid (DTPA) is found to be a versatile radiopharmaceutical and is utilized for renal and neurologic scanning procedures.

Yb^{169} is a rare earth element of the lanthanon group that forms a physiologically stable chelate with DTPA. This radiopharmaceutical has a long physical half life (32 days) and a short biologic half life (1–2 hour plasma clearance, 10 hours retention in the cerebrospinal fluid compartment), high photon yield (63×10^9 photons per rad whole body dose), and rapid excretion as a glomerular substance resulting in low radiation dose to the patient. Although the gamma spectrum is complex, the 177 and 198 kev. photons are suitable for imaging either with a scintillation camera or rectilinear scanner.

The use of Yb^{169} DTPA for brain, renal, and cerebrospinal fluid scanning as well as determination of GFR and extracellular fluid volume was illustrated.

7. *A Computer Controlled Color Coded Scintigram Display System.* Carl Jansen, M.D., Ralph Adams, B.A., Ernest J. Braun, M.D., and Charles Finney, M.S., Department of Radiation Therapy and

for as long as 48 to 72 hours after injection, a radionuclide with an effective half life of 10 hours or longer is preferred.

When the radiopharmaceutical is injected intrathecally into the lumbar subarachnoid space, it normally reaches the basal cisterns in 1–3 hours, the frontal poles and sylvian fissures in 3–6 hours, the cerebral convexities by 12 hours, and the sagittal sinus area by 24 hours.[9,13] Following an intrathecal spinal injection, radioactivity is not normally detected in the ventricular system, probably reflecting the net CSF flow from the ventricles into the remainder of the CSF space. The radionuclide appears to flow normally into all anatomic structures entered by CSF except the ventricles.[10] Movement of radioactivity within the CSF compartment is not influenced by posture or activity of the patient, nor by barbotage at the time of injection. When large volumes of injected material are used, movement is more rapid. Flow of injected radiopharmaceutical has been observed to be faster in younger patients.[10]

When radioactivity is introduced directly into the ventricular system, it normally appears in the cisterna magna and basal cisterns within minutes, confirming the results of Dandy and Blackfan, who used dyes to monitor CSF movement.[5] From the basal cistern area, movement of the radioactivity is similar to the movement observed after intrathecal injections in the lumbar area. Cerebrospinal fluid movement from the basal cisterns of the skull to the area of maximum resorption (parasagittal region) is mainly through the anterior communicating pathways (Fig. 1). Flow of the CSF as reflected by radionuclide movement is both by a central and more superficial route to the parasagittal region. The central flow of radioactivity consists of two principal routes; one superiorly from the suprasellar cistern through the cisterna lamina terminalis to the callosal cistern where it then passes into the interhemispheric cistern medially and then upward to the parasagittal area. The more inferior of the pathways is from the basal cisterns to the quadrigeminal cistern (c. quadrigemina) by way of the ambient cisterns. From the quadrigeminal plate cistern the radionuclide passes to the posterior portion of the callosal cistern and again between the cerebral hemispheres in the interhemispheric cistern to the parasagittal region (Fig. 1).

The lateral pathway of CSF flow is through the sylvian cisterns that occupy the space between the posterior surface of the frontal lobes and the anterior surface of the temporal lobes. From the sylvian cistern the radionuclide flows out over the cerebral convexity to the parasagittal region. The cisterns appear to communicate freely. They are visualized because of the radionuclide they contain.[20–22]

The posterior pia-arachnoid limiting membrane,[8] which almost always prevents free egress of air during pneumoencephalography over the posterior and lateral aspects of the cerebellar hemispheres, does not appear to alter the movement of injected radioactivity. Radioactivity is seen surrounding the cerebellum. However, temporary or transient cessation of movement of radioactivity in a small area may not be detected. Resolution of anatomic detail does not permit identification of delicate structures such as the limiting membrane of Liliquist anteriorly and repeated monitoring by frequent imaging has not been done in humans.

This free movement of radionuclide into the entire CSF space, except the ventricles,

Table I

RADIONUCLIDES UTILIZED FOR CISTERNOGRAPHY AND VENTRICULOGRAPHY

I^{131}—human serum albumin
I^{131}—autologous spinal fluid protein
Tc^{99m}—pertechnetate
Tc^{99m}—human serum albumin
Ga^{68}—ethylenediamine tetraacetic acid
As^{74}—sodium arsenate
In^{113m}—colloid
Yb^{169}-DTPA—diethylenetriaminepentaacetic acid (DTPA)

Nuclear Medicine, Loma Linda University, Loma Linda, California, and White Memorial Hospital, Los Angeles, California.

A computer controlled system was described for the production of scintigrams from the Anger camera as a series of color coded isocount displays.

Comparison of color coding with the conventional gray scale was illustrated with scintigrams.

8. *Lung Changes in Left Heart Failure.* William T. Meszaros, M.D., 104 S. Michigan Avenue, Chicago, Illinois.

There is a wide spectrum of lung changes in left heart failure.

A classification of these changes was presented.

The early findings were stressed and some uncommon manifestations were illustrated.

9. *Radiologic Manifestations of Primary Macroglobulinemia.* Roger R. Renner, M.D., James J. Condon, M.D., and Douglas A. Nelson, M.D., State University of New York, Upstate Medical Center, Syracuse, New York.

Cases of primary macroglobulinemia were first described by Jan Waldenström in 1944 as a possible variant of multiple myeloma.

Since then over 400 cases have been recorded, and this disease is now recognized as part of the malignant myeloproliferative spectrum.

The exhibit gave a short, concise summary of the clinical and pathologic findings and showed a variety of abnormal roentgenographic changes that may occur.

All systems may be affected, but more frequent roentgenographic findings are found in the reticuloendothelial system and the chest.

There was a scientific essay on the same subject.

10. *Herniography; Radiologic Investigation of Inguinal Hernia.* Leonard E. Swischuk, M.D., and Teresa M. Stacy, M.D., Children's Memorial Hospital, Oklahoma City, Oklahoma.

Herniography is a simple and informative procedure which is of considerable aid in the assessment and identification of inguinal hernias and communicating and non-communicating hydroceles. (1) It can confirm the clinical diagnosis of hernia or communicating hydrocele (potential hernial sac). (2) It can identify a potential hernial sac (communicating hydrocele) after the hernia has been spontaneously or otherwise reduced, or when the presence of a hernia or hernial sac is in doubt clinically. (3) It can identify an unsuspected hernia or potential hernial sac (communicating hydrocele) on the opposite side. (4) It can differentiate between incarcerated hernia and non-communicating hydrocele. (5) It can establish the presence of a testicle. (6) It can probably demonstrate tumors of the spermatic cord.

The technical factors, normal anatomy, pitfalls, and complications were discussed, and case examples were shown.

OTHER EXCELLENT EXHIBITS

Pediatric Case of the Day. Donald H. Altman, M.D., Richard E. Litt, M.D., and Leonard A. Greenberg, M.D., Variety Children's Hospital, Miami, Florida.

A different Pediatric Case was presented each day. The history and pertinent films permitted a single diagnosis.

The answer was given the following day with a discussion and color demonstration of the pathology.

A reward was given to all correct diagnosticians.

Ventriculo-Venous Cerebrospinal Fluid Shunts: Radiologic Analysis. Jeremy Altman, M.D., and Alton Everette James, Jr., M.D., The Johns Hopkins Medical Institutions, Baltimore, Maryland.

Increased interest and technical improvements in treatment of hydrocephalus by shunting excess ventricular fluid into the blood stream make it mandatory that the radiologist be familiar with the normal appearance and manifestations of attendant complications of the various devices used.

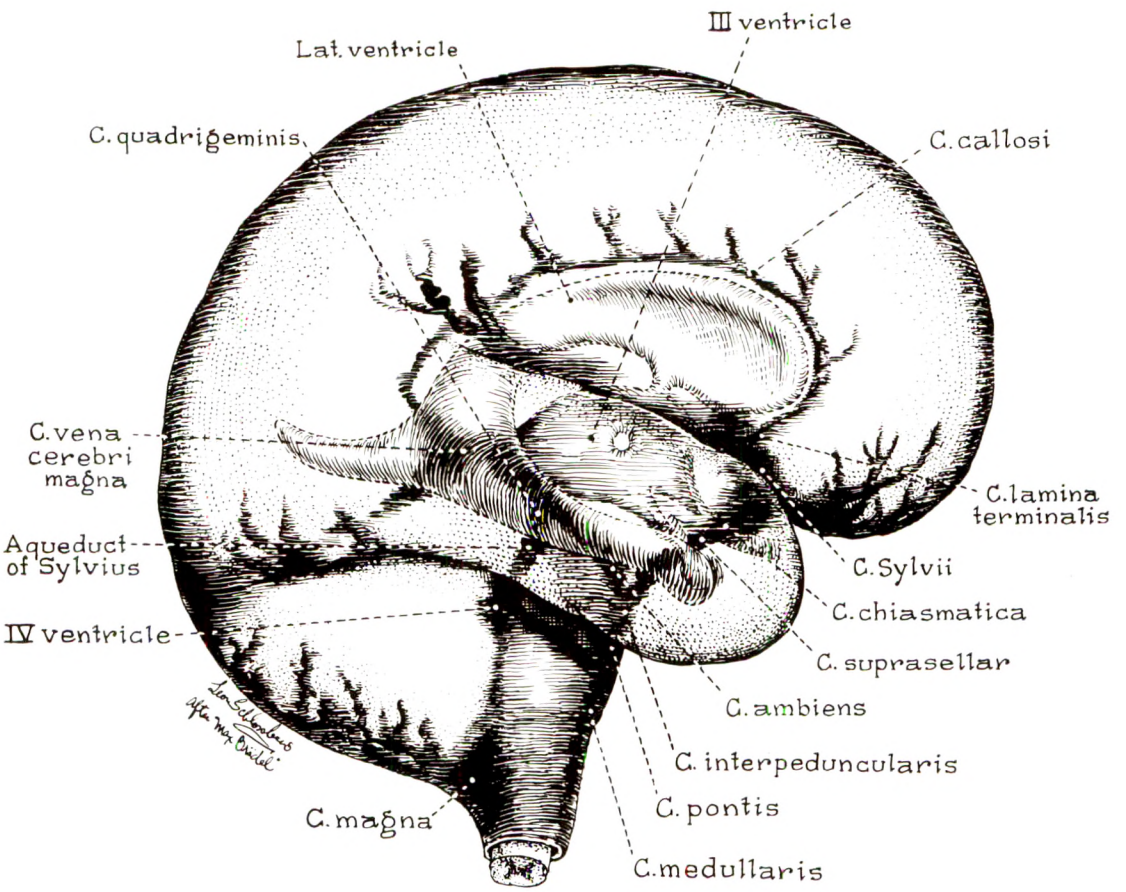


FIG. 1. Cerebrospinal fluid (CSF) pathways.

results in homogeneous distribution of radioactivity on the delayed studies.

RADIOPHARMACEUTICALS

Several radiopharmaceuticals have been employed in cisternography and ventriculography. They offer varied properties (half life, molecular size, energy . . .) that can in different clinical situations be used advantageously [6,18,19] (Table 1). Suitable effective half life determines the usefulness of a radiopharmaceutical for delayed studies (24, 48 and 72 hours) when assessment of flow over the cerebral convexities and absorption of CSF in the parasagittal area are of primary interest.[1] This is one reason I^{131} (T 1/2 = 8.3 days) has been the most commonly employed radionuclide for cisternography. By labeling human serum albumin (HSA) with I^{131}, a radiopharmaceutical is obtained which, when properly introduced: (1) remains localized to the subarachnoid space for the period of the study; (2) does not appear to physiologically alter the CSF flow; and (3) in small (less than 4 mg.) amounts causes no significant meningeal reaction.[29] The gamma energy of I^{131}-HSA (364 kev.) is suitable for rectilinear scanning and acceptable images can be obtained with the scintillation camera, even though the energy is not optimum for this detection device. The chief disadvantage of I^{131}-HSA is the limitation of the amount of injected albumin which limits the amount of injected radioactivity (usually 100 μc). The other commonly employed radiopharmaceutical, Tc^{99m} labeled serum albumin, has both

This was amply demonstrated by the Exhibitors.

Angionephrotomography and Subtraction: Relative Value in Renal Mass Lesions. Christos A. Athanasoulis, M.D., Shiv Navani, M.D., Ernest J. Ferris, M.D., and Jerome H. Shapiro, M.D., Boston City Hospital & University Hospital, Boston, Massachusetts.

Avascular and hypovascular neoplasms of the kidney must be differentiated from simple renal cysts.

At Boston University Medical Center, unless a mass investigated by selective renal angiography is obviously vascular, angionephrotomography is routinely performed.

The relative value of this procedure as well as subtraction in the differential diagnosis of renal masses was presented.

A Means of Reducing Radiation Exposure from Technetium Generator Operations. Huey D. Barnett, B.S., C. Craig Harris, M.S., and Jack K. Goodrich, M.D., Duke University Medical Center, Durham, North Carolina.

Technicians working in a nuclear medicine laboratory are being exposed needlessly to low energy radiations of technetium 99m.

Possible reasons for this are: a false sense of security related to overfamiliarity and repetitious use; lead bricks are cumbersome to use and rather expensive; and there is a need for good visibility.

The Exhibitors have devised a shield, adapted from the design of radium shields, using a plastic cell filled with a saturated solution of lead perchlorate. This shield has proven effective, easy to construct, and relatively inexpensive.

The exhibit demonstrated the physical appearance and shielding properties of this device. The fabricating methods were also explained.

Control of Gastrointestinal Hemorrhage by Selective Mesenteric Arterial Drug Infusion. Stanley Baum, M.D., and Moreye Nusbaum, M.D., Graduate Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

This exhibit described the authors' experience with the pharmacologic control of portal hypertension by means of selective infusion of vasopressin directly into the superior mesenteric artery.

The laboratory work as well as several representative case reports of patients with bleeding esophageal varices, successfully managed by the infusion of vasopressin into the superior mesenteric artery was presented.

The technique has also been used in patients bleeding from arterial and capillary causes, such as peptic ulcers, stress ulcers and hemorrhagic gastritis. Representative cases of this group were also illustrated.

Intravenous Urography—Fact & Fiction. Joshua A. Becker, M.D., and Bruce L. McClennan, M.D., College of Physicians & Surgeons, Columbia University, New York, New York.

With the introduction of high dose urography a considerable amount of confusion has occurred as to the appropriate dose for optimal diagnostic studies of the urinary tract.

The exhibit covered the dosage levels of 0.5, 1.0, and 2.0 cc./lb. of contrast material and correlated the quality of the urogram to this.

Also, the parameters of blood attenuation curves, urine contrast concentration, blood pH changes, and serum electrolyte changes in normal renal function were reviewed.

The effect of initial urine osmolality and the administration of either sodium or methylglucamine salts contrast material were discussed.

Diagnostic Criteria in Hypotonic Duodenography. Karl T. Benedict, M.D., Joseph T. Ferrucci, Jr., M.D., S. Boyd Eaton, M.D., and Donald J. Fleischli, M.D., Massachusetts General Hospital, Boston, Massachusetts.

This exhibit illustrated duodenographic findings in benign and malignant conditions of the pancreas and duodenum.

Examples of mass lesions, strictures and mucosal fold abnormalities have been

desirable and undesirable properties. The short half life (6 hours) and pure gamma emission of Tc^{99m} permit larger doses (1–4 mc Tc^{99m} *versus* 100 μc I^{131}), higher specific activity, and greater photon yield. The gamma energy of 140 kev. is optimum for scintillation camera imaging and suitable for rectilinear scanning. However, a short half life is not suitable for monitoring the CSF flow when delayed studies are required.[31] Tc^{99m} serum albumin has, therefore, found its greatest usefulness in those studies where anatomic detail and short term observations are desired.[5] Tc^{99m}-pertechnetate has been advantageously employed for direct ventricular injection or instillation into neurosurgical shunts.[11,12] Tc^{99m}-pertechnetate readily diffuses out of the subarachnoid space which imposes a severe limitation upon the general use of this radiopharmaceutical in cisternography, and for this reason it is combined with serum albumin.

Recently, Yb^{169}-DTPA has been employed as a radiopharmaceutical for cisternography and ventriculography.[34] A long physical half life (32 days), coupled with a shorter effective half life (10 hours), offers the advantages of both Tc^{99m}-serum albumin and I^{131}-serum albumin; *i.e.*, adequate physical half life for extended studies (24–72 hours) and a sufficiently short biologic half life permitting larger doses (1 mc). This particular radiopharmaceutical has been used successfully in over 50 patients to date.

The proper radiopharmaceutical must be selected according to the information desired from the study. To assess the cerebrospinal fluid flow and absorption the study will require imaging at time intervals of 24 to 48 hours or longer. Therefore, a radiopharmaceutical with a half life in the subarachnoid space of 10–12 hours or greater is desirable. For a study requiring optimal anatomic delineation at the expense of flow characteristics, a radiopharmaceutical with short half life, high specific activity, and high photon yield should be employed.

TECHNIQUE OF MONITORING AND IMAGING OF CSF FLOW

As previously described, when injected into the lumbar intrathecal space, the radioactivity will normally pass cephalad to enter the basal cisterns where it accumulates before moving through the communicating pathways and over the cerebral cortex.[7] It will not normally enter the fourth, third or lateral ventricles. Therefore, our policy is to wait approximately 2 hours before obtaining initial radioisotopic images (Fig. 2, *A–C*). Although the communicating pathways between the basal cisterns and the more superior subarach noid space over the cerebral convexity are often seen on the initial examination, we often most optimally visualize the radiopharmaceutical in these areas on the 6 hour study (Fig. 3, *A–C*).[7] By the time of the 24 hour study, the over-all distribution of radioactivity over the cerebral cortex will usually demonstrate any nonuniformity or unilaterality. The superior sagittal sinus appears as a localized band of increased activity that lies slightly inferior and conforms to the shape of the bony calvarium in the lateral view superiorly (Fig. 4, *A* and *B*). If this concentration of radioactivity is not present in the sagittal area, delayed studies (48 or 72 hours) should be obtained. Any delay of CSF flow in which the radioactivity is present in the basal cisterns at 2 hours but does not progress to concentrate in the parasagittal region by 48 hours should be regarded as abnormal.

As in pneumoencephalography, specific projections are necessary to demonstrate certain anatomic features. Routinely, lateral, anterior, and posterior views are obtained. The lateral view is made with the patient lying prone and the head turned to one side (Fig. 5). In this projection the midsagittal plane of the cranium is parallel to the detection probe face. The radioactivity emanating from the CSF compartments will show optimally those structures in the focal plane of the rectilinear scanner, which in the lateral projection is 5 cm. from the outer table of the skull. The an-

selected to illustrate the differential diagnostic features.

The normal anatomy, including recognition of the swollen papilla of Vater was stressed.

Senographic Mammograms. J. W. Birsner, M.D., M. B. Hermel, M.D., M. G. Murdock, M.D., and J. Gershon-Cohen, M.D., Lucille Reid Cancer Institute, Bakersfield, California and Private Offices, Philadelphia, Pennsylvania.

The technical advantages afforded by the Senograph in mammography were demonstrated in this exhibit.

The Senograph is an apparatus specially designed for expeditious roentgenography of the breast, with a specially designed x-ray tube having a fine focal spot of a molybdenum instead of a tungsten target.

Antemortem Arteriographic Observations in Polyarteritis Nodosa. Klaus M. Bron, M.D., and J. William Stilley, M.D., University of Pittsburgh, School of Medicine, Presbyterian-University Hospital, Pittsburgh, Pennsylvania.

The antemortem diagnosis of polyarteritis nodosa (PAN) based either on clinical findings or biopsy results may be equally frustrating and frequently the lesions are only identified at autopsy.

Arteriography adds a new approach to this diagnostic dilemma.

This technique identifies various features encountered in PAN, namely: (1) characteristic multiple parenchymal arterial aneurysms, in one or multiple organs simultaneously; (2) evidence of increased arterial vascular resistance due to intravascular thrombosis or spasm; and (3) perirenal hematoma—a complication of the disease.

Selective injection of a pharmacologic vasodilator may be necessary to insure adequate demonstration of the diagnostic features of PAN, since the regional blood flow can be severely reduced by the increased vascular resistance.

Pediatric Arteriography for Abdominal and Extremity Lesions; Clinical Experience, Indications and Techniques. Klaus M. Bron, M.D., Max W. Safley, M.D., and Bertram R. Girdany, M.D., University of Pittsburgh School of Medicine, Presbyterian-University Hospital, Pittsburgh, Pennsylvania.

The exhibit emphasized the usefulness of percutaneous arteriography for the diagnosis and management of common pediatric abdominal and extremity problems. To date more than 90 children have been studied, ranging in age from 13 months to 16 years, without any serious complications due to the technique.

The indications for these studies were: (1) to confirm the presence and distinguish the type of clinically suspected mass lesions of the kidney, adrenal gland, liver, pancreas and retroperitoneal area; (2) to locate gastrointestinal bleeding of unknown etiology; (3) to determine acute injury and complications of abdominal and peripheral trauma; and (4) to demonstrate the vascular anatomy and degree of impairment in congenital malformations and acquired obstructions.

Positive arteriographic results were observed in more than 75 per cent of patients studied, and the negative results were equally important in determining subsequent management.

Pediatric Brain Scanning. Hyo H. Byun, M.D., Marion F. Magalotti, M.D., Stasia J. Frank, M.D., Anthony J. Raimondi, M.D., Ki Eun Chung, M.D., and Theodore Fields, M.D., Cook County Hospital, Chicago, Illinois.

Due to the low radiation dosage with Tc^{99m}, brain scanning has become a versatile diagnostic method in pediatric neurologic disorders.

Normal and abnormal findings along with criteria for diagnosis were presented from a review of over 200 cases with scans.

Dosimetry calculations were shown.

Radionuclide Angiography of Chest Masses in Children. James J. Conway, M.D., Children's Memorial Hospital, Chicago, Illinois.

The histologic variation of chest lesions which may be present in childhood is extensive. Knowledge of the vascular dynamics of such a lesion enhances preopera-

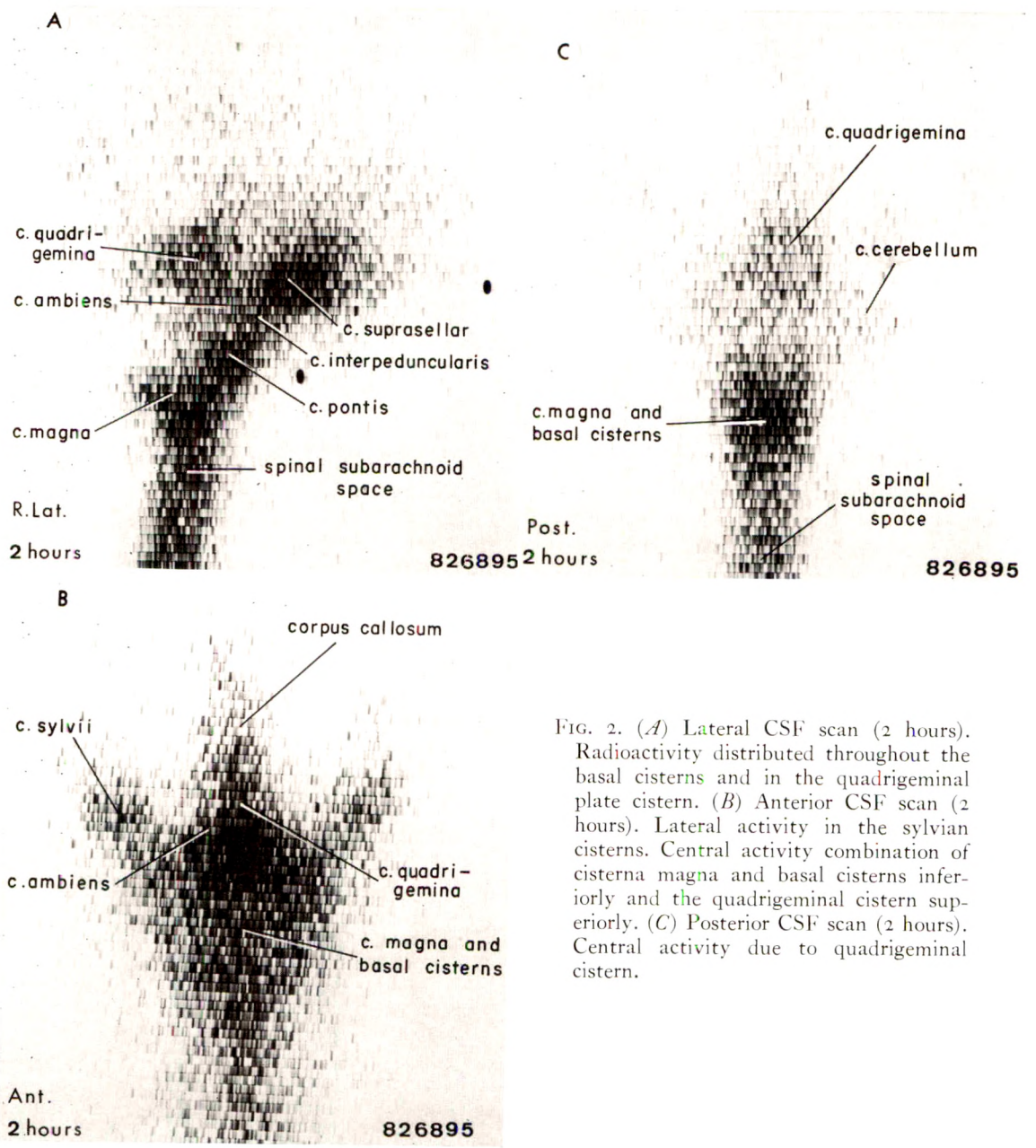


FIG. 2. (*A*) Lateral CSF scan (2 hours). Radioactivity distributed throughout the basal cisterns and in the quadrigeminal plate cistern. (*B*) Anterior CSF scan (2 hours). Lateral activity in the sylvian cisterns. Central activity combination of cisterna magna and basal cisterns inferiorly and the quadrigeminal cistern superiorly. (*C*) Posterior CSF scan (2 hours). Central activity due to quadrigeminal cistern.

terior and posterior views are made with the canthomeatal line perpendicular to the face of the detection device (Fig. 6). This necessitates changing the patient's position for each view if a single probe detection device is employed. Without moving the patient, corresponding roentgenograms are obtained to monitor positioning during the scan and to orient the scan at the time of viewing. Positional markers are placed on the roentgenogram and the photoscan for alignment and orientation; an essential part of the procedure that must be carefully monitored by the physician responsible for interpreting the study.[18] This technique should be repeated for each separate view. The necessity of obtaining both an anterior and posterior view is empha-

tive diagnostic accuracy and aids operative management.

Early and delayed radionuclide angiography following the rapid injection of Tc^{99m} pertechnetate was recorded in 3 phases on a gamma scintillation camera.

The appearance of cystic, solid, and hypervascular lesions was illustrated.

Comparative Diagnostic Studies in Pelvic Neoplasms. Dorothy F. Cooney, M.D., Silvestre P. Talon, M.D., and Ira H. Low, E.E., Christ Community Hospital, Oak Lawn, Illinois.

Combined studies of pelvic masses were made utilizing intraosseous venography, selective pelvic arteriography, and lymphangiography to establish criteria for differentiating benign and malignant neoplasms in female pelves.

These studies, as well as the results, were presented in the exhibit and a motion picture film was utilized with a minified projector to give additional visual information.

Spinal Manifestations of Coccidioidomycosis. Captain M. K. Dalinka, USAF MC, Colonel William H. Greendyke, USAF MC, and Major David B. Goldenberg, USAF MC, Wilford Hall USAF Medical Center, Lackland Air Force Base, Texas.

The exhibit consisted of color transparencies and roentgenograms. It illustrated and discussed the protean manifestations of spinal coccidioidomycosis as seen in 7 patients.

Emphasis was placed on the frequency of multiple spinal and extraspinal foci of disease, relative disk cartilage preservation and indiscriminate involvement of the vertebral body and its appendages.

Coccidioidomycosis is an extremely common disease in the endemic region of the southwestern United States. Millions of tourists who visit this area annually, as well as thousands of resident military personnel and their dependents, are exposed to this disease. Subsequent relocation of these groups to all parts of the U. S. underscores the importance of the awareness of the clinician in the nonendemic areas to the varied manifestations of coccidioidomycosis.

Development and Clinical Trials of a New Mammographic Unit. Maurice R. Dufresne, M.D., Denise O. Oliva, M.D., and Rene A. Beique, Ph.D., Notre Dame Hospital, Montreal, Quebec, Canada.

Various statistical reports indicate clearly that approximately 1 out of 16 North American women will develop breast cancer in her lifetime, so that early diagnosis of breast cancer is imperative.

Doctor Charles Gros, from Strasbourg, France, and his group, by their intensive study and research work on the fixed molybdenum target tube have definitely improved the contrast quality of the radiographic image in mammography.

In early 1969, the Exhibitors started investigating the efficiency of a rotation molybdenum target, having in mind that the output of such a tube could be considerably higher than the one with a fixed anode. They first designed a very rudimentary prototype tube mount to be used for mammography purposes only. A tungsten tube was employed for a period of 2 months and, then, a rotating molybdenum target tube was installed in March 1969. In late March 1969, a research program was instituted. During this last year and with the help of specialized x-ray engineers as well as the devoted collaboration of their chief technician in mammography, Mrs. Louise Lacroix, they have been able to accumulate and study sufficient research material to establish a definite technique and design of the apparatus, which were summarized in this exhibit.

The exhibit was beautifully displayed, encompassing many photographs in color of the finished apparatus, details of the practical techniques, choice of target film distance, clinical trial of isocentric tube mount, choice of films, and other pertinent factors accounting for a high performance diagnostic mammography.

The Bony Bridge in Otoroentgen Diagnosis. Lewis E. Etter, M.D., Western Psychiatric Institute & Clinic and Falk

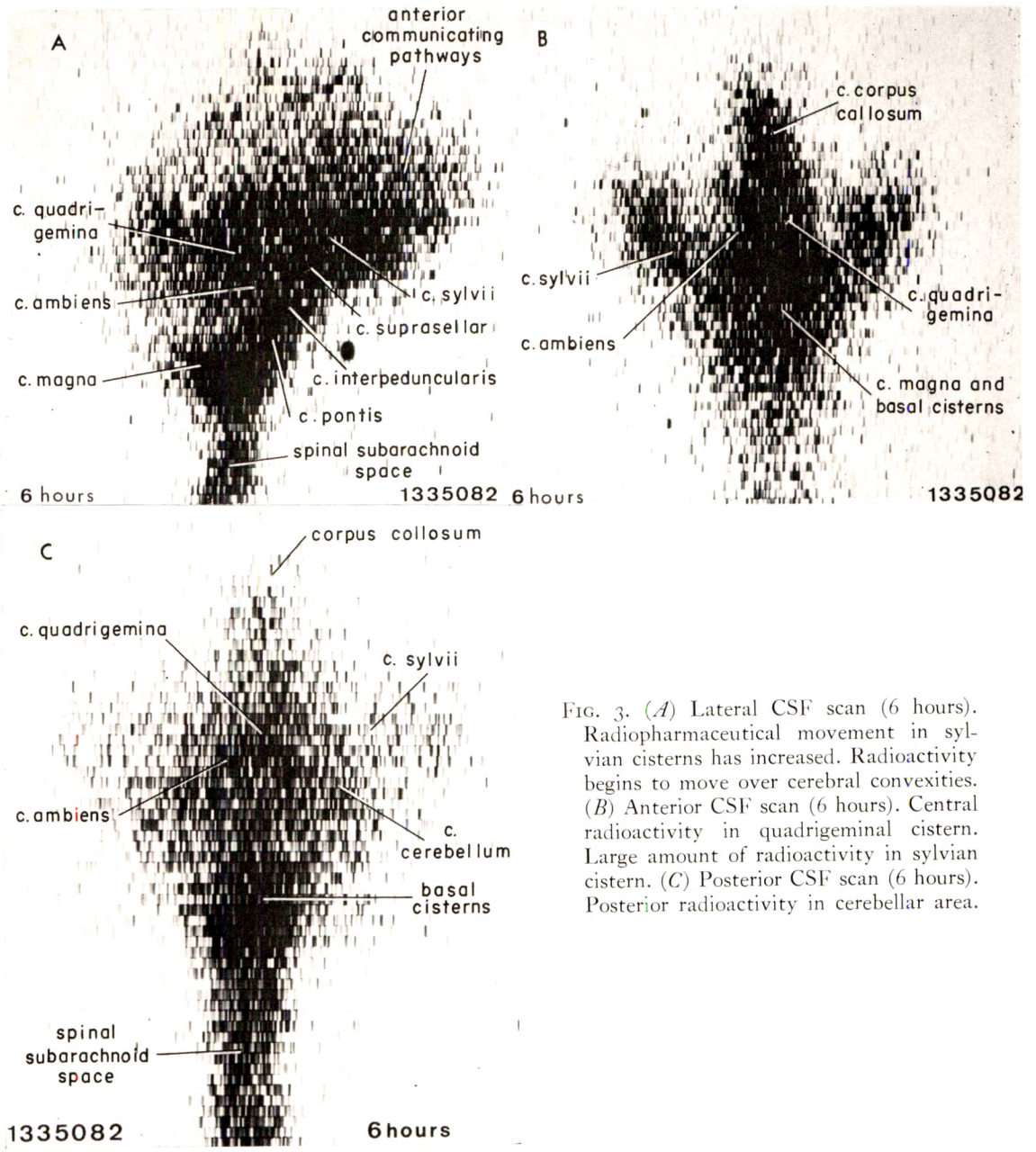


FIG. 3. (*A*) Lateral CSF scan (6 hours). Radiopharmaceutical movement in sylvian cisterns has increased. Radioactivity begins to move over cerebral convexities. (*B*) Anterior CSF scan (6 hours). Central radioactivity in quadrigeminal cistern. Large amount of radioactivity in sylvian cistern. (*C*) Posterior CSF scan (6 hours). Posterior radioactivity in cerebellar area.

sized by the fact that with a rectilinear scanner the focal plane of the collimator will optimally visualize different anatomic structures in each view. An example of this is the appearance of the sylvian cisterns in the anterior and posterior views. The major portion of the sylvian cisterns are anterior in the cranium and better visualized in the anterior than the posterior view (Fig. 7, *A* and *B*). In communicating hydrocephalus, radioactivity usually enters the ventricles and the most superior portion of the activity in the lateral ventricle has the appearance of a heart-shaped collection of radioactivity with a very active center (Fig. 8, *A–C*). This corresponds to the configuration of the posterior portion of the body and atrium of the lateral ventricle at pneumoencephalography.[32] On the posterior view the radioactivity in the

Clinic, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania.

Complete roentgen examination of the middle ear and its associated structures requires roentgenograms of the utmost clarity for the clearest demonstration of the structures in the epitympanic space, including the bony bridge.

In everyday roentgen otologic practice it becomes imperative to recognize this very important anatomic structure—the bony bridge—extending over the roof of the epitympanum and beneath which the aditus passes to the antrum of the mastoid process.

In the presence of cholesteatoma and/or erosions as from granulation tissue, tympanosclerosis, and/or polyps, definite changes in this structure can be demonstrated. This was documented by the Exhibitor.

There was a scientific essay on the same subject.

The Importance of Contrast Material Absorption in the Evaluation of the Nonvisualized Gallbladder. Ronald G. Evens, M.D., Coretta Schroer, M.D., and P. Ruben Koehler, M.D., Mallinckrodt Institute of Radiology, St. Louis, Missouri.

Nonvisualization of the gallbladder with oral cholecystography is not always indicative of gallbladder disease but can be due to inadequate intestinal absorption of the contrast material in the colon.

Models have been created to aid in the evaluation of the amount of residual contrast material present in the colon.

It was shown by the Exhibitors that when significant residual was present in the colon it was usually possible to visualize the gallbladder on the following day.

The Many Facets of Ischemic Colitis. David Faegenburg, M.D., Harold Chiat, M.D., Heywood Y. Epstein, M.D., Perry R. Mandel, M.D., and Stuart T. Ross, M.D., Nassau Hospital, Mineola, New York.

The Exhibitors have examined 13 cases which fulfilled the clinical and radiologic criteria for ischemic colitis. A wide age group was represented, ranging from 26 to 68 years.

Although most were idiopathic, 2 cases appeared related to oral contraceptives, 1 to occult carcinoma of the pancreas, 1 to atherosclerotic occlusion of the inferior mesenteric artery and 1 to volvulus of the sigmoid colon.

While the characteristic "thumbprints" were often present, much smaller digitations were also considered diagnostic.

Neurogenic Pulmonary Edema. Alvin H. Felman, M.D., University of Florida Hospital, Gainesville, Florida.

Pulmonary edema resulting from intracerebral abnormality was the subject of this exhibit.

Cases were presented illustrating this unusual complication of neurologic disease.

The differentiation of this entity from the other causes of pulmonary edema is important for the institution of correct therapy.

Angiography in Rare Renal and Perirenal Lesions. John Fenlon, M.D., L. Silber, M.D., and P. Ruben Koehler, M.D., The Edward Mallinckrodt Institute of Radiology, St. Louis, Missouri.

The 3 groups of lesions presented in this exhibit were: Group I, intrarenal masses; Group II, extrarenal masses; and Group III, nonfunctioning kidneys.

The 3 signs serving to suspect tumors consisted of: (1) relatively orderly arrangement of vessels; (2) primary blood supply from other vessels than renal; and (3) early appearing, rapidly accumulating stain.

Seven cases formed the material of the exhibit. The pertinent findings in each case were presented.

Pulmonary Interstitial Emphysema in the Newborn. Barry D. Fletcher, M.D., Eugene W. Outerbridge, M.D., and J. Scott Dunbar, M.D., Montreal Children's Hospital, Montreal, Quebec, Canada.

This exhibit demonstrated the roentgen findings in pulmonary interstitial emphysema in 5 newborn patients.

Four of the patients were premature in-

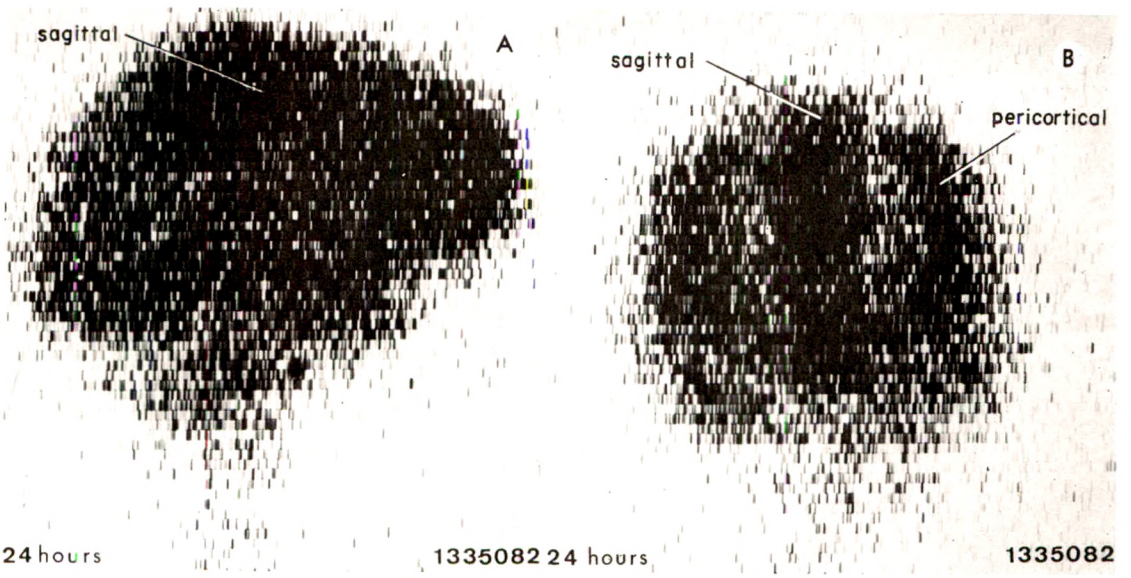


FIG. 4. (*A*) Lateral CSF scan (24 hours). Generalized radioactivity over cerebral hemispheres. Concentration of radioactivity in sagittal area. (*B*) Anterior CSF scan (24 hours). Concentration of activity in parasagittal region.

lateral ventricles does not appear as nearly concentrated in the focal plane. The superior border of the radioactivity in the lateral ventricles has a "rounded" configuration (Fig. 8*C*) and the temporal horns are seen.

The optional vertex view is obtained with the patient in the prone position with the neck hyperextended (Fig. 9, *A* and *B*). The canthomeatal line is as closely parallel to the face of the detection device as possible. This view is not difficult to obtain, but does require patient cooperation. The face of the detection device is placed in an orientation so that the sylvian cisterns are separated from the more posterior communicating pathways. The image may be symmetrically divided by radioactivity in the sagittal area.

Another view that separates the communicating CSF pathways is the isotopic "half axial" view. Simulation of the radiographic half axial projection is most easily accomplished with the scintillation camera because the face of the detection device can be angled. To orient the anatomic structures to approximate this radiographic projection,[32] the surface of the detection device is placed anterior in the frontal area at an angle of 30° with the canthomeatal line orienting the detection surface toward the feet. This results in a radioisotopic view which is a combination of a radiographic postero-anterior and half axial projection (Fig. 10, *A* and *B*). Using a perpendicular line drawn to the center of the detection face as a reference point, the skull can be angled by flexing the patient's head to simulate the 20-25° tube angle. At present, the clinical usefulness of the separation of the communicating cisterns during cisternography has not been fully determined, but simulation of the pneumoencephalographic anatomy aids in correlative studies.[16] We are presently analyzing each view to ascertain if specific communicating pathway abnormalities can be detected, postulating that in the various forms of communicating hydrocephalus, identification of specific areas of cessation of CSF flow is essential.[16]

ANATOMIC CONSIDERATIONS

Only the larger CSF compartments will be delineated. With present imaging devices the lower limit of resolution is approximately 2 cm. Therefore, cisterns of

fants with hyaline membrane disease in whom chest roentgenograms showed the abrupt onset of a striking "bubbly" lung pattern due to interstitial emphysema which resulted in death.

A fifth patient with meconium aspiration syndrome, who survived, developed large cysts resembling pneumatoceles during resolution of the interstitial emphysema.

Ovarian Angiography. Richard E. Frates, M.D., Rhode Island Hospital, Providence, Rhode Island.

Successful ovarian arteriography is an important component in the angiographic evaluation of the female pelvis. Selected cases illustrated its value.

The ovary can be outlined and even avascular masses defined. The ovarian contribution to the uterine circulation and the resulting distinctive and definitive opacification of the uterus were emphasized.

Preoperative Angiography in Pediatrics. Richard E. Frates, M.D., Frank DeLuca, M.D., and Conrad Wesselhoeft, M.D., Departments of Radiology and Surgery, Division of Pediatric Surgery, Rhode Island Hospital, Providence, Rhode Island.

The value, technique and results of preoperative angiography in pediatric surgical practice were illustrated.

Selected cases demonstrated how angiography can: (1) establish a diagnosis requiring urgent surgery; (2) allow preservation of a vital organ; (3) determine the operative approach; and (4) help exclude need for surgical intervention.

Contoured X-Ray Filtration: A Means to Improve Roentgenograms and Decrease Patient Radiation Exposure. Gerald Freedman, M.D., Michael Febbroriello, R.T., and Jack Lontz, B.S., Yale-New Haven Medical Center, New Haven, Connecticut.

A wedge-shaped aluminum filter interposed between the patient and the radiation beam compensates for the increased midline absorption in abdominal roentgenography.

Using this filter for intravenous pyelography, improved results were obtained, with an associated 21 per cent decrease in radiation exposure.

Angiography of Trauma. Whei-Rung Fu, M.D., and William W. Stanbro, M.D., The George Washington University Hospital, Washington, D.C.

Percutaneous retrograde arteriography is a simple and safe procedure in the hands of a well trained angiographer. It is very valuable in patients with acute trauma. The information gained from arteriography is not available through any other preoperative diagnostic procedure.

In this exhibit selected cases of acute traumatic laceration of the aorta and brachiocephalic vessels were illustrated, as well as cases of renal and splenic trauma.

Arthrograms of the Wrist in Rheumatoid Arthritis. Margaret O. Harrison, M.D., Chitranjan Ranawat, M.D., and Robert H. Freiberger, M.D., Hospital for Special Surgery, New York, New York.

Rheumatoid arthritis primarily affects synovial tissues with secondary destruction of cartilage and bone.

The wrist arthrogram reveals the gross synovial and soft tissue abnormalities and the early phases of cartilage destruction not visible on routine roentgenograms. It leads to a better understanding of the type and progress of the destructive changes and facilitates the interpretation of routine roentgenograms.

Wrist arthrograms play no major role in the diagnosis or differential diagnosis of rheumatoid arthritis, but are of value when synevectomies are planned.

There was a scientific essay on the same subject.

The Evolution of a Technical System for Coronary Angiography and Ventriculography. Donald C. Jackson, M.D., Edward L. Lansdown, M.D., and David Mymin, M.D., St. Boniface General Hospital and University of Manitoba, Winnipeg, Manitoba, Canada.

In the past 5 years over 500 selective coronary angiographies and ventriculographies were performed at the St. Boniface General Hospital.

small volume which contain little CSF and, hence, a small amount of radioactivity at cisternography, are below the limits of spatial resolution and will not be identified as separate structures. Cisterns such as the cisterna veli interpositi and superior cerebellar cisterns are examples.

By the time of the initial cisternographic imaging in 1 to 3 hours (as a compromise we obtain our initial study at 2 hours), radioactivity is present in the basal cisterns as well as in the spinal region of the neck. The various anatomic areas are separated in the lateral projection. The anatomic structures that can be seen with certainty in a single lateral projection are the spinal CSF canal, pontine, interpeduncular, and suprasellar cisterns (Fig. 2*A*). Although the radiopharmaceutical is present in the quadrigeminal plate and ambient cisterns, multiple views (anterior, posterior and vertex) may be necessary to separate these structures from other areas of superimposed radioactivity. Radioactivity within the sylvian cisterns is superimposed upon the suprasellar cisterns in the lateral view. However, medial activity should be searched for and the more inferior narrower zone of radioactivity in the ambient cisterns should be differentiated from ventricular radioactivity which will extend laterally and be more superior in location (Fig. 8, *B* and *C*).

The 6 hour examination will normally

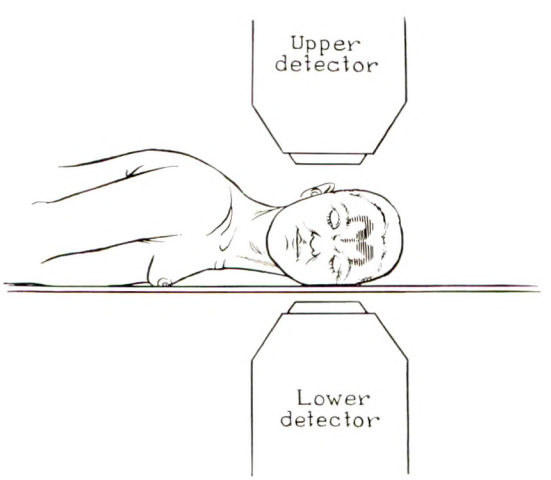


Fig. 5. Position for lateral view.

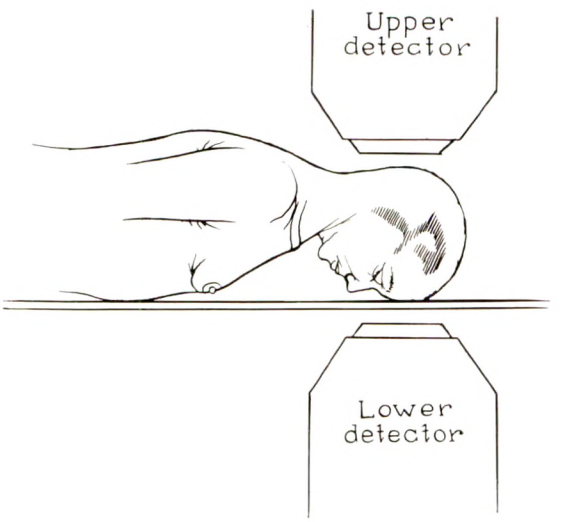


Fig. 6. Position for anterior and posterior views.

show the more dorsal CSF cisterns, such as the quadrigeminal plate cistern, optimally.[7] There will usually be a beginning of diffuse movement of activity over the cerebral cortex (Fig. 3*A*); the sylvian cisterns will be seen as a dense band of radioactivity extending laterally in the anterior view (Fig. 2*B*; and 3*B*). Radioactivity in other communicating channels (ambient cisterns and cisterna lamina terminalis) will be noted anteriorly and posteriorly, but concentrated nearer the base than the vertex of the skull. The normal 24 hour examination will show diffuse activity over the cerebral cortex and concentration of radioactivity in the sagittal region. This concentration is seen superiorly as a dense line of radioactivity (Fig. 11, *A* and *B*). The delayed examination reveals increased localization of this parasagittal activity and diminished activity over the cerebral convexity and in the basal cisterns. In older patients, movement of CSF is generally slower but there should be concentration of radioactivity in the parasagittal area by 24 hours. If this concentration is not present, 48 and 72 hour examinations should be obtained and the earlier studies reviewed to (a) search for evidence of an improper injection (epidural or subdural), and (b) to detect specific anatomic blocks to CSF movement.

Initially these were performed on a standard moving x-ray table top with 16 mm. cine filming supplemented by 10×8 inch spot roentgenograms.

Because of the technical difficulties involved in patient mobility and catheter stability with the Sones technique, a special C-arm image intensification system has been developed. The information is recorded on 70 mm. spot films, 35 mm. cine film, video tape, and biplane 16 mm. film.

The results obtained with this equipment were illustrated and evaluated.

Intercostal Bulging in the Emaciated Patient without Emphysema. Kenneth R. Kattan, M.D., Harold B. Spitz, M.D., and Myron Moskowitz, M.D., University of Cincinnati Medical School, Cincinnati, Ohio.

Intercostal pulmonary bulging was found in emaciated patients. Autopsy report of the same patients showed no emphysema. Scalloping was seen on the outer surface of the chest wall corresponding to the intercostal spaces that showed the pulmonary bulging.

Intercostal bulging (or scalloping) developed in a patient suffering from carcinoma of the esophagus after he became emaciated. Again, no emphysema was seen at autopsy.

Case of the Day. Kenneth L. Krabbenhoft, M.D., Wayne State University, Detroit General Hospital and Harper Hospital, Detroit, Michigan.

An interesting adult roentgenologic diagnostic problem was presented daily with an Award given for each correct diagnosis, posted on the following day.

Re-Evaluation of the P^{32} Eye Tumor Identification Test. James H. Larose, M.D., John M. Palms, Ph.D., and William S. Hagler, M.D., Emory University School of Medicine, Atlanta, Georgia.

The P^{32} eye tumor identification test has shown no false negatives in the Exhibitors' hands. This is because the detector is placed directly over the lesion.

Since the majority of melanomas are posteriorly located, this often entails incision of the conjunctiva.

Semiconductor detectors are currently being evaluated. These have the theoretic advantages of pulse height analysis, minification and reduction of the P^{32} patient dose.

Compression Mammography of the Pendant Breast. Harold J. Lasky, M.D., 55 East Washington, Chicago, Illinois.

Breast suspension provides maximum coverage. Positioning is fast, accurate and anatomic. Oblique views are facilitated. Adequate compression insures reduced uniform thickness.

A device, illustrative mammograms and a technique were exhibited.

Histoplasmosis: Clinical and Roentgenographic Manifestations. Kyo R. Lee, M.D., David L. Hall, M.D., and Lewis J. Garrotto, M.D., University of Missouri School of Medicine, Columbia, Missouri.

There are few reports describing extrapulmonary manifestations of histoplasmosis in the radiologic literature.

The Exhibitors are from an endemic area where up to 90 per cent of the adult population reacts positively to histoplasmin. They comprehensively summarized the clinical and roentgenographic manifestations in various organs.

Roentgenologic Features of Regional Enteritis of the Upper Gastrointestinal Tract. David A. Legge, M.D., Harley C. Carlson, M.D., and Edward S. Judd, M.D., The Mayo Clinic, Rochester, Minnesota.

The material of this exhibit was from a review of the cases of regional enteritis seen at the Mayo Clinic since 1964. There were 11 cases of involvement of the upper gastrointestinal tract, 1 of the esophagus, 2 of the stomach, 5 of the stomach and duodenum, and 3 of the duodenum.

The roentgenologic findings were demonstrated.

Some of these cases were difficult diagnostic problems; *e.g.*, where the distal involvement by regional enteritis did not give rise to symptoms and where the stomach, but not the duodenum, was involved.

There was a scientific essay on the same subject.

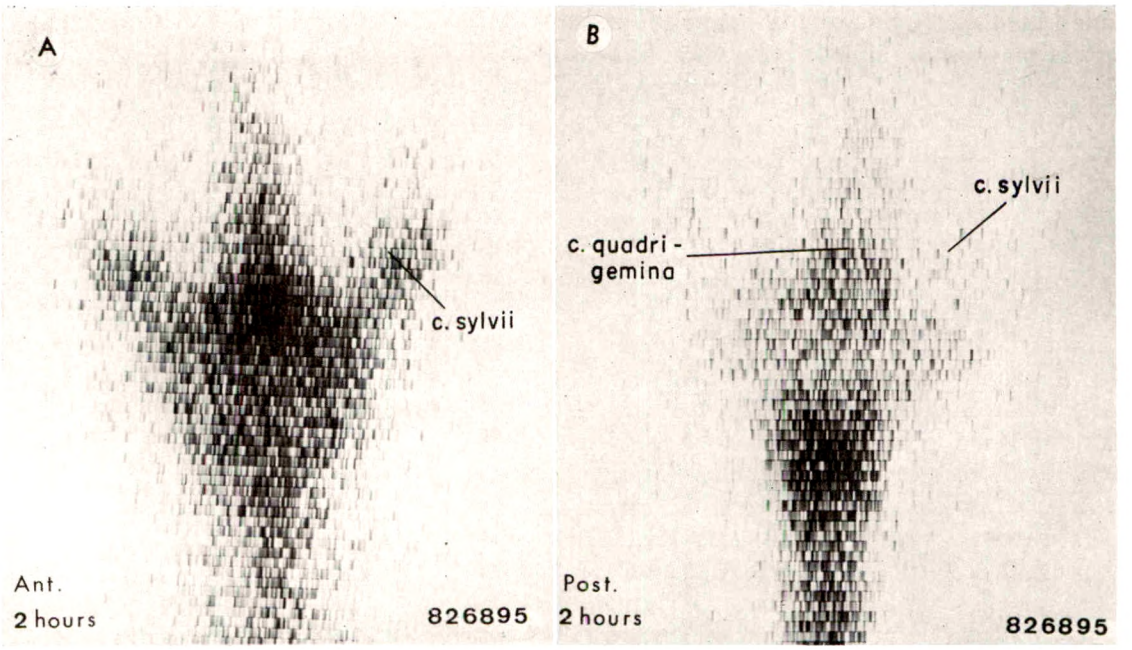


FIG. 7. (*A*) Anterior and (*B*) posterior CSF scans (2 hours). Sylvian cisterns more optimally visualized in anterior view; quadrigeminal cistern in posterior view.

Improper injection, if not recognized, can lead to gross misinterpretations regarding CSF movement. Although generally recognized but repeated for emphasis, removal of CSF does not assure a subarachnoid injection. Both epidural and subdural injections will result in delayed CSF movement. Epidural injections may be detected by imaging the area of injection as the radionuclide will be temporarily confined to the lumbar region or may diffuse out into the soft tissues. One should wait several days following a lumbar puncture and one week following pneumoencephalography before obtaining a cisternogram. A method of detecting improper injections is to instill 5 cc. of air at the time of lumbar injection and obtain a skull roentgenogram after elevating the patient's head.[16,17] The presence of injected air in the cranial area will ensure proper radionuclide injection. If a subdural or epidural injection is detected, the study should be terminated and another cisternography initiated after suitable time lapse to allow radionuclide decay or biologic removal.

The CSF flow patterns in various forms of hydrocephalus are often characteristic. In noncommunicating (obstructive) hydrocephalus the CSF movement will be normal or slightly delayed. As one would expect, no radioactivity appears in the ventricular system. Communicating hydrocephalus may reveal one of several patterns depending upon the type present. If the etiology of the communicating hydrocephalus is principally cerebral atrophy ("hydrocephalus ex vacuo") then radioactivity will only occasionally enter the ventricular system, there will be normal to slightly delayed CSF flow over the cerebral hemispheres and concentration of radionuclide in the parasagittal region. Communicating hydrocephalus due to a specific obliteration of the CSF space delaying CSF movement will manifest a concentration of radionuclide at the anatomic location of the obstruction. The third form of communicating hydrocephalus is often referred to as occult hydrocephalus. In this form there is no specific block to CSF flow demonstrated nor is the etiology primarily cerebral atrophy. "Normal pressure hydrocephalus" is an example of the type in which the radio-

Radiology of the Hip with Traction; The Poor Man's Arthrogram. William Martel, M.D., Andrew K. Poznanski, M.D., and Lawrence R. Kuhns, M.D., The University Hospital, Ann Arbor, Michigan.

Traction usually produces a "crescent of gas" in the joint space ("vacuum phenomenon") delineating the articular cartilage.

If the joint contains fluid, traction with the patient supine causes widening of the interosseous space without such a crescent of gas.

It was demonstrated that this simple technique provides a sensitive, reliable method of detecting joint fluid and evaluating the articular cartilage.

Trimethobenzamide: An Antiemetic in Radiation Therapy. Raymond Marty, M.D., Tumor Institute of the Swedish Hospital Medical Center, Seattle, Washington.

Using a double-blind technique, trimethobenzamide was compared with placebo in relieving nausea and vomiting in 60 patients undergoing supervoltage roentgen therapy for either intra- or extraabdominal malignancies.

Thirty-one patients with intraabdominal malignancy and 29 patients with extraabdominal malignancy (usually mediastinal) were studied. Each group was further subdivided so that 15 patients received placebo and 15 patients received the medication.

The statistical evaluation of the findings was presented.

Clinical Sonography in Obstetrics and Gynecology. Lajos I. von Micsky, M.D., Nathaniel Finby, M.D., Harold M. M. Tovell, M.D., and Shirley J. Staiano, B.S., Ultrasonic Division of Radiology Department, Woman's Hospital, St. Luke's Hospital Center, New York, New York.

The objective of this exhibit was to present a comprehensive survey of the current clinical uses of ultrasonic imaging techniques in obstetrics and gynecology and in other allied fields.

The illustrative material was selected from sonographic studies performed at the Ultrasonic Division of the Radiology Department during a 6 month period.

Challenge of Cardiac Tumors. R. Moncada, M.D., T. Matuga, M.D., R. Miller, M.D., I. Carr, M.D., A. Halstreiter, M.D., R. Meadows, M.D., and P. Szanto, M.D., Cook County Hospital, Chicago, Illinois.

A critical analysis of the current developments in cardiac tumors was presented based upon experience with 14 primary neoplasms.

Specific attention was given to the diagnostic approach showing electrocardiographic, hemodynamic and angiocardiographic changes in right and left sided cardiac tumors.

The Angiographic Manifestations of Gas Endarterectomy. Harold Moskowitz, M.D., Martin J. Kaplitt, M.D., Sol Sobel, M.D., Philip N. Sawyer, M.D., and Harry Z. Mellins, M.D., State University of New York Downstate Medical Center, Brooklyn, New York.

A new technique of endarterectomy was described.

It utilizes carbon dioxide injected under high pressure to dissect the diseased inner layers of intima and media from the more normal outer adventitia.

This technique has permitted endarterectomy over extremely long distances, with technical ease and marked decrease in operative time.

Follow-up on 100 patients over the past 2 years has revealed an extremely high patency rate in the nondiabetic patient.

MUST–A New Concept in Military Field Hospitalization. Colonel William S. Mullins, MSC, The Historical Unit, US Army Medical Department, Walter Reed Army Medical Center, Washington, D.C.

The exhibit featured MUST (Medical Unit, Self-contained, Transportable), the revolutionary combat support hospital equipment system developed to provide definitive medical treatment for Army casualties in the combat zone.

Official histories of the United States Army Medical Department were also displayed.

Leptospiral Pneumonia. Alex E. Newman, M.D., and Harry Newman, M.D., 24th Evacuation Hospital, APO San Francisco, California 96491.

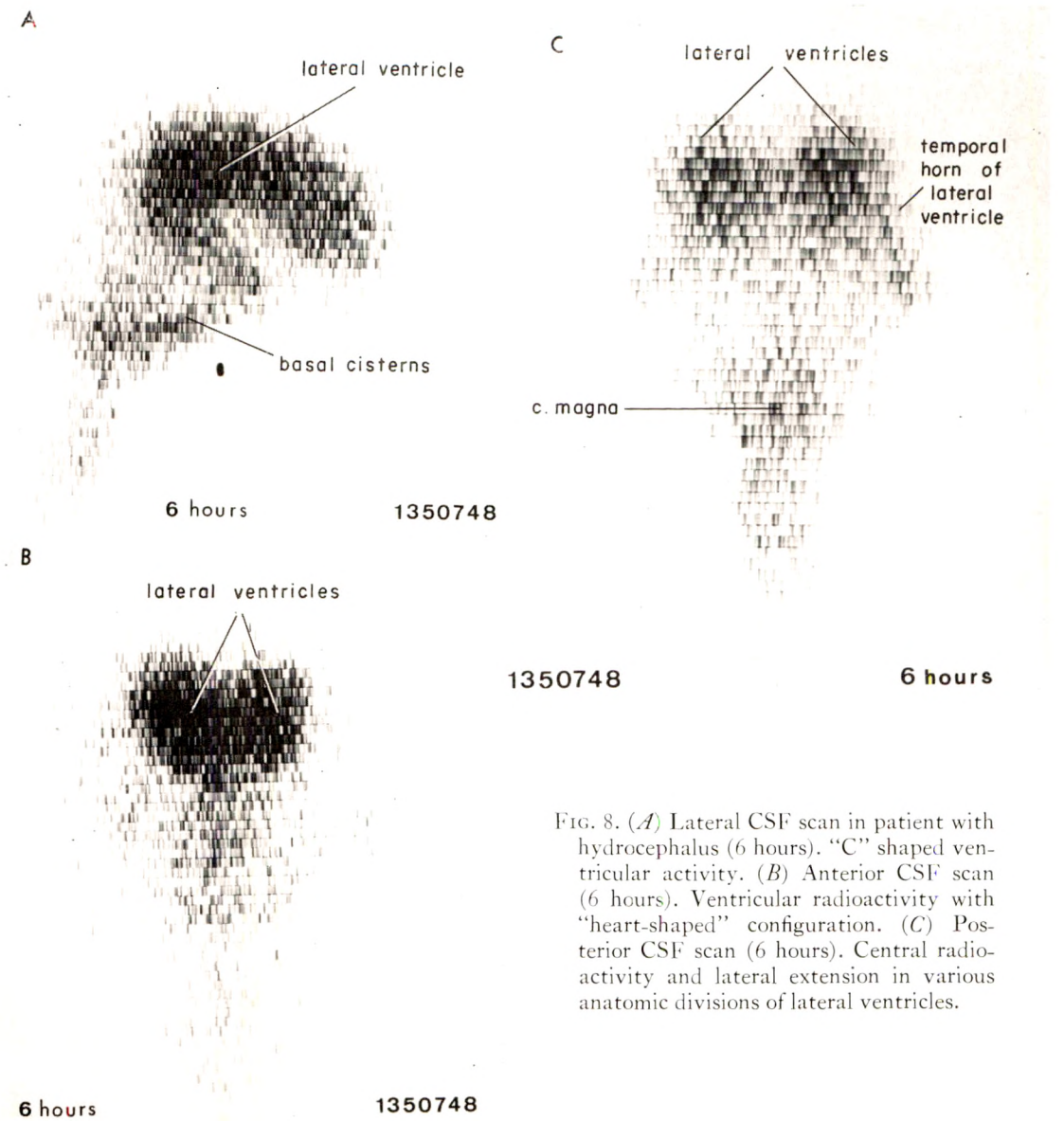


Fig. 8. (*A*) Lateral CSF scan in patient with hydrocephalus (6 hours). "C" shaped ventricular activity. (*B*) Anterior CSF scan (6 hours). Ventricular radioactivity with "heart-shaped" configuration. (*C*) Posterior CSF scan (6 hours). Central radioactivity and lateral extension in various anatomic divisions of lateral ventricles.

nuclide will enter the ventricles and is associated with marked delay in CSF flow over the cerebral cortex and failure of concentration of radioactivity in the parasagittal region.[30] Again it is emphasized that delayed studies are necessary to assess CSF movement and a radiopharmaceutical with a proper effective half life should be employed.

Each radioisotopic view has certain advantages over the others for visualization of specific CSF compartments. Although the anterior and posterior views are similar, the plane of optimal resolution for both the scintillation camera and the rectilinear scanner is different in each view, facilitating selective identification of structures. In these views, the pontine, interpeduncular and inferior portions of the suprasellar cisterns are superimposed. The sylvian cisterns and quadrigeminal plate cisterns are readily identified as they lie respectively lateral and superior to the central concentration of the basal cisterns. The anterior view most

Pulmonary involvement is occasionally seen in leptospirosis and is a manifestation of hemorrhagic pneumonitis. This may occur early and may present several roentgenographic appearances.

A series of such cases seen during the past year in Vietnam were presented.

Leprosy in Children. Harry Newman, M.D., and James DuBois, M.D., Letterman General Hospital, San Francisco, California.

Through the ages, leprosy as a disease has been superstitiously feared and it has been treated with revulsion and prejudice. Even at present, the very word leprosy still arouses unreasonable anxiety.

There are approximately 15 million people with leprosy, mainly in the tropics, and at least 2,000 in the United States.

With the increased travel by both military and civilian personnel to endemic areas, it was the purpose of the exhibit to alert physicians to the possibility of leprosy, because early diagnosis and treatment can prevent most of the disabling complications of the disease.

Congestive Heart Failure in the Newborn. Barry M. Potter, M.D., University of Cincinnati Medical Center & Children's Hospital, Cincinnati, Ohio.

A simple classification of disorders causing congestive heart failure in the newborn based on presence or absence of cyanosis and heart murmurs was presented.

Angiocardiographic approach was discussed and the angiocardiographic findings in both common and uncommon entities, including transposition of the great vessels, hypoplastic left heart syndrome, endocardial cushion defects, coarctation syndrome, total anomalous pulmonary venous return, anomalous origin of the left coronary artery, persistent truncus arteriosus and pulmonary atresia with intact ventricular septum were illustrated.

Hand Changes in Sarcoidosis. Pedro P. Purcell, M.D., Jack W. Bowerman, M.D., A. Everette James, Jr., M.D., and Martin W. Donner, M.D., The Johns Hopkins Medical Institutions, Baltimore, Maryland.

The roentgenographic features of involvement of the hands in sarcoidosis were presented. The Exhibitors' large experience was from the Sarcoid Clinic of the Johns Hopkins Hospital.

The changes of osseous lesions with and without therapy were emphasized.

Epiphyseal Injuries: Diagnostic and Prognostic Considerations. Lee F. Rogers, M.D., University of Texas Medical School, San Antonio, Texas.

The gross and microscopic anatomy of the epiphyseal complex and its blood supply were reviewed. The mechanism, pathophysiology and classification of epiphyseal injuries were presented.

Radiographic findings of diagnostic and prognostic significance were emphasized. Finally, the prognosis was related to the classification of these injuries.

Vein Graft for Coronary Disease. Charles E. Schmidt, M.D., and Adam Fueredi, M.D., St. Luke's Hospital, Milwaukee, Wisconsin.

This exhibit illustrated preoperative disease and postoperative correction of coronary arteriosclerosis, using saphenous vein graft.

The procedure consists of anastomosis from aorta to all major branches of the coronary systems.

Remotely Controlled Compression in the Examination of the Upper Gastrointestinal Tract. Gerhart S. Schwarz, M.D., New York Medical College, Center for Chronic Disease, Bird S. Coler Hospital, Welfare Island, New York, New York.

Two years' experience with a modern remotely controlled ringstand has caused the Exhibitor to change his method of examination of the upper gastrointestinal tract.

The remote control compression device, which had been installed in the factory, was found to be unsatisfactory. A special remote control compression device with optical beam alignment was developed.

The model of this device and some of the roentgenographic results were shown.

Afterloading Treatment of Endometrial Cancer; Modification of Heyman Applicators. Norman Simon, M.D., Sidney M.

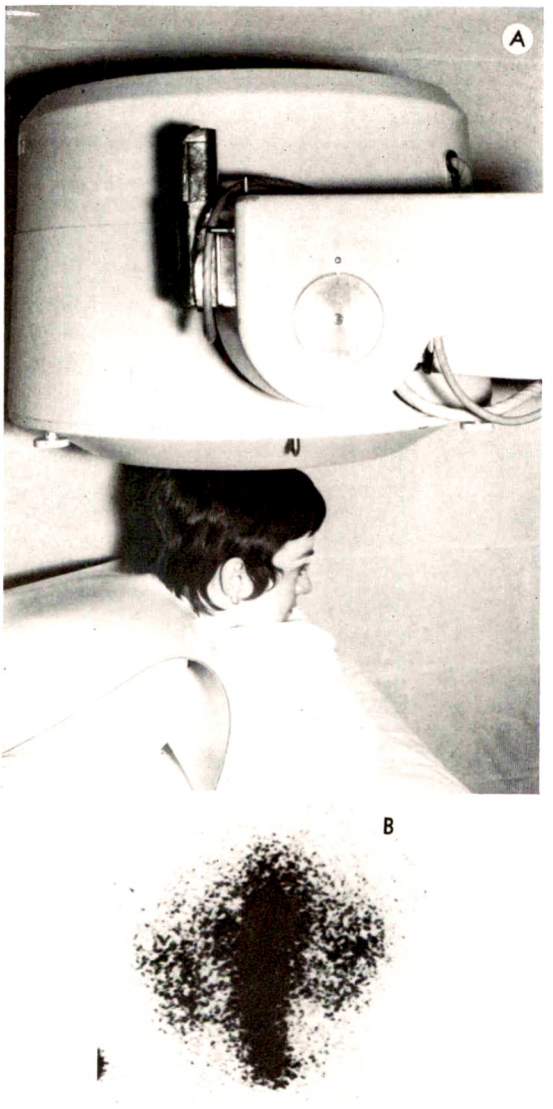


FIG. 9. (*A*) Position for vertex view. (*B*) Vertex CSF scan (24 hours). Central band of radioactivity in parasagittal area. Lateral concentration in sylvian cisterns.

optimally visualizes the sylvian cisterns, whereas the posterior view better demonstrates the quadrigeminal plate cisterns. Ambient cisterns are only identified by location in these views and are not separated from radioactivity in adjacent or superimposed structures.

In the posterior and anterior projections, the cerebellopontine angle cisternal activity is superimposed upon that of the sylvian cisterns. For this reason, we have utilized the vertex and half axial views in an effort to delineate the cerebellopontine angle cisterns as well as the communicating pathways surrounding the brain stem. In these optional views the sylvian cisterns which are anterior and superior are separated from the cerebellopontine angle cisterns posteriorly and inferiorly (Fig. 9*B*; and 10*B*). The vertex view orients the sylvian cistern in such a manner that it can be seen for most of its length and separates the sylvian from the basal cisterns (Fig. 9*B*; and 10*B*). Again, the cerebellopontine angle cisterns are suggested by a lateral extension of activity from the basal cisterns that is separate from and posterior to the sylvian cisterns. The vertex view is excellent for verification of a suspected unilateral abnormality or variation.

As was previously discussed, cisternography is often performed to differentiate noncommunicating from communicating hydrocephalus.[15–17,30] This differentiation

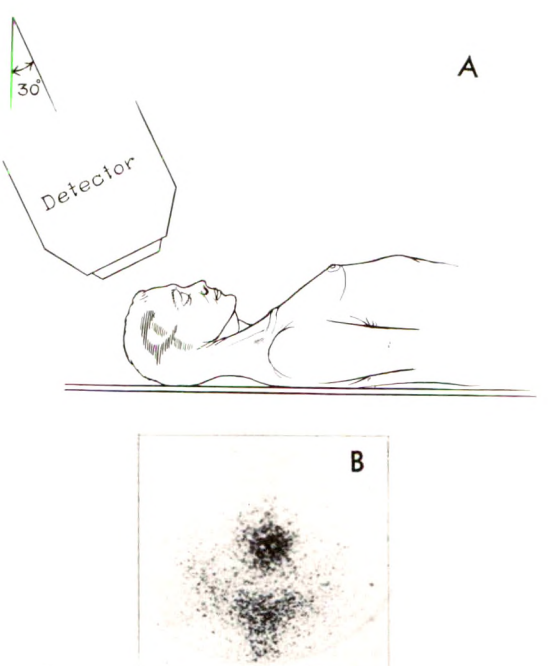


FIG. 10. (*A*) Position for half axial view. (*B*) "Half axial" CSF scan (6 hours). This view separates the quadrigeminal cistern from the basal cisterns and the sylvian cisterns.

Silverstone, M.D., and Lawrence Roach, M.D., The Mount Sinai School of Medicine, New York, New York.

Multiple capsules of radium (Heyman applicators) have been used effectively for decades in the treatment of cancer of the corpus uteri.

The Heyman applicators have the disadvantages of exposure of personnel to radiation and limited accuracy because of haste in handling radioactive material.

Plastic capsules with hollow thin tubing allow afterloading of miniaturized sources of Cs^{137} and Co^{60} with safety and accuracy.

Evaluation of Renal Trauma by Infusion Urography. Robert H. Smalley, M.D., University of New Mexico School of Medicine, Albuquerque, New Mexico.

Forty patients with traumatically induced hematuria at the University of New Mexico Affiliated Hospitals were studied by infusion urography as a part of the immediate roentgenographic evaluation.

Eighty-three per cent had complete visualization of the renal parenchyma, collecting systems and ureters on the initial post-infusion roentgenogram. Six cases had renal trauma and 3 were further evaluated by angiography.

Infusion urography has minimal acceptable side effects, and more hazardous procedures, such as retrograde urography, can be avoided.

Double Contrast Arthrography of the Knee; Experience with 500 Examinations. T. W. Staple, M.D., and H. J. Llewellen, M.D., Mallinckrodt Institute of Radiology, Washington School of Medicine, St. Louis, Missouri.

Five hundred examinations of the knees have been performed with surgical proof in more than one-half of the cases.

In the exhibit both meniscal and extrameniscal lesions were demonstrated and the technique was illustrated.

Role of Mammography in Reduction of Mortality Rate from Breast Cancer Through Periodic Mass Screening. Philip Strax, M.D., Sam Shapiro, B.S., and Louis Venet, M.D., Health Insurance Plan of Greater New York, New York, New York.

Preliminary data from the periodic mass screening for breast cancer conducted by the Health Insurance Plan of Greater New York under contract with the National Institutes of Health indicate a greater than one-third reduction in mortality rate in the study group compared to the controls.

In a randomized group of 60,000 women with 30,000 in the study group and a matched control, detection rate was 2.72/1,000 on initial screening and 1.45/1,000 subsequently compared to 1.51/1,000 in the controls.

The role of mammography as well as clinical examination in the mortality reduction was assessed.

There was a scientific essay on the same subject.

Vascular Patterns in Congenital Heart Disease: A Diagnostic Scheme. Leonard E. Swischuk, M.D., Children's Memorial Hospital, Oklahoma City, Oklahoma.

The key to the roentgenographic interpretation of congenital heart disease lies in the assessment of the pulmonary vascularity. Whether the vascularity is increased, decreased, or normal will allow one to categorize a lesion into: (1) those with left to right shunt; (2) those with right outflow tract obstruction; and (3) those with valvular or vascular disease of the left side of the heart (with or without pulmonary venous obstruction).

A scheme for the orderly categorization of these vascular patterns was developed, the emphasis being on identification and assessment of pulmonary vascularity rather than on a detailed study of any one abnormality (*i.e.*, if you see Pattern 1, you should consider certain lesions, while if you see Pattern 2, a totally different group must be considered, etc.).

In addition to this categorization, the different vascular patterns were explained in terms of pathophysiology, for without such a correlation, interpretation of the roentgenographic patterns is less than de-

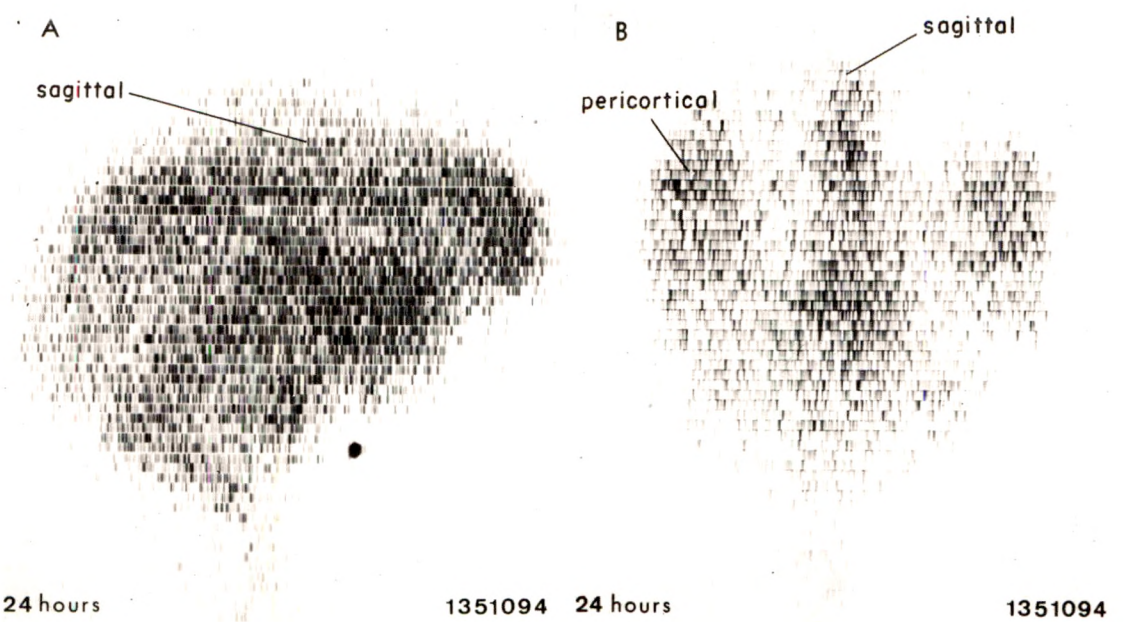


Fig. 11. (*A*) Lateral and (*B*) anterior CSF scans (24 hours). Parasagittal radionuclide concentration seen as dense line of radioactivity.

depends upon detection of radioactivity in the ventricular system. The lateral ventricles are more easily identified than the third or fourth ventricles which cannot be separated from radioactivity in the basal cisterns. In the lateral view the lateral ventricles are seen as "C-shaped" structures containing radioactivity that extend much farther anteriorly than radioactivity in the quadrigeminal plate cistern with which it may be confused. The radioactivity in the lateral ventricles appears as a much broader band of activity than that of the callosal cistern which is in the same general location in this view.

Occasionally, in patients with communicating hydrocephalus, identification of the lateral ventricles is difficult when the lateral ventricles are markedly abnormal in their configuration or location. In infants and young children, the lateral ventricles appear more inferior in their location than in the adult and the temporal horns are not as clearly delineated. The lack of delineation of the temporal horn may be due to the fact that the anatomic structures in the children are not adequately separated for optimum resolution. Lateral ventricular activity can be properly identified by simultaneous analysis of radioactivity in the lateral and anterior views. Although the characteristic "C-configuration" of the radioactivity in the lateral view will usually identify the lateral ventricles, when there is uncertainty, attention to the central radioactivity in the anterior view will confirm the correct anatomic location. The lateral ventricles will not be separated on the anterior scan, but the radioactivity will appear superior to and wider than the normal radioactivity in the quadrigeminal plate and ambient cisterns. Importance of correctly identifying radioactivity in the ventricular system cannot be overemphasized, for cisternographic diagnosis of communicating hydrocephalus rests upon this observation.

Conclusion

Cisternography is an excellent method to depict CSF compartments and to document CSF flow. Multiple views are necessary to accurately depict the anatomic configuration of a cerebrospinal fluid com-

sirable—a total roentgenographic-pathophysiologic approach was emphasized.

The Adult Symphysis Pubis—Normal and Abnormal. Vernon A. Vix, M.D., and Chi Yol Ryu, M.D., Vanderbilt University and Veterans' Administration Hospitals, Nashville, Tennessee.

Abnormalities of the symphysis pubis may be the result of local or systemic diseases.

Because this area is so often seen on roentgenograms taken for other purposes, it may at times suggest or confirm a diagnosis.

The normal anatomy of the adult symphysis pubis has been studied and illustrations of various disease processes were presented. In some instances, the findings were relatively specific and in others a useful differential diagnosis was apparent.

There was a scientific essay on the same subject.

Quantitating Bone Mineral Using an External Gamma Ray Source. John M. Vogel, M.D., Ronald J. Friedman, M.D., and Jerry T. Anderson, B.S., USPHS Hospital, San Francisco, California.

Bone mineral content may be measured by using x-ray densitometry or the gamma ray absorption technique.

The Exhibitors' modification of the latter method permits not only accurate quantitation of bone mineral content, but also rapid visualization of the results.

It is the method proposed to study bone mineral changes in future Apollo flights.

Evaluation of the Transplanted Lung with Pulmonary Angiography and Xenon 133 Scanning. E. L. Walker, M.D., P. Hutchin, M.D., and O. F. Gabriele, M.D., University of North Carolina School of Medicine, Chapel Hill, North Carolina.

The sequential changes occurring in pulmonary angiograms and xenon 133 scans during unmodified canine lung allograft rejection were compared.

In angiograms, there is a minimal prolongation of the duration of the arterial phase and changes in angioarchitecture of the graft 4 to 6 days after transplantation.

In lung scans, there is an accompanying decrease in ventilation and perfusion.

Pulmonary scanning after intravenous xenon 133 administration provides a convenient method for continuous evaluation of the functional changes in the transplanted lung, while angiography is reserved for the differential diagnosis of impaired function.

Significance of Unilateral Absence of Pulmonary Artery Perfusion by Lung Scanning. Robert I. White, Jr., M.D., A. Everette James, Jr., M.D., and Henry N. Wagner, M.D., The Johns Hopkins Medical Institutions, Baltimore, Maryland.

Analysis of one year's experience with 607 lung scans revealed 13 cases (2 per cent) of unilateral absence of perfusion.

The causes were: bronchogenic carcinoma (3), massive pulmonary embolism (3), "acquired" hyperlucent lung (Swyer-James) (1), inflammatory disease of lung (3), and surgical removal of the lung (1). Shunting procedure for congenital heart disease (2/18) and the Swyer-James syndrome (1/3) were the most common disease entities associated with unilateral absence of radioactivity on a percentage basis.

The information provided by chest radiography, and lung scanning was presented and the usefulness of this correlation was demonstrated.

Computer Aided Diagnosis of Gastric Ulcer. William J. Wilson, M.D., David Teates, M.D., and Roger K. Harned, M.D., University of Nebraska Medical Center, Omaha, Nebraska.

The elements of the public model used in computer aided diagnosis of gastric ulcers were displayed. Line drawings and roentgenograms were used to define the features found to be important. This technique permitted a diagnostic accuracy of greater than 97 per cent.

Opportunity for self-evaluation in actual case analysis was an integral part of this exhibit.

partment, thus making its identification possible. We recommend as a routine both lateral, anterior and posterior views in each study. Whether or not the addition of the vertex scan should be a matter of routine has not been determined, but should probably be reserved for the problems involving unilateral abnormalities and questions of anatomic variation. When cisternography is performed in the presence of suspected hydrocephalus, identification of radioactivity within the ventricular structures confirms the diagnosis of communicating hydrocephalus as a result of defective CSF resorption, and delayed studies with particular reference to concentration of radioactivity in the parasagittal area are essential. The location of a specific block to CSF flow may be identified by attention to the anatomic CSF compartments visualized on the cisternographic study.

The introduction of radioactive substances into the cerebrospinal fluid space is not a new procedure, but recent progress in imaging techniques and development of new radiopharmaceuticals has greatly increased the anatomic detail contained in the radioisotopic images and has allowed identification of specific CSF pathways and compartments. Numerous reports of the efficacy of surgical CSF shunting procedures in obstructive and normal pressure hydrocephalus have stimulated exploration of new methods in an attempt to select those patients who will benefit from these shunting procedures. Intrathecal injection of radiopharmaceuticals with subsequent imaging offers an innocuous and physiologic method to study cerebrospinal fluid dynamics.

Cisternography may be used to: (1) determine the various forms of hydrocephalus; (2) separate the types of porencephalic cysts and subarachnoid cysts; (3) examine patients with pseudotumor cerebri and patients with meningocele; (4) as well as predict those patients who will benefit from CSF diversionary shunts.

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THE TECHNICAL EXHIBITS

THE TECHNICAL EXHIBITS of the Seventy-first Annual Meeting of the American Roentgen Ray Society held at the Deauville Hotel, Miami Beach, Florida, September 29–October 2, 1970, again surpassed all expectations in excellence and artistic visual display. They were housed in the spacious Napoleon Rooms No. 1, 2 and 3, where, utilizing to the fullest extent the ultramodern engineering facilities of the Deauville Hotel, they reflected magnificently the tremendous advance made in the technical aspect of Radiology during the past few years.

To Mr. Clifford L. Sherratt, Honorary Member, and Dr. Ernst F. Lang, Manager of the Annual Meeting, the Society expresses its gratitude for organizing these superb exhibits which contributed immeasurably to the over-all success of the Annual Meeting.

The Society also expresses its sincerest thanks and deepest appreciation to the following firms which took part in the Technical Exhibits:

Acan X-Ray, Detroit, Michigan; Ames Color-File Corporation, Somerville, Massachusetts; Atomic Energy of Canada Limited, Ottawa, Ontario, Canada; Automatic Seriograph, Division of Litton Industries, Des Plaines, Illinois; Barber-Colman Company, Rockford, Illinois; Barnes Engineering Company, Stamford, Connecticut; Barnes-Hind Barium Products, Sunnyvale, California; Bar-Ray Products, Incorporated, Brooklyn, New York; John Bunn Corporation, Alden, New York; Cenco Medical/Health Supply Corporation, Miami, Florida; Cook, Incorporated, Bloomington, Indiana; Cordis Corporation, Miami, Florida; Dunlee Corporation, Bellwood, Illinois; Eastman Kodak Company, Rochester, New York; E. I. Du Pont de Nemours and Company, Wilmington, Delaware; Eimac—Division of Varian, Salt Lake City, Utah; Elema-Schonander, Incorporated, Elk Grove Village, Illinois; Encyclopaedia Britannica, Incorporated, Chicago, Illinois; Eureka X-Ray Tube Corporation, Division of Litton Industries, Chicago, Illinois; E-Z-Em Company, Westbury, New York; Field Emission Corporation, McMinnville, Oregon; GAF Corporation, New York, New York; General Electric Company, Milwaukee, Wisconsin; Gordon Consultants, Incorporated, New York, New York; Warren H. Green, Incorporated, St. Louis, Missouri; Grune & Stratton, Incorporated, New York, New York; Hoechst Pharmaceutical Company, Cincinnati, Ohio; Hoffrel Instruments, Norwalk, Connecticut; Ilford Incorporated, Paramus, New Jersey; Keleket/CGR Corporation, Waltham, Massachusetts; Liebel-Flarsheim Company, Division of Sybron Corporation, Cincinnati, Ohio; Litton Industries; LogEtronics Incorporated, Huntington Station, Long Island, New York; Low X-Ray Corporation, New York, New York; Machlett Laboratories, Stamford, Connecticut; Mallinckrodt Chemical Works, St. Louis, Missouri; Medico-International Incorporated, Tampa, Florida; Medi-Tech, Incorporated, Watertown, Massachusetts; Medrad Incorporated, Allison Park, Pennsylvania; 3M Company, St. Paul, Minnesota; N & H Instruments Incorporated, Carrollton, Texas; Nuclear Associates Incorporated, Westbury, New York; Nuclear-Chicago, Incorporated, Des Plaines, Illinois; Ohio-Nuclear, Incorporated, Mentor, Ohio; Pako Corporation, Minneapolis, Minnesota; Philips Medical Systems, New York, New York; Picker Corporation, Cleveland, Ohio; Profexray, Division of Litton Industries, Des Plaines, Illinois; Purdue Frederick Company, Yonkers, New York; W. B. Saunders Company, Philadelphia, Pennsylvania; Schick X-Ray Company, Wilmette, Illinois; Frank Scholz X-Ray Corporation, Boston, Massachusetts; Siemens Corporation, Iselin, New

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Jersey; Spectrum X-Ray Corporation, Westville, New Jersey; E. R. Squibb & Sons, Incorporated, New York, New York; Texas Instruments Incorporated, Dallas, Texas; Charles C Thomas, Publisher, Springfield, Illinois; U. S. Catheter & Instrument Corporation, Glens Falls, New York; United States Radium Corporation, Brooklyn, New York; Wellington X-Ray, Tampa, Florida; Williams & Wilkins Company, Baltimore, Maryland; Westinghouse Electric Corporation, Pittsburgh, Pennsylvania; Winthrop Laboratories, New York, New York; Wolf X-Ray Corporation, Jamaica, New York; Year Book Medical Publishers, Chicago, Illinois.

PRIMARY CEREBROSPINAL FLUID FISTULA*

By H. L. FLOYD, M.D.,† H. F. W. PRIBRAM, M.B., D.M.R.D.,† *and* A. G. VELO, M.D.‡
RICHMOND, VIRGINIA

COMMUNICATIONS between the subarachnoid space and a body surface are potentially lethal. Many are in the form of occipital or lumbosacral dermal sinuses, while others are cerebrospinal fluid fistulae. Most of these occur as a result of basal skull fracture with a tear of the underlying dura and arachnoid. A spontaneous cerebrospinal fluid fistula is rare and the first clear description is attributed to Charles Miller in 1826,[6] although Thomas Willis in 1676 and Morgagni in 1762 may have described similar cases.[6]

O'Connell[9] divides spontaneous fistulae into: (1) primary, where the cause cannot be found; and (2) secondary, those that accompany intracranial or sinus neoplasms, sinus infection, or those that follow surgery or trauma.

Most primary cerebrospinal fluid fistulae occur in the cribriform plate, but they have also been described in the middle and posterior fossa,[4] wherein they occur in the tegmen tympani,[7] in the internal auditory canal,[12] or in the fifth nerve canal.[10] They are commoner in the female by a ratio of 10 to 1,[8] and they may defy precise clinical, radiologic or even surgical localization.

Dandy[2] observed that rhinorrhea on one side does not necessarily imply that the fistula is on the same side, nor even in the cribriform plate. He described defects which were bilateral or on the opposite side. Posterior fossa defects with leakage of fluid into the nasopharynx via the eustachian tube also result in rhinorrhea. Profuse rhinorrhea probably indicates communication with the ventricular system or a spacious basal cistern. It is the role of the radiologist to localize such fistulae and to determine their multiplicity before surgery is undertaken.

Two cases are presented in which localization was made preoperatively by radiographic means.

REPORT OF CASES

CASE 1. E. C., a 7 year old Negro male, was hospitalized 3 times for pneumococcal meningitis in the period from September 1965 to October 1967. All 3 attacks followed upper respiratory infections associated with rhinorrhea from both nostrils. There was no rhinorrhea between attacks. In all 3 instances *D. pneumoniae* was identified in smears and cultures of the cerebrospinal fluid. All 3 attacks responded to intravenous antibiotics. No dermal sinus was present.

Skull roentgenograms on the occasion of the second attack in September 1967 were considered normal. A posteroanterior roentgenogram in October 1967, at the time of the third attack, showed opacified ethmoid air cells on the right and a defect in the right cribriform plate. Tomography (Fig. 1) confirmed these

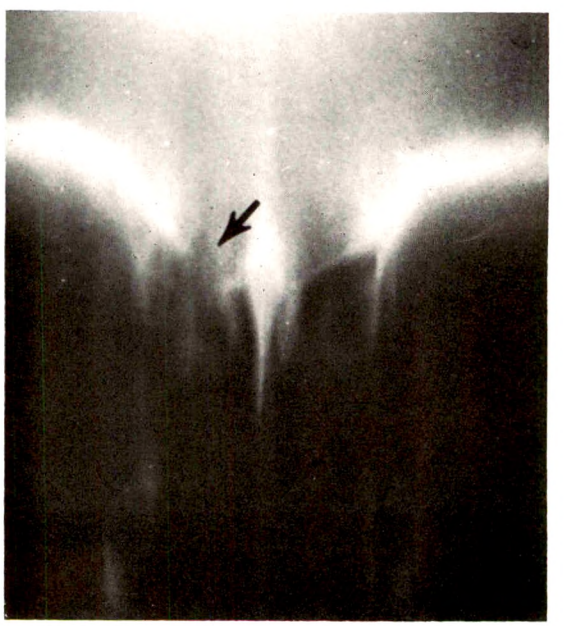


FIG. 1. Frontal tomogram showing defect (arrow) in the right cribriform plate.

* From the Departments of Radiology (Neuroradiology),† and Neurosurgery,‡ Medical College of Virginia, Richmond, Virginia.

NEWS ITEMS

DR. EUGENE P. PENDERGRASS RECEIVES PMS DISTINGUISHED SERVICE AWARD

The Distinguished Service Award of the Pennsylvania Medical Society was presented to Eugene P. Pendergrass, M.D., at the festive State Dinner, held Tuesday evening, October 6, 1970 in Lancaster, Pennsylvania.

Dr. Pendergrass is an illustrious member of the Consulting Editorial Board of the AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE, and for many years has been an exceptionally active and most influential Delegate of the Section on Radiology to the House of Delegates of the American Medical Association.

In receiving the PMS Distinguished Service Award, Dr. Pendergrass is the fourth great Pennsylvania physician thus honored. The other recipients of the Award were: Dr. Jonas Salk, developer of the poliomyelitis vaccine; Dr. John H. Gibbon, Jr., who perfected the heart-lung open surgery machine; and Dr. James Z. Appel, eminent Past President of the American Medical Association.

To quote from the *Pennsylvania Medicine*, September 1970, Volume 73, which is devoted to a tribute to Gene, "I can tell you this, he is truly a great man."

NEW OFFICERS OF THE AMERICAN ROENTGEN RAY SOCIETY

The following new officers were elected at the Seventy-first Annual Meeting of the American Roentgen Ray Society, held at the Deauville Hotel, Miami Beach, Florida, September 29–October 2, 1970: *President*, John F. Roach, M.D., Albany, New York; *President-Elect*, Thomas M. Fullenlove, M.D., San Francisco, California; *1st Vice-President*, Russell H. Morgan, M.D., Baltimore, Maryland; *2nd Vice-President*, William B. Seaman, M.D., New York, New York; *Secretary*, Ted F. Leigh, M.D., Emory University Clinic, Atlanta, Georgia 30322; and *Treasurer*, Colin B. Holman, M.D., Mayo Clinic, Rochester, Minnesota 55901.

The *Chairman of the Executive Council* is Roderick L. Tondreau, M.D., Holy Redeemer Hospital, 1648 Huntingdon Pike, Meadowbrook, Pennsylvania 19046.

The Seventy-second Annual Meeting of the Society will be held at the Sheraton Hotel, Boston, Massachusetts, September 28–October 1, 1971.

SOCIETY FOR PEDIATRIC RADIOLOGY

At the Thirteenth Annual Meeting of the Society of Pediatric Radiology, which was held in the Casanova Room of the Deauville Hotel, Miami Beach, Florida, on September 27–28, 1970, the following officers were elected: *President*, Norman Glazer, M.D., Akron, Ohio; *President-Elect*, Bertram R. Girdany, M.D., Pittsburgh, Pennsylvania; and *Secretary-Treasurer*, John L. Gwinn, M.D., 4650 Sunset Blvd., Los Angeles, California 90027.

The Fourteenth Annual Meeting of the Society will be held at the Sheraton Hotel, Boston, Massachusetts, September 26–27, 1971.

THE CANADIAN ASSOCIATION OF RADIOLOGISTS

Dr. Maurice Dufresne, Chairman of the Committee on Annual Meetings of the Canadian Association of Radiologists announces that the Thirty-fourth Annual Meeting will be held at the Palliser Hotel, Calgary, Alberta, March 16–19, 1971.

The Scientific Program Committee invites radiologists, physicists, etc. to submit papers and scientific exhibits for this meeting.

All communications should be sent to: Chairman, Scientific Program Committee, The Canadian Association of Radiologists, Suite 101, 1555 Summerhill Avenue, Montreal 109, P.Q., Canada.

findings. Tomograms of petrous ridges as well as air encephalograms were normal. Counts of cotton pledgets placed in the nasal cavities at the time of instillation of 15 microcuries of RISA (I^{131}) into the lumbar subarachnoid space were 1,219 cpm on the right and 1,049 on the left at 24 hours. A scan of the head at 24 hours was interpreted as showing leakage of cerebrospinal fluid into the right nasal cavity.

A right frontal craniotomy was performed November 20, 1967, and a 3×4 mm. defect was present in the middle of the right cribriform plate. This was patched with acrylic plastic. The patient tolerated the procedure well and was discharged 10 days later. There has been no recurrence of cerebrospinal fluid rhinorrhea.

Case II. E. S., a 35 year old Negro female, was admitted on November 29, 1968 for investigation of a continuous watery drip from her right nostril. Three weeks previously she had suffered a febrile illness accompanied by lacrimation, bilateral rhinorrhea, sore throat, myalgia and malaise. There was no history of trauma. Physical and neurologic examinations were normal. Upon leaning forward there was a continuous drip of clear watery fluid from her right nostril. The fluid reduced Fehling's solution.

Skull roentgenograms were normal as were tomograms of the petrous ridges and the anterior cranial fossa. Tomograms of the middle fossa showed an opacified right sphenoid sinus (Fig. 2). After injection of 15 microcuries of RISA (I^{131}) into the lumbar subarachnoid space, counts of cotton pledgets in the nose at 60 minutes were 161,000 cpm on the right and 900 on the left. An air study showed an air containing cyst (Fig. 3) just to the right of the midline above the sella. An air fluid level was noted in the sphenoid sinus. The encephalogram was otherwise normal. Positive contrast encephalograms showed immediate opacification of the right sphenoid sinus by pantopaque which entered from the pituitary fossa (Fig. 4, *A* and *B*).

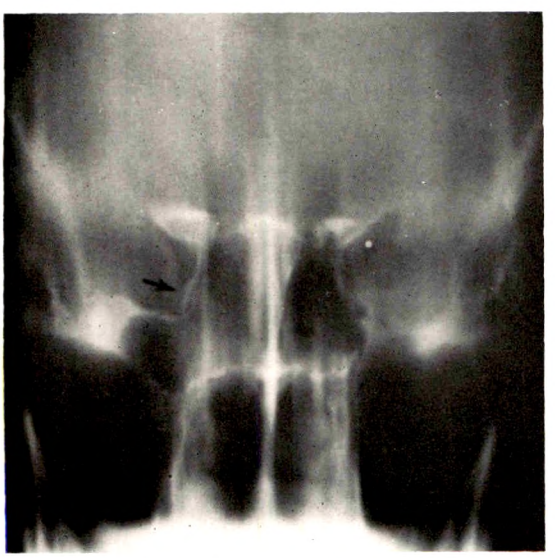


Fig. 2. Frontal tomogram showing opacified right sphenoid sinus (arrow).

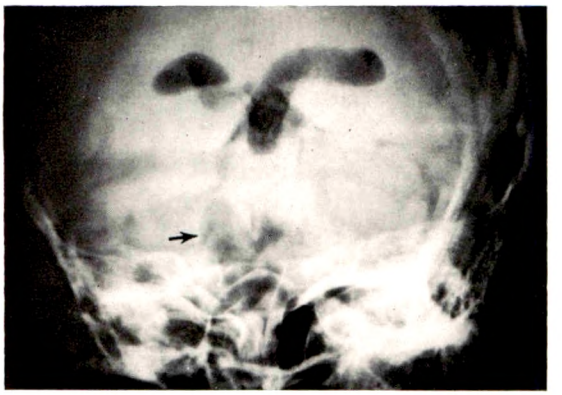


Fig. 3. Encephalogram showing an air containing cyst (arrow) just to the right of the midline above the sella.

On December 4, 1968 a bifrontal craniotomy was performed. The sphenoid sinus was opened through the planum sphenoidale and packed with muscle and fascia. The patient tolerated the procedure well. The rhinorrhea ceased and the patient was discharged 10 days later. There has been no recurrence of rhinorrhea.

DISCUSSION

The identification of a cerebrospinal fluid fistula exercises the ingenuity and persistance of the radiologist. Investigation proceeds on a logical basis in an attempt, first of all, to define the side on which the fistula is situated. Thereafter some hint must be obtained as to whether the fistula originates in the anterior, middle, or posterior fossa, since subsequent studies depend on this information.

Plain roentgenograms of the skull must be scrutinized with unusual care in order to detect any minor abnormality. The opacification of one of the air sinuses may

SEMINARS IN DIAGNOSTIC RADIOLOGY

Two Seminars will be presented in Miami Beach in March of 1971 as follows:

1. Ninth Annual Seminar on "Selected Topics in Roentgen-diagnosis: Radiologic-Pathologic Correlations," at Fontainebleau Hotel, March 9–13, 1971, sponsored by the Department of Radiology, University of Miami School of Medicine, Jackson Memorial Hospital, Miami, Florida. During the four-day course, selected topics in chest, bone, alimentary tract, and urinary tract roentgenology will be discussed by radiologists and pathologists, who will stress the correlation between radiographic and anatomical pathology. An outstanding faculty of Radiologists and Pathologists will participate with the faculty of the University of Miami School of Medicine in presenting this course. The Guest Faculty includes: Benjamin Castleman, M.D., Boston, Massachusetts; Arthur Clemett, M.D., New York, New York; Sheldon F. Markel, M.D., Ann Arbor, Michigan; Harry Z. Mellins, M.D., Boston, Massachusetts; Maurice M. Reeder, M.D., Honolulu, Hawaii; Elias G. Theros, M.D., Washington, D.C., Jerome F. Wiot, M.D., Cincinnati, Ohio; and John P. Wyatt, M.D., Winnipeg, Canada.

2. Seminar on "Special Procedures in Diagnostic Radiology," Eden Roc Hotel, March 14–17, 1971, sponsored by the Department of Radiology at Mount Sinai Hospital, Miami Beach, in collaboration with the Department of Radiology at Jackson Memorial Hospital, University of Miami School of Medicine. This 2½ day seminar is intended to discuss technical aspects of Special Procedures in Diagnostic Radiology. Where, with what, how, why and when to do them will be discussed. A question and answer period will allow discussion of the topics presented. Technical and scientific exhibits will be displayed. This seminar for the first time will cover all the technical aspects of all Special Procedures excluding Neuroradiology.

These 2 Seminars will be preceded by a Seminar in Nuclear Medicine "Hematopoietic and Gastrointestinal Investigation with Radionuclides," March 3–7, 1971, at the Fontainebleau Hotel.

For further information please contact Manuel Viamonte, Jr., M.D., Professor and Chairman, Department of Radiology, School of Medicine, P. O. Box 875, Biscayne Annex, University of Miami, Miami, Florida 33152.

SYMPOSIUM ON INFLAMMATORY DISEASES OF THE GASTROINTESTINAL AND BILIARY TRACTS

The Department of Diagnostic Radiology of the University of Kentucky School of Medicine sponsors a "Symposium on Inflammatory Diseases of the Gastrointestinal and Biliary Tracts," April 28 to April 30, 1971 at the Albert B. Chandler Medical Center, Lexington, Kentucky.

The Guest Faculty will include: Stanley M. Baum, M.D., John A. Campbell, M.D., Richard A. Marshak, M.D., Rene Menguy, M.D., Maurice M. Reeder, M.D., John W. Schaefer, M.D., Richard Schatzki, M.D., Benjamin H. Sullivan, Jr., M.D., and Bernard S. Wolf, M.D.

Registrants will have an opportunity to purchase 4 tickets to the Kentucky Derby on May 1.

Further information may be obtained from Arthur Lieber, M.D., Program Director, Department of Diagnostic Radiology, University of Kentucky School of Medicine, Lexington, Kentucky 40506.

TWENTY-FOURTH INDIAN CONGRESS OF RADIOLOGY, 1971

The Twenty-fourth Indian Congress of Radiology will be held in Calcutta from January 22 to 24, 1971.

Delegates from all over India and abroad are invited to read papers and take part in the scientific deliberations of the Congress with special attention to the systematic confrontation of theory and practice of Radiology. There will be a Scientific and Commercial Exhibition during the Congress. A social program is also being arranged.

All further information will be available from Dr. S. Bose, Organizing Secretary, 6 Harrington Street, Calcutta-16, India.

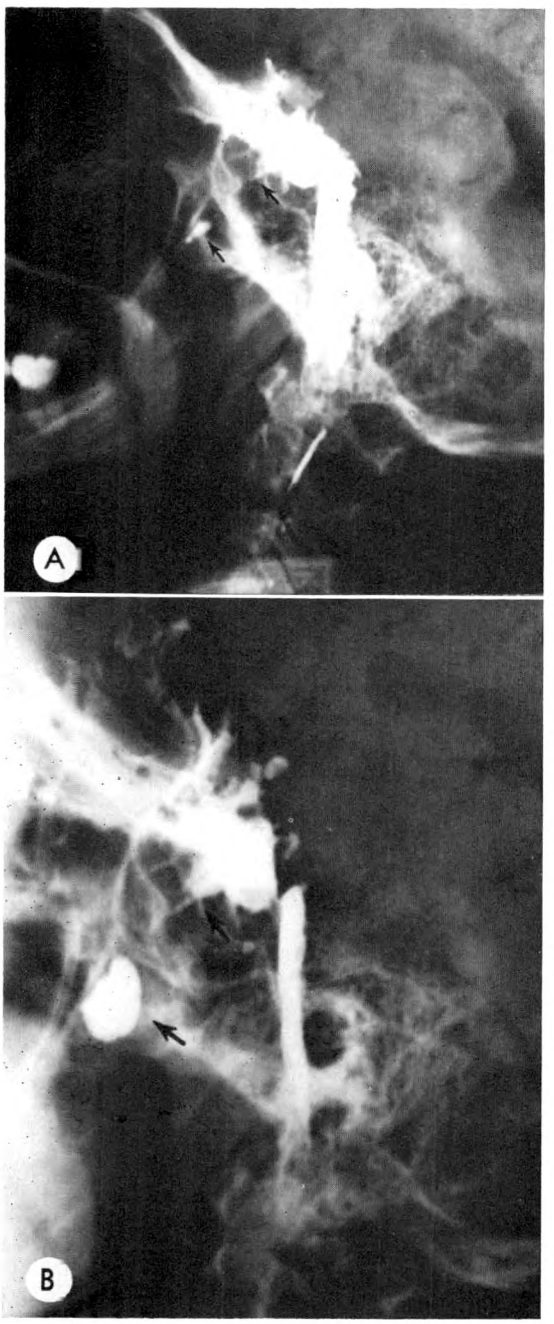


FIG. 2. (*A* and *B*) Pantopaque myelograms showing contrast material flowing from the floor of the pituitary fossa into the right sphenoid sinus (arrow).

be valuable evidence and an air fluid level may be disclosed on the brow up lateral roentgenogram. Defects in the cribriform plate are usually impossible to see since they are so small. However, good fortune afforded detection of the defect in Case I. In the half axial projection a congenital abnormality of the inner ear may be disclosed and these have been associated with dural defects within the internal auditory canal. Tomography of the cribriform plate region, the petrous temporal bone and the sphenoid sinuses in both projections is important.

We have found encephalography to be helpful in most of the primary cerebrospinal fluid fistulae as well as the post traumatic ones.[11] Tethering of a ventricular or arachnoid diverticulum may suggest the site of the fistula. In Case II, air was seen within the sphenoid sinus during encephalography and an unusual collection of air was noted in relation to the right side of the sella. Some fistulae occur as a result of obstruction of the ventricular pathways and thus encephalography is helpful in delineating the cause.

Isotope encephalography[1] may be invaluable in demonstrating the size and site of the fistula. However, in neither of our 2 cases did it give us any additional information. Isotope scans of the head are performed in anteroposterior and lateral projections after the instillation of high activity RISA (I^{131}) into the lumbar subarachnoid space. Scans may be superimposed on the skull roentgenograms to demonstrate paranasal, nasal and nasopharyngeal radioactive cerebrospinal fluid leakage. Counts of cotton pledgets placed in the region of the cribriform plate, the sphenoethmoidal recess, sphenoid sinus, and the pharyngeal orifice of the eustachian tube allow more precise localization of the leakage site. The latter technique fails when leakage is profuse as in Case II.

Positive contrast studies[5] are useful in fistulae originating in the middle or posterior fossa. We have fundamental reservations about the wisdom of positive contrast studies of the nose in the case of cribriform plate fistulae.[13] In performing a positive contrast study, care and great patience are necessary. The pantopaque is injected in the lumbar subarachnoid space and ma-

BOOK REVIEWS

RADIOLOGY OF THE SMALL INTESTINE. By Richard H. Marshak, M.D., Clinical Professor of Radiology, Mount Sinai School of Medicine, New York; and Arthur E. Lindner, M.D., Assistant Professor of Medicine, New York University School of Medicine, New York. Cloth. Pp. 510, with 498 figures. Price, $32.00 U.S.; $34.60 Canada. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1970.

This book with 21 chapters and a very large number of beautiful illustrations is by far the best text on radiology of the small bowel. The text is concise, up-to-date and covers the frequent, the unusual and the rare.

Two chapters, one by Arthur R. Clemett on "Uncommon Lesions of the Small Intestine" and one by John E. Moseley and Jack G. Rabinowitz on "The Small Bowel in Infants and Children" add further strength to this book with the superb expertise of the primary authors.

It is amazing that in only 500 pages such a wealth of information can be conveyed. The references are carefully chosen and show discrimination and dedication to the important. The loving care of the authors, who insist on atlas-sized film reproductions to emphasize detail, is to be commended. Anyone publishing books of this nature knows how difficult and costly it is to have illustrations like Figures 18–3, 19–28, 20–18 and 21–29 among many others published.

While there may be some disagreement from individuals about the organization of chapters like "Uncommon Lesions" and "Miscellaneous Lesions," it should be remembered that no two individuals organize anything in the same way and that the final presentation must be left to the judgment of the author.

Nothing can detract from the over-all impression that this is truly a masterpiece which will serve as the yardstick by which other books on the small intestine will be judged for many years to come.

ALEXANDER R. MARGULIS, M.D.

DISEASES OF BONE AND JOINTS. By Louis Lichtenstein, M.D., Clinical Professor of Pathology, University of California, San Francisco; Professor Extraordinario, National University of Mexico; Honorary Member, Spanish Orthopedic Society (SECOT); Honorary Member, Western Orthopedic Association; Fellow, New York Academy of Medicine; Consultant in Pathology, Children's Hospital, San Francisco; Consultant in Pathology, U. S. Naval Hospital, Oakland; Consultant in Orthopaedic Pathology, St. Joseph's Hospital and Mt. Zion Hospital and Medical Center, San Francisco, Calif. Cloth. Pp. 228, with 287 illustrations. Price, $17.50. C. V. Mosby Company, 3207 Washington Blvd., St. Louis, Mo. 63103, 1970.

This text is offered as a companion volume to "Bone Tumors" written in 1965 by the same author and covers skeletal diseases in general, apart from neoplasms. The author makes no pretense of contributing an encyclopedic treatise covering in detail all aspects of every conceivable subject in the field. The purpose of the monograph, as stated, is to provide a concise yet informative comprehensive survey for basic orientation with emphasis on pathology, and as such to help meet the needs of orthopedists, general surgeons, radiologists, pathologists, pediatricians, internists, and other men in medicine and its allied sciences who have an interest in disease of bone and joints.

In the preparation of certain chapters, the author, an eminent pathologist, has drawn upon published experience of others in internal medicine and endocrinology. In addition roentgenographic reproductions have been freely utilized as invaluable aids in graphically depicting many conditions. Much of the material has been selected from the author's own teaching files and experience accrued over the past 20 years or more. Relevant physiologic, genetic, immunologic, and biochemical data have been integrated at every opportunity with clinical and pathologic findings.

As the table of contents indicates, the structure, function, and pathologic alterations of bone joints may reflect in many obvious and subtle ways diseases occurring elsewhere in the body. Thus, genetic abnormalities, altered renal function, metabolic disorders, vitamin imbalances, endocrine malfunctions, neurotrophic disorders, blood diseases, and many other dis-

nipulated into each cerebello-pontine angle and thereafter into the sella. It may also be possible to place it in the anterior fossa in the region of the cribriform plate. Unless the fistula is large, as in Case II, the contrast material must be left in position for a lengthy period as its viscosity usually prevents the instantaneous demonstration of a fistula. Low density pantopaque might be useful in certain cases.[3]

SUMMARY

The identification of the site of a primary cerebrospinal fluid fistula is of considerable importance in the surgical treatment, and exercises all the ingenuity and patience of the radiologist.

We have reported 2 cases of primary cerebrospinal fluid fistula, one originating in the anterior fossa and the other in the middle fossa and have described our thoughts on the method of investigation.

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ease states are discussed insofar as they are reflected in changes of bones and joints.

As in any book, certain areas are more outstanding than others and the chapters on systematized Anomalies of Skeletal Development, Histiocytosis-X, Pathology of Some Common and Unusual Orthopedic Conditions, and the Disorders of the Synovial Joints are especially well presented. On the whole, the well organized 14 chapters provide a quick and ready simple reference text in which the subjects, although briefly treated in some instances as intended by the author, can be pursued in greater detail by referring to the many specific and general references provided at the end of the chapter.

E. Nicholas Sargent, M.D.

BOOKS RECEIVED

Formulating X-ray Techniques. Seventh edition. By John B. Cahoon, R.T., F.A.S.R.T., Assistant Professor and Director, Education, Research and Development in Radiologic Technology, Duke University Medical Center, Durham, N. C. Cloth. Pp. 381, with some figures. Price, $12.50. Duke University Press, 6697 College Station, Durham, N. C. 27708, 1970.

Overcoming the Fear of Death. By David Cole Gordon. Cloth. Pp. 115. Price, $3.95. The Macmillan Company, 866 Third Avenue, New York, N. Y. 10022, 1970.

Elementary Anatomy and Physiology: for Students in Radiologic and Laboratory Technology. By Gerold J. Wax, R.T. (A.R.R.T.), Staff Radiologic Technologist, Department of Radiology, Swedish Covenant Hospital, Chicago, Ill. Cloth. Pp. 153, with many figures. Price, $7.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1970.

Atlas of Nuclear Medicine. Volume 2: Lung and Heart. By Frank H. DeLand, M.D., Associate Professor of Radiology and Radiological Science, Johns Hopkins Medical Institutions; and Henry N. Wagner, Jr., M.D., Professor of Radiology, Radiological Science, and Medicine, Johns Hopkins Medical Institutions; with the assistance of Wendy A. North, M.I.R., Research Assistant, Johns Hopkins Medical Institutions, Baltimore, Md. Cloth. Pp. 284, with 810 illustrations. Price, $20.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1970.

Morphologic Pathology of the Alimentary Canal; Gross, Radiographic and Microscopic. By Antonio M. Valdes-Dapena, M.D., Associate Professor of Pathology, Graduate Division, School of Medicine; Director of Pathologic Anatomy, Graduate Hospital, University of Pennsylvania; and George N. Stein, B.A., M.D., Professor of Clinical Radiology, Graduate Division, School of Medicine; Associate in Radiology, Graduate Hospital and Presbyterian-University of Pennsylvania Medical Center. Cloth. Pp. 902, with 1,374 illustrations. Price, $30.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1970.

Roentgenographic Interpretation for the Dentist. By S. N. Bhaskar, B.D.S., D.D.S., M.S., Ph.D., Colonel, United States Army Dental Corps; Director, United States Army Institute of Dental Research; Associate Professor of Pathology, Georgetown University School of Dentistry, Washington, D. C.; Consultant in Oral Pathology to the Assistant Surgeon General and Chief of the Dental Corps, United States Army; Member, Dental Research Advisory Committee to the Medical Research and Development Division, Department of the Army; formerly Chief, Department of Oral Pathology, United States Army Institute of Dental Research, Walter Reed Army Medical Center. Cloth. Pp. 196, with 298 illustrations. Price, $18.50. C. V. Mosby Company, 3207 Washington Boulevard, St. Louis, Mo. 63103, 1970.

Indications and Alternatives in X-ray Diagnosis. By Melvyn H. Schreiber, M.D., Professor of Radiology, University of Texas Medical Branch, Galveston, Texas. Paper. Pp. 117. Price, $4.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1970.

International Conference on Proctology. Section of Proctology with the American Proctologic Society and the Section of Colonic and Rectal Surgery of the Royal Australasian College of Surgeons. Proceedings of the Royal Society of Medicine, Supplement to Volume 63, 1970. Paper. Pp. 160. The Royal Society of Medicine, 1 Wimpole Street, London WiM 8 AE, England, 1970.

La Hipofisectomía en Clínica. By Alfredo Jadresic, Professor Extraordinario de Medicina, Facultad de Medicina, Universidad de Chile. Cloth. Pp. 190, with some figures. Editorial Universitaria, S. A., Santiago de Chile, 1969.

Thorax. By Heinz Bohlig. Band I: Röntgen, wer—wie—wann? Paper. Pp. 151, with 43 figures. Price, DM 9.-. Georg Thieme Verlag, 7 Stuttgart 1, Germany, 1970.

Annual Report, 1969. International Agency for Research on Cancer: World Health Organization, Lyon, France. Paper. Pp. 109. WHO, Geneva, Switzerland, 1970.

A NEW SIMPLE MEASUREMENT OF PINEAL CALCIFICATION IN THE LATERAL CRANIOGRAM*

By YOSHITOKI MURASE, SENGAI TANAKA, ATSURO FUTAMURA, TAKAO ITO, KEN IMAMURA *and* KAZUKI SAKATA
GIFU, JAPAN

A NEW simple method of measuring the position of pineal calcification in the lateral plain craniogram has been devised and its reliability has been confirmed.

MATERIAL

Of the lateral craniograms made on recent outpatients at our clinic, in which intracranial space-occupying lesions and cerebral atrophy were excluded clinically, those showing clear pineal calcification and having appropriate clarity for measurement were selected. One hundred and fifty such craniograms (1 each from 150 cases) were chosen randomly. The age of the patients ranged from 10 to 74 years and was 35.4 years on the average. The lateral craniograms were made as follows: The head was placed on the cassette with the sagittal plane parallel to the film. The central ray was directed perpendicular to the film through a point about 2 cm. anterior to the external auditory meatus on the cantho-meatal line. The distance between the tube and the film was 1 meter.

METHOD OF MEASUREMENT

On the lateral craniogram, the curved line formed by the anterior border of the middle cranial fossa and the curved line formed by the inferior surface of the orbital roof were identified and the crossing point A was marked, as shown in Figure 1. When these lines were separately observed on each side, the crossing points (A_1 and A_2) were determined on each side and the midpoint between them (A_0) was marked as shown in the inset of Figure 1. The point A or A_0 was connected to the outer lambda and the crossing point of this line with the inner table of the lambda region was named L. The midpoint M of the line AL or A_0L was determined and the distance from M to the center P of pineal calcification was measured as the components parallel (horizontal) and perpendicular (vertical) to the line.

RESULTS

In Figure 2, the horizontal and the vertical components of MP distance were taken as abscissa and ordinate, respectively, and the results of measurement were plotted. As shown in the Figure, the center P of normal pineal calcification showed a concentrated distribution around the point M. P was found to lie, in 79 (93 per cent) of 85 cases, on the vertical lines 10 mm. before M and 2 mm. behind M, and on the horizontal lines 4 mm. above M and 8 mm. below M, or in a square of 12×12 mm.^{2} with the center 4 mm. before and 2 mm. below M. The results in young (10–17 years of age) subjects (indicated by x) and those in older (61–74 years of age) patients (indicated by black triangles) showed no particular tendency as compared to the average results.

In applying this method of measurement, the most frequently encountered difficulty is when the lambda cannot be identified precisely. In such cases it is necessary to determine a substitute L point by some other means. For this purpose we selected the most caudal point B (Fig. 1) on the inner table of the occipital bone as a reference point. In the cases where the lambda was clearly identifiable the distance BL was measured. As shown in Figure 3, the distance showed fairly little individual difference, the average being about 100 mm.

In the cases where the lambda was not identifiable, the point L′, a substitution for L, was determined as a point on the occi-

* From the Second Surgical Division (Chief Prof. Dr. Takao Taketomo), Gifu University School of Medicine, Gifu, Japan.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual meeting: Sheraton Hotel, Boston, Mass., September 28–October 1, 1971.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Jerome M. Vaet, Saroni Tumor Institute, 1600 Divisadero St., San Francisco, Calif. 94115. Annual meeting: Mexico City, Mexico, March 15-18, 1971.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary-Treasurer, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., November 29–December 4, 1970.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: St. Louis, Mo., Chase-Park Hotel, March 30–April 3, 1971.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga., 30322. Annual meeting: Atlantic City, N. J., June 20–24, 1971.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Denver, Colo., Dec. 7–11, 1970, Denver-Hilton Hotel; Bal Harbour, Fla., June 7–11, 1971, Americana Hotel; and Dallas, Tex., Dec. 6–10, 1971, Statler-Hilton Hotel.

Written examinations are scheduled in June of each year in 12 large centers, and applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Beginning in 1970 the deadline for receiving applications for any examination is Sept. 30 of the year preceding.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, Leonard Stanton, Hahnemann Medical College, 230 N. Broad St., Philadelphia, Pa. 19102. Annual meeting to be announced.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Carl R. Bogardus, Jr., University of Oklahoma Medical Center, Oklahoma City, Oklahoma 73104

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, F. J. Fry, M.Sc., Bioacoustics Lab., University of Illinois, Urbana, Ill.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary-Treasurer, Dr. Eugene V. Leslie, Edward J. Meyer Memorial Hospital, 462 Grider St., Buffalo, N. Y. 14215.

THIRTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Meeting: Madrid, Spain, Oct. 13–19, 1973.

TENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Manuel Viamonte, Jr., University of Miami School of Medicine, Jackson Memorial Hospital, Miami Fla. 33136.
President, Dr. Victor A. Marcial, Puerto Rico Nuclear Center, Caparra Heights Station, San Juan, Puerto Rico 00935.
Meeting: San Jeronimo-Hilton Hotel, San Juan, Puerto Rico, May 16-22, 1971.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.

ALABAMA CHAPTER OF ACR
Secretary, Dr. William V. Weldon, Medical Arts Building, Birmingham, Ala. 35205. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY
Secretary, Dr. Bruce C. Wright, Providence Hospital, Anchorage, Alaska. Meets third Wednesday each month.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Wesley S. Fee, 2421 E. 6th St., Tucson, Ariz. 85719. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. Wilma C. Diner, Univ. of Arkansas Medical Center, Little Rock, Ark. 72201. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Elliott C. Lasser, University Hospital of San Diego County, San Diego, Calif. 92103. Annual Meeting: Durham, N. C., May 13–15, 1971, with the Duke University and University of North Carolina Radiology Departments serving, as co-hosts.

ATLANTA RADIOLOGICAL SOCIETY
Secretary, Dr. Richard S. Colvin, Emory University Clinic, Atlanta, Ga. 30322. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Advisor, Colonel Paul E. Sieber. *Secretary*, LTC Peter B. Riesz, USAH Bad Cannstatt, APO 09154, New York, N. Y. Meets quarterly.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. R. John Gould, 41 Lombardy Rd., Drexel Hill, Pa. 19026.

BLUEGRASS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Joseph A. Sayeg, Ph.D., Radiation Physicist, University of Kentucky, Lexington, Ky. 40506. The Society meets once each month during the school year.

BRONX RADIOLOGICAL SOCIETY, NEW YORK STATE, CHAPTER ACR
Secretary-Treasurer, Dr. David Bruce Hayt, 600 E. 233rd St., Bronx, N. Y. 10466. Meets 4 times a year.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Rubem Pochaczevsky, 300 Skillman Ave., Brooklyn, N.Y. 11211. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Glen M. Ebersole, 405 Spring St., Jamestown, N.Y. 14701. Meets second Monday evening each month, October to May inclusive, at University Club.

CALIFORNIA RADIOLOGICAL SOCIETY, CALIFORNIA CHAPTER of ACR
Secretary-Treasurer, Dr. John L. Gwinn, Childrens Hospital of Los Angeles, P.O. Box 54700, Los Angeles, Calif. 90054.

CATAWBA VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Emmett R. White, P. O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 P.M.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

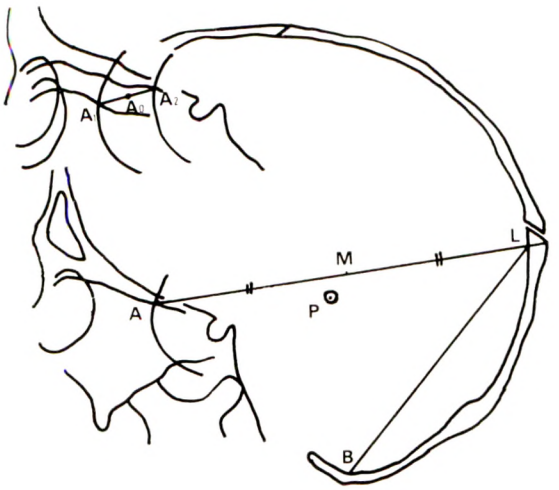


Fig. 1. Schematic drawing indicating the method of measurement. A = Crossing point of the curved line formed by anterior border of the middle cranial fossa and the curved line formed by inferior surface of the orbital roof. B = Most caudal point on the inner table of the occipital bone. L = Crossing point of the line, connecting A to outer lambda, and the inner table. M = Midpoint of AL. P = center of pineal calcification. *Inset* (left upper corner). A_0 is the midpoint between A_1 and A_2, which are the crossing points of A on each side.

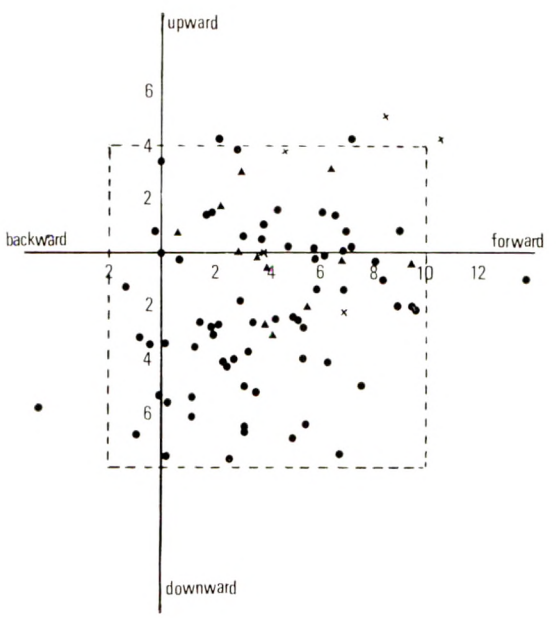


Fig. 2. Distribution of P in relation to M in manifest lambda cases. Abscissa: horizontal distance in mm. Ordinate: vertical distance in mm. Origin is the point M. x = young subjects. Black triangles = older patients. For dotted square see text.

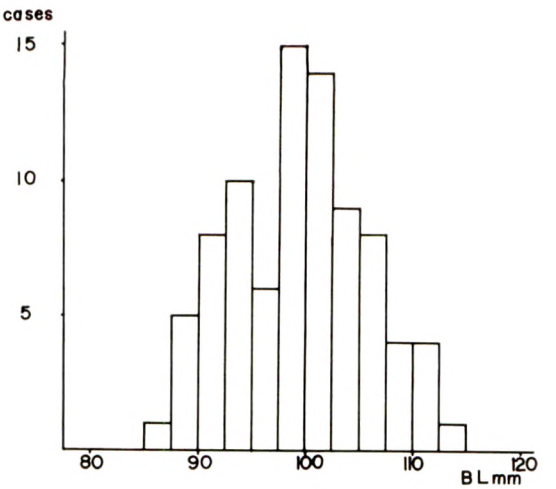


Fig. 3. BL distance in manifest lambda cases. Abscissa: BL distance in mm. Ordinate: number of cases.

pital inner table having a straight distance of 100 mm. from B. The position of P in relation to M was measured as previously. As shown in Figure 4, the point P was found to lay within the same square as described above (Fig. 2) in 62 (95 per cent) of the 65 cases. The point L′ was, therefore,

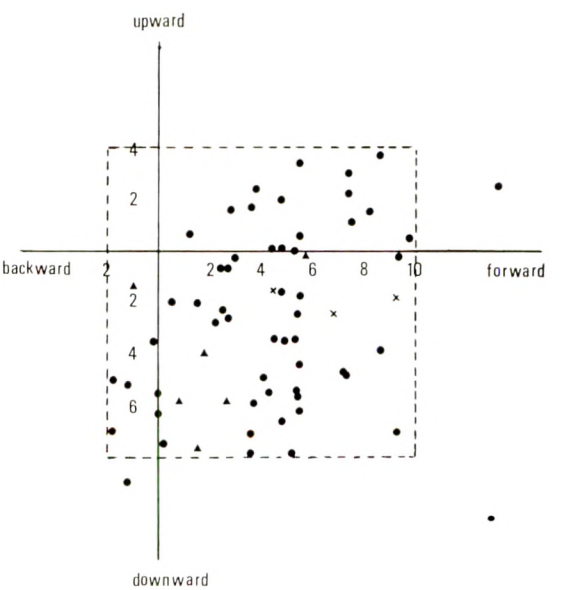


Fig. 4. Distribution of P in relation to M in obscure lambda cases. Details appear in the legend of Figure 2.

Central New York Radiological Society
Secretary-Treasurer, Dr. David N. Cheris, Community General Hospital of Greater Syracuse, Broad Road, Syracuse, N. Y. 13215. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary, Dr. James V. Blazek, 2586 Lane Rd., Columbus, Ohio 43220. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Chicago Roentgen Society
Secretary-Treasurer, Dr. William T. Moss, 250 E. Superior St., Chicago, Ill. 60611. Meets third Thursday of each month, October to April, except December, at the Bismarck Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. James Farmer, 18869 Canyon Rd. Fairview Park, Ohio 44126. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Marvin L. Daves, Univ. of Colorado Medical Center, 4200 E. Ninth Ave., Denver, Colo. 80220. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. V. V. Kirby, 1722 Spring Lake Dr., Arlington, Tex. 76010. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. James H. Taylor, Wilmington Medical Center, Wilmington, Del. 19899.

East Bay Radiological Society
Secretary-Treasurer, Dr. S. Walter Kran, Doctors' Hospital of San Leandro, 13855 East 14th St., San Leandro, Calif. 94578. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. Eberhard F. Besemann, Baroness Erlanger Hospital, Chattanooga, Tenn. 37403. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary, Dr. Wm. F. Lindsey, 1215 Hodges Dr., Tallahassee, Fla. 32303. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Allen L. Sheer, University Community Hospital, 13505 N. 31st St., Tampa, Fla. 33612. Meets in January, March, May, July, September and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. Walker Harris, The Medical Center, Columbus, Ga. 31902. Meets in spring and fall at Annual State Society Meeting.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. John Kathe, North Shore Hospital, Miami, Fla. 33150. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. Roland P. Ernst, 3720 Washington Ave., St. Louis, Mo. 63108.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Virgil R. Jobe, Jr., 888 South King St., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, John H. Pingel, Argonne National Laboratory, 9700 S. Cass Ave., Argonne, Ill. 60439. Annual Meeting: Waldorf Astoria Hotel, New York City, July 11–15, 1971.

Houston Radiological Society
Secretary, Dr. Kenneth M. Jensen, 1615 St. Joseph Prof. Bldg., Houston, Texas 77002. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025

Idaho State Radiological Society, Chapter of ACR
Secretary, Dr. Hugh P. Smith, Jr., 130 E. Bannock, Boise, Id. 83702. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. Jack L. Melamed, 1230 Sunset Rd. Winnetka, Ill. 60093. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. Dale B. Parshall, Elkhart General Hospital, P.O. Box 1329, Elkhart, Ind. 46514.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Wm. R. Allen, 155 S. 18th St. Kansas City, Kan. 66102. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Joan R. Hale, 402 Heyburn Bldg. Louisville, Ky. 40202. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N.Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Harold L. Atkins, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Harry T. Vanley, St. Mary's Long Beach Hospital, Long Beach, Calif. 90083. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Annual Midwinter Conference: Jan 30–31, 1971.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. Nathan Stofberg, 4519 Hawksbury Rd., Pikesville, Md. 21208.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Norman L. Siadowsky, The Faulkner Hosp., 1153 Centre St., Jamaica Plain, Mass. 02130

Memphis Roentgen Society
Secretary-Treasurer, Dr. Webster Riggs, Jr., The University of Tennessee College of Medicine, Department of Radiology, Walter F. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Ind. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary, Dr. David P. Corbett, Harper Hospital, De-

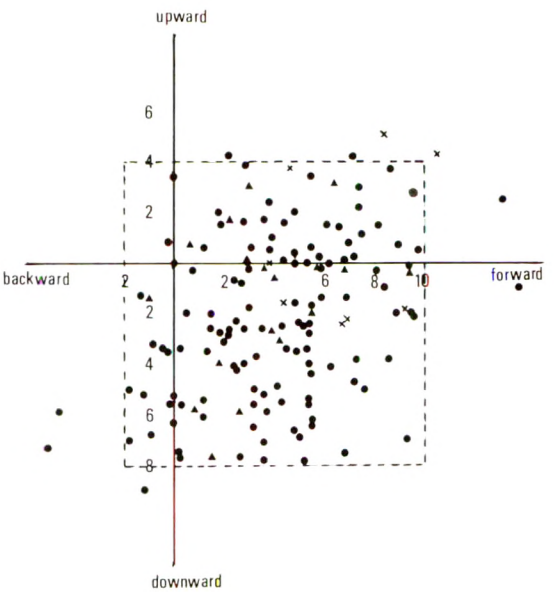


Fig. 5. Distribution of P in relation to M in total cases. Details appear in the legend of Figure 2.

a good substitution for the point L for all practical purposes.

In Figure 5, the results in the cases where point L was used and those in which point L′ was used are shown in combination. P point was within the square in 141 (94 per cent) of the 150 cases.

DISCUSSION

Since Vastine and Kinney[7] reported their pineal measurement in 1927, various methods have been proposed by various authors to estimate the normal position of the pineal calcification in the lateral plain craniogram. To determine displacement of the pineal calcification by any of these methods, it is necessary to have a chart on which standard values of the distances between the pineal gland and various points on the inner table are recorded,[3,7] or have a specific ruler,[2,5] or a specific elastic cord with markers on it.[1] Pousner's formula[4] is relatively simple, but gives no information on vertical displacement of the calcification. For this reason, it would be quite convenient if a line could be drawn which passes through, and is bisected by the pineal calcification, similar to the Twining line[6] in relation to the fourth ventricle. With this view in mind the line AL or A_0L, as described above, was devised. As regards the individual cases, it was found that normal pineal calcifications were distributed fairly closely around the midpoint of the line AL or A_0L. Thus, rough estimation of the normal position of a pineal calcification is quite easy, if it is near the point M; *i.e.*, the center of the pineal calcification is within a square of 12×12 mm.^{2} or 4 mm. before and 2 mm. below point M. If the center of the pineal calcification is outside the square, we can conclude with a high probability that it is displaced.

In the cases where the lambda is not identifiable a substitute L point is determined. The internal or external occipital protuberance may serve as a reference point for this purpose; however, since its tip sometimes is difficult to identify exactly due to its indeterminate appearance or individual difference in its position, we selected point B as a reference point. The result of measurement gives a figure of 100 mm. on the average for BL distance. It was subsequently confirmed that the L′ point, 100 mm. distant from B, is a reliable substitution for the point L for evaluating the position of point P. In other words, the present method of measuring the position of pineal calcification in the lateral craniogram is applicable for all cases with high reliability.

CONCLUSION

A new simple method of measuring the position of pineal calcification in the lateral plain craniogram was devised.

The crossing point A (or the midpoint A_0 between A_1 and A_2 on each side) of the curved line formed by the anterior border of the middle cranial fossa and the curved line formed by the inferior surface of the orbital roof, is connected to the outer lambda, and the crossing point of this line with the inner table is named L.

Normal pineal calcification was found in high probability to lie around midpoint

troit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. James E. Bell, 8700 W. Wisconsin Ave., Milwaukee, Wis. 53213. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Warren L. Kump, 4243 Glenwood Ave., Minneapolis, Minn. 55422. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ottis G. Ball, 5356 Balmoral Drive, Jackson, Miss. 39211. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Arthur A. Porporis, 100 N. Euclid Ave., St. Louis, Mo. 63108.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. Jon A. Anderson, Doctor's Building, 1231 N. 29th Street, Billings, Mont. 59101.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. Gordon F. Johnson, 4239 Farnam, Omaha, Neb. 68131. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. Harris W. Knudson, 2020 W. Charleston Blvd., Las Vegas, Nev. 89102.

New England Roentgen Ray Society
Secretary, Dr. Stefan C. Schatzki, 1180 Beacon St., Brookline Mass. 02146. Meets third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary, George Farmlett, 33 Round Bay Rd., Keene, N. H. 03246. Meets four to six times yearly.

New Mexico Society of Radiologists Chapter of ACR
Secretary, Dr. Donald A. Wolfel, Albuquerque, New Mexico. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New York Roentgen Society
Secretary-Treasurer, Dr. Samuel H. Madell, 1. E. 82nd St., New York, N. Y. 10028. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference: Waldorf Astoria Hotel, New York, April 29–May 1, 1971. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
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Ohio State Radiological Society, Chapter of ACR
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Secretary, Dr. Richard B. Price, 204 Medical Tower Bldg., Oklahoma City, Okla. 73112. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Roderick A. Silveira, 100 E. Valencia Mesa Dr., Fullerton, Calif. 92632. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at the Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Gerlad L. Warnock, 11699 N. E. Glisan St., Portland, Ore. 97220. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd.; Beaverton, Oregon 97005. Meets annually in Portland, Oregon, Seattle, Washington or Victoria or Vancouver, British Columbia, in early May.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Theodore A. Tristan, Harrisburg Polyclinic Hosp., Harrisburg, Pa. 17105.

Philadelphia Roentgen Ray Society
Secretary, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Norman Williams, Department of Radiotherapy, Montefiore Hospital, 3459 Fifth Ave., Pittsburgh, Pa. 15213. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

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Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meetings are held quarterly

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Donald E. Gunderson, 3553 Bayard Dr., Cincinnati, Ohio 45208. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
Secretary, Dr. Ken C. Davidson, St. Luke's Hospital of Kansas City, Kansas City, Mo. 84111. Meets 5 times a year on given dates.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana, Chapter of ACR
Secretary, Dr. Ralph B. Bergerson, 154 Brockenbraugh Ct. Metairie, La. 70005. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic

M of the line AL or A_0L, or within a square of 12×12 mm.^{2}, having its center 4 mm. before and 2 mm. below the M point.

In the cases where the lambda point is difficult to determine, point L′, 10 cm. distant from the most caudal point on the inner table of the occipital bone, was found to be a reliable substitution for the point L.

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ROENTGEN DIAGNOSIS

SKELETAL SYSTEM

GOLDENBERG, RAPHAEL R., CAMPBELL, CRAWFORD J., and BONFIGLIO, MICHAEL. Giant-cell tumor of bone: an analysis of two hundred and eighteen cases. *J. Bone & Joint Surg.*, June, 1970, *52-A*, 619–664. (Address: R. R. Goldenberg, M.D., New Jersey College of Medicine, Newark, N. J. 07107.)

Giant cell tumors of bone are rare. There are few reports of a large series. This study presents the characteristics of the lesion based on more material than hitherto available.

Criteria for including cases in this study were good evidence of the tumor by roentgenogram and by tissue examination, all clinical details available for study, and follow-up of at least 2 years.

Additional cases were obtained from orthopedic colleagues.

Tumors had no distinctive clinical features. The most common complaints were pain and enlarging mass.

Giant cell tumors, which occurred in the epiphyseal ends of the long tubular bones, were located either eccentrically or centrally or they involved the entire end of the bone. Occasionally an enlarging lesion involved both the epiphyseal and the adjacent metaphyseal portion of the bone. The surrounding cortex is thinned and may be perforated or even fractured; the articular cartilage may be destroyed and occasionally an intraarticular line is visible. When the tumor is actively growing, little or no periosteal reaction is noted. No giant cell tumor was found in the diaphysis of a long bone. Lower end of the femur, the upper part of the tibia, and the lower end of the radius were the 3 most common sites for the lesion. These 3 locations contained 55 per cent of the tumors.

Radiolucency of a giant cell tumor is the result of extensive destruction of cancellous and cortical bone. There is no stippling or calcification within the tumor mass since a giant cell tumor possesses little or no osteogenic capacity except when a fracture has occurred. Roentgenographic diagnosis of a giant cell tumor in sites other than the long tubular bones is fraught with many problems.

Giant cell tumors in the spine present a lytic appearance with expansion of both cortices and may simulate an aneurysmal bone cyst, multiple myeloma, or a metastatic carcinoma.

Grossly, the giant cell tumors were composed of vascular, friable tissue, whose cut surface was described variously as reddish brown, yellowish brown, red, plum red, pink, brown, yellow, gray, grayish yellow, or whitish gray. Histologic features were a moderately vascularized network of round, oval, or spindle-shaped stromal cells and multinucleated giant cells.

Definite procedures for removal of the tumors in this series were curettage, curettage and bone grafting, resection or excision, resection and bone grafting, and amputation. Resection and prosthetic replacement were employed for 2 lesions, one in the proximal end of the femur, the other in the skull.

Secondary procedures included repetitions of those done initially and in addition lobectomy, radiation therapy, cryotherapy, and chemotherapy. In all, 180 secondary procedures were performed.

Radiation therapy was given to 46 patients either alone or combined with one or more of the aforementioned operative procedures.

A successful result in 5 of the 6 patients, treated by surgery for metastases to the lungs in this series, indicates the value of lobectomy in the treatment of giant cell tumors.

Complications were encountered in 126 of 218 patients. They included recurrence of seeding, metastasis, nonunion of the graft, fracture of a graft, infection, foot drop, irradiation sarcoma, and death.

Of the surgical procedures employed, the poorest results were obtained after curettage.

Treatment of giant cell tumors by *curettage combined with bone grafting*, to a large extent, is only successful when complete removal of the tumor is accomplished and the defect left by the tumor is repaired adequately by bone grafts. This method is recommended as the primary procedure for giant cell tumors with slight expansion of the involved bone, leaving the cortices clearly intact, and located in the proximal or distal end of the femur, the proximal end of the tibia, and in the sacrum.

Resection with or without bone grafting was successful in 68 per cent of the 66 tumors in this series.

Resection and bone grafting are strongly recommended for giant cell tumors which appear to be expanding and perforating the thinned cortices.

Amputation as the primary operation should be reserved for giant cell tumors which appear to be malignant and *have perforated the cortex* and invaded adjacent soft tissues so that treatment by complete resection with or without bone grafting is not possible.

Giant cell tumors of bone are not very radiosensitive. Neither the indications for radiotherapy nor the optimum time-dose relationship for these tumors is well known.

Danger of postirradiation sarcoma must be borne in mind.

Sixty-five per cent of the primary giant cell tumors in this series were successfully treated by the initial operative procedures; 35 per cent recurred.

Accurate prediction as to local recurrence or pulmonary metastasis could not be made on the basis of the histologic grade.

After primary curettage, 35 of 45 tumors recurred; after primary curettage combined with bone-grafting, 22 of the 91 tumors recurred.

THICKENING OF THE SKULL IN SURGICALLY TREATED HYDROCEPHALUS*

By ROBERT ANDERSON, M.D., STEPHEN A. KIEFFER, M.D., JUSTIN J. WOLFSON, M.D., DONLIN LONG, M.D., *and* HAROLD O. PETERSON, M.D.
MINNEAPOLIS, MINNESOTA

SURGICAL treatment of hydrocephalus has been shown to be beneficial in most cases,[5] but at present is not without occasional serious complications. Most reports of complications deal with problems occurring in the early postoperative period, such as epidural and subdural hemorrhage, collapse of the thinned cerebral mantle, and infection.[6]

Moseley, Rabinowitz and Dziadiw[9] recently reported a late complication of shunting procedures in which the calvaria become remarkably thick and the sutures fuse prematurely. This sequence of events has been briefly discussed by others as well.[1,4]

The purpose of this communication is to report 7 additional cases of this complication, to discuss possible causative factors and clinical significance.

CLINICAL AND ROENTGENOGRAPHIC FINDINGS

Of some 230 patients operated upon at the University of Minnesota Hospitals for hydrocephalus between 1938 and 1967 and reviewed by Long and co-workers,[8] 7 were found in whom serial skull roentgenograms following surgery showed the particular pattern of skull change described above. Six of the seven were from a group of 93 treated since 1961 with some type of ventriculoatrial shunt. The seventh patient had choroid plexus coagulation in 1957. Clinical and roentgenographic features of these cases are presented in Table 1.

The group includes 3 females and 4 males. Five of the seven were initially operated upon in the first year of life and the other 2 patients at ages 7 and 11 years, respectively.

Four of the cases were diagnosed as communicating hydrocephalus preoperatively, either by intraventricular instillation of pigmented dye with recovery from the lower lumbar sac, or by pneumoencephalography. The other 3 were found at operation to have adhesions in the cisterns of the posterior fossa blocking cerebrospinal fluid circulation. Each of these 3 had a history of antecedent meningitis.

The largest contribution to this increased thickness of the skull appears to be due to widening of the diploe (Fig. 1). The increased thickness is generalized in most cases, extending from nasion to the inion. Compared to the normal standards compiled by Roche,[12] the thickness of the skull at several sites of comparison (midfrontal, vertex, and lambda) averaged 2.5 times normal measurements for age and sex. In 1 patient the calvarial thickness was 5 times normal for age and sex. The earliest documented increase in thickness beyond the normal range was shown on skull roentgenograms made 12 months following implantation of a ventriculoatrial shunt in an 8 month old male infant. Length of follow-up varied from 3 years 2 months to 8 years 6 months (Fig. 2, *A-E*; and 3, *A-C*).

In 5 of the cases, more than one roentgenographic examination of the skull was available following surgery. In all these instances the greatest increase in skull thickness occurred during the first follow-up interval. However, all showed additional increments of lesser degree on later studies made after comparable intervals.

All 7 patients demonstrated premature closure of the cranial sutures, which varied from only partial synostosis of the sagittal suture to virtually complete fusion of all the sutures.

* From the Departments of Radiology and Neurosurgery, University of Minnesota Hospitals, Minneapolis, Minnesota.

At the time of this study, 189 of the 218 patients were alive and well, and 29 had died.

Grading seems to be of little value.—*Stephen N. Tager, M.D.*

WALL, JOHN J. Congenital pseudarthrosis of the clavicle. *J. Bone & Joint Surg.*, July, 1970, *52-A*, 1003–1009. (Address: 100 O' Connor Drive, San Jose, Calif.)

Five cases of congenital pseudarthrosis of the clavicle with long time follow-up are reported, indicating that this deformity does not cause symptoms or any disability. Therefore, the author advises against any attempts at surgical repair.

The embryology of this defect was studied and is described in the article.

The differential diagnosis from cleidocranial dysostosis is discussed, and a brief comparison is made between this condition and post-traumatic pseudarthrosis.—*John H. Harris, M.D.*

BLOOD AND LYMPH SYSTEM

MENON, A. N. K. Radiology of portal hypertension. *Indian J. Radiol.*, Nov., 1969, *23*, 180–190. (Address: Director of Medical Education, Government of Tamil Nadu, Madras, India.)

Portal hypertension is a syndrome caused by different etiologic factors, and its study is a multidisciplined process involving biochemistry, manometry, immunology, enzymology, and even neuropsychiatry. It is caused by obstruction either before the blood reaches the sinusoids of the liver, in the liver, or in the posthepatic venous system. In India, by far the most common causes of portal hypertension are nutritional and postnecrotic cirrhosis. Alcoholism is rarely a factor. The prognosis is less grave than in western countries. Ascites is a presenting symptom in 99 per cent of the cases in India and hematemasis is very rare.

The radiologic methods of study are plain roentgenography of the abdomen, barium contrast study of esophagus and stomach, splenoportography, and, more recently, isotopic studies of liver and spleen (both uptake and scanning), selective visceral arteriography, selective venography or cavography, and ultrasonic scanning.

All methods are briefly discussed in the article but the bulk of the dissussion concerns the experience gained in 300 spleno-venograms in 74 patients in Madras, India. Although vascular distribution in the liver follows several fairly distinct patterns, the variations are such that these do not prove practical in interpretation of the vascular study. The examinations were performed using 40 to 50 cc. of opaque material injected into the spleen through a large bore needle. By this method the flow to liver took 4 seconds and at 8 seconds there was a diffuse hepatogram. The portal vein averaged 22 mm. at its commencement and 19 mm. near the liver. Nonvisualization of the portal vein did not necessarily mean thrombosis; it occasionally occurred when there were large collateral veins or in severe cirrhosis with reversal of flow.

Early cirrhosis does not change the appearance of the hepatic radicles but in established cases there may be dilatation of the proximal portal vein with abrupt tapering creating the "tree in winter" appearance. A reticular or pooled appearance may be due to Eck fistulae. Mottling may be seen in postnecrotic cirrhosis. In severe cirrhosis no branches are seen.

Dilated collateral veins are practically always visible. The most common are the esophageal veins which normally do not exceed 0.5 mm. in width, but coronary varices are also very commonly demonstrated. The localization of the varices is important in management. If only esophageal varices are present, any form of shunt operation may be used in treatment. If splenorenal collaterals are evident, a portocaval shunt could be used and if mesenterocaval varices are present, only a splenorenal shunt will be helpful.

Limited lymphangiographic studies in portal hypertensive cases revealed 2 instances of lymphatic hypertension.

In the author's series, there were 4 cases of suprahepatic obstruction, 3 of which were due to congestive heart failure and 1 to constrictive pericarditis. The intra- and infrahepatic obstructions were due mainly to cirrhosis. Four were idiopathic (no obstruction demonstrated in the liver or portal vein). Splenic venograms with enormous portocaval shunts may be associated with hepatic encephalopathy, thus clarifying neurologic and psychiatric disorders previously unexplained.—*Lois Cowan Collins, M.D.*

KAUFMANN, HERBERT J., OLAFSSON, ARNI, and WEISSER, KURT. Angiographic evaluation of the renal artery in the newborn. *Australasian Radiol.*, Feb., 1970, *14*, 64–69. (From: Department of Radiology, Basel University Children's Hospital, Basel, Switzerland.)

The renal arteries in newborns were studied by transumbilical arterial catheterization in 14 cases.

This method was used in a total of 50 cases and no mortality, or morbidity was reported.

The average diameter of the renal arteries observed was between 1 and 2 mm. at their origin. The renal arteries angulated caudally at an average angle of 56° on the right and 54° on the left.

The procedure was helpful in evaluating abdominal masses, genital urinary anomalies, and a case of ascites.—*Victor G. Mikity, M.D.*

GENERAL

PAVLICA, P., and SARTONI-GALLONI, S. Considerazioni clinico-radiologiche sulla pene-

TABLE I

THICKENING OF THE SKULL IN SURGICALLY TREATED HYDROCEPHALUS

Patient and Sex	Age at First Surgery	Surgical Diagnosis	Total Number of Procedures	Duration of Follow-up	Roentgenographic Findings at Last Follow-up					
					Skull Thickness (Times Normal for Age and Sex)			Sutural Fusion		
					midfrontal	vertex	lambda	sagittal	coronal	lambdoid
S.F. F.	3 mo.	Communicating hydrocephalus	2	3 yr., 4 mo.	2.7	1.7	1.1	+	+	Partial
J.L. F.	4 mo.	Communicating hydrocephalus	6	3 yr., 2 mo.	2.4	1.7	2.1	+	Partial	Partial
J.M. M.	5 mo.	Postmeningitis posterior fossa adhesions	6	7 yr.	2.9	1.7	2.2	Partial	Partial	—
S.N. M.	9 mo.	Communicating hydrocephalus	2	8 yr., 6 mo.	2.8	1.7	1.4	+	+	Partial
J.W. F.	11 yr.	Posterior fossa adhesions	3	4 yr., 10 mo.	4.0	2.5	1.7	+	+	+
A.M. M.	7 yr.	Posterior fossa adhesions	4	7 yr., 11 mo.	3.9	4.1	2.4	+	+	Partial
K.F. M.	2 mo.	Communicating hydrocephalus	1	5 yr., 8 mo.	2.7	2.1	2.0	+	+	—

DISCUSSION

While it seems logical to assume that the release of abnormally high intracranial pressure in some way results in the increase in skull thickness noted in this group of patients, the exact cause of this remarkable thickening is unknown. It is also unclear why this small group of patients showed these changes while the great majority of similarly treated patients did not.

Experimental evidence regarding growth and development of the skull suggests that the bones of the cranial vault are relatively passive structures responsive to external stimuli. Moss and Young[11] state that the development of the skull, whether normal or abnormal, occurs within an interacting functional matrix consisting of cerebral capsule, skull base, and meninges. They further state that the entire capsule continues to enlarge at a rate determined by the expansion of its contents. They and Dorst[3] agree that the inner table of the skull is very sensitive to the development of the brain beneath it, and Dorst adds that if the stimulation of the neural mass becomes static, the bones of the calvaria increase in thickness but not in area. Campbell[2] suggests that a conspicuous diploe at an early age may indicate a decreased stimulation to the skull from hypoplasia of the underlying brain. Similar changes are seen in cerebral hemiatrophy on the side of involvement, a finding which again demonstrates the importance of the developing brain to calvarial configuration and thickness.[13]

The etiology of premature closure of the cranial sutures in these cases is also unclear. Experimental studies suggest that the sutures are not analogous to epiphyseal growth zones but rather respond to expan-

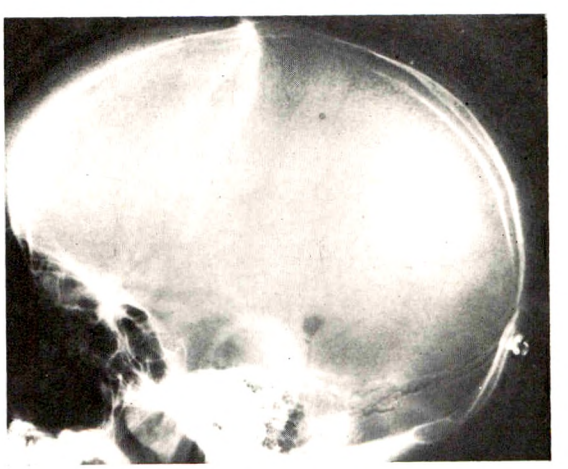


FIG. 1. S.N., first shunt operation at the age of 9 months. At the age of 8 years, the calvaria are thickened from the nasion to the foramen magnum. The inner and outer tables are relatively thin and the majority of the increase in thickness is in the diploe. The coronal sutures are fused.

trazione accidentale di mercurio metallico nel sottocutaneo. (Clinical and radiologic considerations on the accidental subcutaneous penetration of mercury.) *Ann. radiol. diagn.*, 1970, *42*, 389–395. (From: Istituto di Radiologia e Terapia Fisica, Ospedale Maggiore, Bologna, Italy.)

Two cases of accidental introduction of mercury in the subcutaneous tissues of a digit are reported.

Roentgenologically there was evidence of tiny numerous droplets of mercury in the soft tissues where the penetration occurred.

Roentgen studies repeated from 4 to 9 months after the accident showed minimal or no absorption of the droplets of mercury.—*A. F. Govoni, M.D.*

HASSAN, M. A. Subcutaneous liposarcoma of forearm followed by liposarcoma of omentum. *Brit. J. Surg.*, May, 1970, *57*, 393–396. (From: Royal Alexandra Hospital, Rhyl, Wales.)

A case of subcutaneous liposarcoma is presented. The author states that this appears to be the second case of subcutaneous liposarcoma reported.

In contrast to lipomas, liposarcomas are uncommon tumors. Usually they originate in the retroperitoneum, omentum or in the mesentaries. When they occur in the limbs they are in the deep intermuscular planes. Lipomas are commonly found in the subcutaneous fat.

In the case reported, the liposarcoma which was apparently well controlled, occurred in the subcutaneous fat of the left forearm. Eight years later the patient had massive involvement of the abdomen with liposarcoma of the omentum.

The author questions whether these were related or if they represent multicentric involvement.—*David C. Alftine, M.D.*

BRUTON, C. J., CORSELLIS, J. A. N., and RUSSELL, A. Hereditary hyperammonaemia. *Brain*, 1970, *93*, 423–434. (From: Runwell Hospital, Wickford, Essex, and the Hadassah University Hospital, Jerusalem, Israel.)

Hereditary hyperammonemia has been documented in several individuals who have been found to be lacking a basic enzyme ornithine transcarbamylase which is essential in the urea cycle for formation of urea from ammonia.

The children so involved have shown both physical and mental retardation.

At autopsy these patients were found to have marked cerebral atrophy with a predominance of an abnormal astrocyte described as astrocyte Type II.

An incidental finding was acute bronchitis due to bullous changes within the lungs.—*Victor G. Mikity, M.D.*

RADIATION THERAPY

BERNDT, H. Results of treatment of accidentally discovered and symptomless bronchial carcinomas. *German Med. Monthly*, Feb., 1970, *15*, 103–107. (Address: Robert-Rossle-Klinik, Lindenberger Weg 80, x 1115 Berlin-Buch, GDR.)

The survival rates of patients with bronchial carcinoma discovered on routine roentgenograms of the chest are higher than those of carcinoma diagnosed on the basis of complaints.

The more favorable prognosis of accidentally discovered lung cancers apparently applies mainly to patients who are still symptom-free when treatment is started.

The authors review 2,209 patients admitted from 1949 to 1962 and treated for lung cancer. Of this number, 462 cases had been detected in the course of routine roentgenography of the chest. Of the accidentally discovered tumors 183 had no associated symptomatology up to the time of treatment. The accidentally discovered lung cancers are characterized by a high proportion of early cases and peripheral tumors, a larger proportion of squamous cell carcinoma, a higher operation and excision rate even though the time elapsed from the routine roentgenography or from the first symptom to therapy is longer.

The survival rate for accidentally discovered cancers is 12.5 per cent as compared to the survival rate of 6 per cent for cancers diagnosed by reason of complaints.

Under present conditions of cancer therapy only systematic, yearly, routine roentgenograms of the chest of the entire population hold any promise of real progress in combating lung cancer.—*Luther W. Brady, M.D.*

GUNNLAUGSSON, GUNNAR H., WYCHULIS, ADAM R., ROLAND, CHARLES, and ELLIS, F. HENRY, JR. Analysis of the records of 1,657 patients with carcinoma of the esophagus and cardia of the stomach. *Surg., Gynec. & Obst.*, June, 1970, *130*, 997–1005. (From: Mayo Clinic and Mayo Foundation, Mayo Graduate School of Medicine, University of Minnesota, Rochester, Minn.)

The treatment of carcinoma of the esophagus and cardia continues to be viewed with pessimism by the medical profession and many physicians believe that little improvement has occurred in survival rates in recent years.

The authors report the experience at the Mayo Clinic with carcinoma of the esophagus and cardia with an effort to evaluate current modes of therapy by comparing the results in 2 separate, but consecutive, 10 year periods.

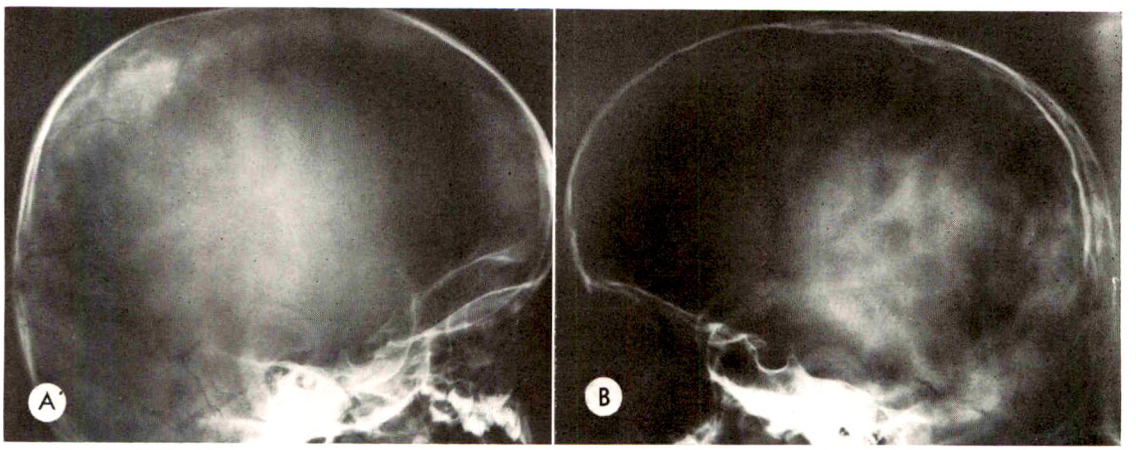


FIG. 2. J.W., hydrocephalus likely secondary to meningitis in infancy with resultant adhesions blocking the cisterns of the posterior fossa. (*A*) Age 1 year. The skull is enlarged but the calvaria are thin. Fissure fracture in occipital region. No surgical treatment. (*B*) Age 10 years. Patient complained of headaches, found to be lethargic with signs of increased intracranial pressure. The skull is large, but calvaria remain thin with prominent convolutional markings.

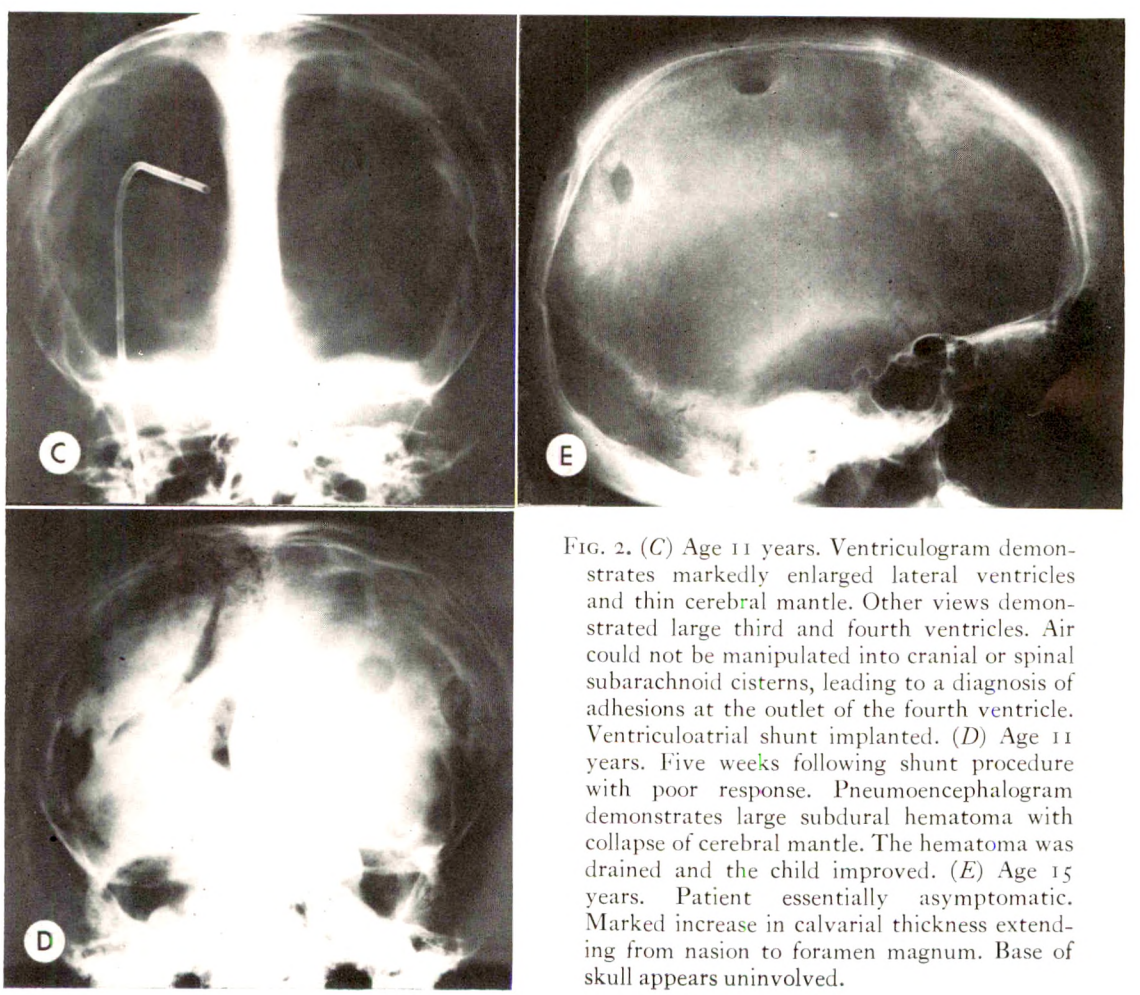


FIG. 2. (*C*) Age 11 years. Ventriculogram demonstrates markedly enlarged lateral ventricles and thin cerebral mantle. Other views demonstrated large third and fourth ventricles. Air could not be manipulated into cranial or spinal subarachnoid cisterns, leading to a diagnosis of adhesions at the outlet of the fourth ventricle. Ventriculoatrial shunt implanted. (*D*) Age 11 years. Five weeks following shunt procedure with poor response. Pneumoencephalogram demonstrates large subdural hematoma with collapse of cerebral mantle. The hematoma was drained and the child improved. (*E*) Age 15 years. Patient essentially asymptomatic. Marked increase in calvarial thickness extending from nasion to foramen magnum. Base of skull appears uninvolved.

All patients with carcinoma of the esophagus and cardia seen at the Mayo Clinic from 1946 through 1965 were included in the study for a total of 1,657 patients divided into 4 groups depending on the anatomic location of the lesion. The division into 4 groups was used because each constituted a separate surgical problem from the technical standpoint and the therapeutic approach differed for each location: (1) tumors situated below the hypopharynx, and mostly extrathoracic, were classified as being located in the cervical region; (2) tumors in the upper one-half of the intrathoracic esophagus were those requiring anastomosis above the arch of the aorta; (3) lesions that would permit an anastomosis below the arch and did not involve the esophagogastric junction were in the lower half of the intrathoracic esophagus; and (4) the final group consisted of carcinomas of the cardia with the lesion involving the esophagogastric junction.

Nearly 50 per cent of the tumors were located at the cardia, one-quarter of the lesions were found in the upper part of the esophagus and the remainder in either the cervical or lower portions of the esophagus, the latter predominating 2 to 1.

The analysis of data was carried out with the patients divided into 2 groups depending on whether or not the diagnosis of carcinoma was made in the period from 1946 to 1955 or in the second period from 1956 to 1965. In the earlier group were 909 patients and in the later group 748 patients. The review of data revealed an increase in operability rate from 46 to 67 per cent between the 2 periods, and in resectability rate from 27 to 45 per cent, whereas the hospital mortality rate after resection was lowered from 15.9 to 11.7 per cent. The mortality rate was highest after total esophagectomy with colonic interposition—a procedure that did not give better survival rates than did partial esophagectomy with esophagogastrostomy.

The over-all 5 year survival rate was doubled to 9 per cent, mainly because of the increase in resectability and the decrease in hospital mortality.

Results of surgical treatment were best for squamous cell carcinoma of the esophagogastric junction and at the lower thoracic level of the esophagus, but radiation therapy was the most effective means of treatment for carcinoma of the cervical and upper thoracic regions of the esophagus. Curative radiation therapy for squamous cell carcinoma resulted in a 1 year survival rate of 41 per cent. Resection as a palliative measure, 70 per cent of which was done for carcinoma of the cardia, gave good relief of dysphagia in most instances and 19 per cent of the patients survived more than 1 year. Patients with nonresectable lesions rarely lived more than 6 months but esophageal intubation gave worthwhile palliation of dysphagia in 70 per cent of patients so treated, and irradiation as a palliative measure for squamous cell carcinoma had beneficial effects in 50 per cent.

The best method of palliation in terms of survival was resection followed by radiation therapy. Of 10 patients with squamous cell carcinoma treated in this manner, 8 survived more than 1 year and 1 was alive and well at 7 years.—*Luther W. Brady, M.D.*

MARCOVE, RALPH C., MIKÉ, VALERIE, HAJEK, JOSEPH V., LEVIN, ARTHUR G., and HUTTER, ROBERT V. P. Osteogenic sarcoma under the age of twenty-one: a review of one hundred and forty-five operative cases. *J. Bone & Joint Surg.*, April, 1970, *52-A*, 411–423. (From: Bone Service of the Department of Surgery and the Department of Pathology, Memorial Sloan-Kettering Cancer Center, the Biomathematics Division, Cornell University Graduate School of Medical Sciences and Sloan-Kettering Institute, and the Division of Applied Therapy, Sloan-Kettering Institute, New York, N. Y.)

The authors review the experience at the Memorial Sloan-Kettering Cancer Center with the operative treatment and subsequent course of the central type of osteogenic sarcoma in patients under 21 years of age with operable tumors in the long bones. The criteria for selection of cases were established and the present report consists of 145 operative cases seen in the interval from January 1, 1949 through December 10, 1965.

The authors studied the patients by analysis of the interval from surgery to the onset of pulmonary metastasis and the interval from the onset of pulmonary metastasis to death. The onset of pulmonary metastasis is the first objective sign of disease activity following definitive surgery.

There was no evidence in this series of a statistical relationship between the date of operation, race, sex, preoperative duration of symptoms or preoperative irradiation on the course of the disease. Age seems to have a significant correlation in that lesions in younger patients metastasize sooner. The location of the tumor also seems to correlate. Lesions in locations other than the lower end of the femur take longer to metastasize in older patients. One hundred and twenty-one patients had pulmonary metastasis prior to the end of the study and died of their disease. Twenty-four patients were free of metastasis at the end of the study with a 5 year survival rate of 17.4 per cent.

Age and location of the tumor were found to affect the time of onset of pulmonary metastasis but not the 5 year survival rate. The first 2 years after definitive surgery were shown by the authors to be the critical period for studying the course of the disease. —*Luther W. Brady, M.D.*

PARRISH, FRANK F., and MURRAY, JOHN A. Surgical treatment for secondary neoplastic fractures: a retrospective study of ninety-six

sion of the cranial contents by producing bone as the suture is spread.[10] However, simple compression across the suture is not sufficient to cause fusion.[16]

Moss[10] calls attention to the importance of the "skull base-dura mater complex" in the sutural fusion process and provides evidence that deformities of the skull base lead to abnormal tensile forces being transmitted to the sutures via the dura. These forces are thought to be sufficient to initiate the osteogenic processes which lead to premature fusion.

A review of the 7 cases included in this study did not produce any definite reason why these children should have developed the skull changes described while the majority—similarily treated—did not. Clues did emerge, however, which may prove significant as more experience with this condition is presented.

This group of 7 was atypical when compared to the entire group tabulated by Long and associates.[8] Four of the seven had communicating hydrocephalus, more than twice the incidence in the entire series. Three of the seven had posterior fossa adhesions with a history of meningitis compared to an incidence of only 10 per cent with meningitis in the complete series. In addition, these 7 patients had undergone more operative procedures on the average than the entire group, 3.4 compared to 1.5.

A subdural hematoma was diagnosed in one of the patients following shunt surgery (Fig. 2*D*). She was the only member of the series, however, in whom hemorrhage was diagnosed, so it seems doubtful that sub-

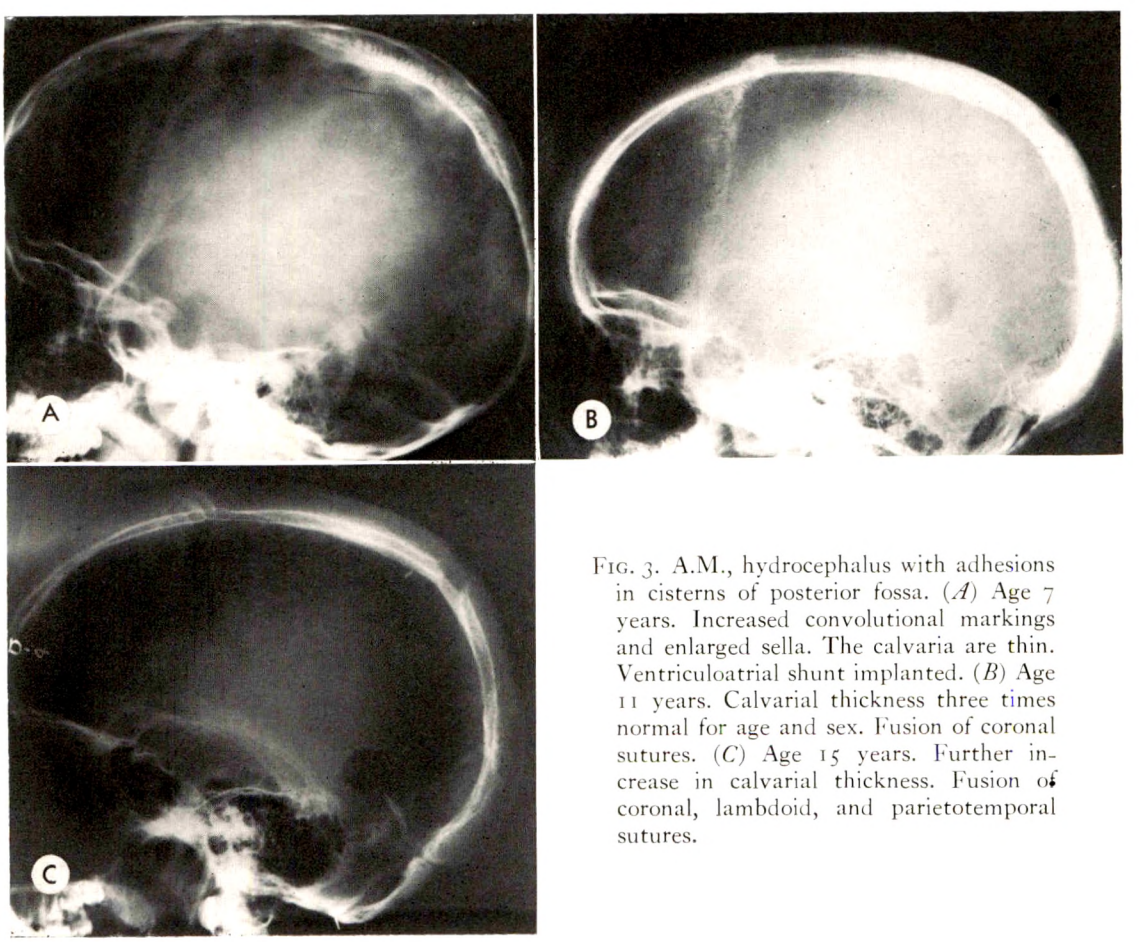


Fig. 3. A.M., hydrocephalus with adhesions in cisterns of posterior fossa. (*A*) Age 7 years. Increased convolutional markings and enlarged sella. The calvaria are thin. Ventriculoatrial shunt implanted. (*B*) Age 11 years. Calvarial thickness three times normal for age and sex. Fusion of coronal sutures. (*C*) Age 15 years. Further increase in calvarial thickness. Fusion of coronal, lambdoid, and parietotemporal sutures.

patients. *J. Bone & Joint Surg.*, June, 1970, *52-A*, 665–686. (From: The University of Texas at Houston M. D. Anderson Hospital and Tumor Institute, Houston, Texas.)

The authors' experience for 20 years has demonstrated that surgical management of neoplastic fractures of the long bones is feasible and advantageous for the majority of patients. Some become ambulatory who otherwise would have remained bedridden; many others achieve a significant increase in their general functional level.

Patients included in this study were operated on at the M. D. Anderson Hospital and Tumor Institute between July 1948 and July 1968. There were 96 patients who had 104 neoplastic fractures. One hundred fourteen operations were performed on patients, divided into 2 groups: Group I, 57 patients, all women, who had primary carcinoma of the breast; Group II, 39 patients, whose primary tumors arose in other sites. All operations for complete fractures were performed a few days after the fractures occurred.

The median age of both groups, when combined, was 58 years at the time of the fracture.

The following requirements must be fulfilled before undertaking a surgical procedure for a neoplastic fracture of a long bone:

1. The patient's general condition and life expectancy must be sufficiently good to justify the procedure.
2. The surgeon must be convinced that the operation will be more beneficial than the closed treatment.
3. The quality of the bone both proximal and distal to the fracture must be adequate for stable fixation.
4. The procedure must expedite mobilization of the patient or facilitate general care.

Surgical treatment was most applicable to fractures of the femur. Traction or a plaster spica should be supplanted by intramedullary fixation for fractures of the diaphysis of the femur, by nail-plate devices for intertrochanteric fractures, and by prostheses for fractures of the femoral head and neck. With other methods of treatment the patients must remain in bed.

Plaster splints were used for the majority of patients with neoplastic fractures of the tibia and for the few with supracondylar fractures of the femur. With this treatment satisfactory stability and relief of pain were achieved and healing of the fracture took place.

Posterior plaster splints, bulky compression dressings, or sling-and-swathe bandages were usually adequate for fractures of the long bones of the upper extremity, providing stability of the fracture and affording sufficient comfort without undue restriction of activity. Rarely was internal fixation of the humerus indicated.

If a patient has hypercalcemia secondary to skeletal metastases, early mobilization after fracture is especially important.

The theoretic dangers of dissemination of tumor cells by this type of surgery may be ignored.

Presence of multiple fractures in itself is not a valid reason for withholding surgical treatment.

Twenty-two intertrochanteric fractures, constituting 21 per cent of the fractures in the series, were observed in 21 patients.

Fixation with an intramedually rod was used for 51 fractures in the subtrochanteric region and the mid-shaft of the femur (48.5 per cent of all the fractures).

Twenty-five of the 96 patients (26 per cent) received radiotherapy to the affected region of the bone before the fracture. Postoperatively, 45 (43.2 per cent) were given radiotherapy, cobalt 60 being used in 9 of the patients. Average tumor dosage was 2,500 rads, given in 13 days.

In all the patients in whom radiotherapy was administered, no adverse effect was identified.

Nineteen surgical complications occurred in 17 (18 per cent) of the 96 patients in this series.

Patients with primary carcinoma of the breast and those with multiple myeloma had the most promising outlook. The prognosis of patients with neoplastic fractures from primary tumors in the lung and those with undifferentiated metastatic carcinoma was less favorable. The median survival rate of these 10 patients following the surgical procedures was only 2 months.

Of the 21 patients with intertrochanteric fractures stabilized with nail-plate devices, 3 (all with cancer of the breast) became fully ambulatory, 6 were weight-bearing with support, 9 confined to wheel chairs, and 3 were bedridden.

Evaluation of the results in relation to the fracture and implant is difficult because of the short median survival.

Incidence of neoplastic fractures is extremely high in patients with carcinoma of the breast and skeletal disease.

In the neoplastic fractures that healed after open reduction and internal fixation, the nature of the callus surrounding the fracture was of interest. Histologic sections of the fracture site demonstrated the extraordinary ability of bone to heal and regain its original integrity by remodeling.

Irradiation is the most effective of the ancillary therapeutic modalities for neoplastic fractures.—*Stephen N. Tager, M.D.*

ENRIGHT, L. P., TRUEBLOOD, H. WARD, and NELSEN, THOMAS S. The surgical diagnosis of abdominal Hodgkin's disease. *Surg., Gynec. & Obst.*, May, 1970, *130*, 853–858. (From: Department of Surgery, Stanford University Medical Center, Stanford, Calif.)

Operations on patients with Hodgkin's disease are

dural hematoma with resultant collapse of the cerebral mantle plays an important role in skull thickening.

It is tempting to relate these features of the 7 patients to the experimental observations cited above regarding premature sutural closure; namely, that alterations in the dura or periosteal trauma can initiate sutural fusion. How these differences from the larger unaffected group relate to the increased calvarial thickness remains unclear.

A recent report[7] cites 7 cases of seizure disorders in children and adolescents in whom generalized skull thickening was found following prolonged use of dilantin. However, only 3 patients in the present series had been treated with this medication, indicating that dilantin therapy was not a contributing factor.

Clinical significance of this post-shunt syndrome as suggested by Moseley *et al.*[9] relates to the fact that these children are now dependent upon the proper functioning of the shunt tube for maintenance of normal intracranial pressure. To date, however, none of these 7 patients have had documented episodes which could be blamed upon failure of the shunt with subsequent rapid elevation in intracranial pressure. In addition, further growth of the brain will be impeded by a fixed cranial capacity.

Differential diagnosis should present little difficulty. Several other conditions result in generalized increased thickness of the skull in childhood including healed rickets, severe longstanding anemias,[15] and as a part of certain rare syndromes, such as craniometaphyseal dysplasia,[14] but these conditions do not exhibit premature sutural fusion. The presence of the shunt apparatus should suggest the correct etiology for the increased skull thickness and sutural fusion.

SUMMARY

A review of a large series of patients treated surgically for hydrocephalus yielded a group of 7 children with generalized thickening of the calvaria (averaging 2.5 times normal for age and sex) and premature fusion of the cranial sutures of a varying extent.

The reasons why these few patients (out of a much larger post-shunt group not so affected) show the changes described is unknown, although some parallels with findings from animal studies can be drawn.

The clinical significance of this syndrome appears to derive primarily from the premature sutural fusion, which may impede brain growth and render the child more dependent upon the proper function of his shunt tube for maintenance of normal intracranial pressure.

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performed for diagnosis, for cure, for the treatment of concurrent disease, and for disorders secondary to the disease itself, such as hypersplenism or compression of vital structures by lymphoid masses. The surgeon encounters the patient with Hodgkin's disease primarily to perform a diagnostic procedure, often in the form of a simple lymph node biopsy. Operative therapy as a curative intervention in Hodgkin's disease is championed by only a few surgeons and then only in very selected instances.

The authors report their experience at Stanford University Medical Center where supervoltage radiation therapy constitutes the treatment of choice for Hodgkin's disease, both at first presentation and when symptoms secondary to compression by lymphoid masses occur; *i.e.*, trachea, superior vena cava, or spinal cord compression.

There were 68 consecutive patients with Hodgkin's disease who underwent abdominal exploration by splenectomy, liver biopsy and periaortic lymph node biopsy. In this group of patients, mortalities occurred and the morbidity rate was small. Significant information resulting in restaging and alterations in therapeutic course was discovered by laparotomy in 50 per cent of those patients previously having a complete work-up including lymphangiograms. Laparotomy affords otherwise unavailable diagnostic information in Hodgkin's disease which can potentially improve over-all cure rates for the disease process.

The authors emphasize the positive aspects of abdominal exploration, noting that 35 per cent (24 patients) had more severe involvement than suspected. The resultant treatment of these previously undefined areas of disease, in their belief, results in a higher over-all cure rate for these patients. Removal of the spleen involved with Hodgkin's disease has the additional advantage of avoiding high dose radiotherapy to the left upper quadrant since this will increase the risk of radiation pneumonitis of the lower lobe of the left lung and radiation nephritis of the left kidney.—*Luther W. Brady, M.D.*

BLAKENEY, CATHERINE M., and CULLUM, PATRIC A. A single hepatic metastasis from bronchial carcinoid treated by right hepatectomy. *Brit. J. Surg.*, March, 1970, *57*, 237–238. (From: Department of Thoracic Surgery, Westminster Hospital, London, England.)

Bronchial adenoma and its relationship to the carcinoid syndrome was reported in 1956 by Kincaid-Smith and Brossy. By 1965, 19 cases had been documented, and a further 3 added to the literature by Melmon, Sjoerdsma and Mason.

The authors report a case with florid symptoms appearing 6 years after resection of the primary adenoma, without direct clinical evidence of secondary involvement. The patient was successfully treated by right hepatectomy for a single metastatic lesion.

This patient represents a case of bronchial carcinoid syndrome with a single hepatic lesion and the only one who has had a right hepatectomy followed by complete loss of symptoms and reduction of urinary 5-HIAA to normal levels. It exemplifies the need to search for hepatic involvement after a long latent period, regardless of the absence of clinical signs of enlargement of the liver.—*Luther W. Brady, M.D.*

RADIOISOTOPES

WINCHELL, H. S., SANCHEZ, P. D., WATANABE, C. K., HOLLANDER, L., ANGER, H. O., and MCRAE, J. Visualization of tumors in humans using ^{67}Ga-citrate and the Anger whole-body scanner, scintillation camera and tomographic scanner. *J. Nuclear Med.*, July, 1970, *11*, 459–466. (From: Donner Laboratory, University of California, Berkeley, Calif.)

Gallium 67 citrate was administered to 18 patients with a variety of neoplasms (5 lymphomas, 13 carcinomas) to evaluate the distribution of the lesions. Images were obtained with the Anger scintillation camera, whole body scanner, and tomographic scanner.

The optimum time for imaging was 48 hours after radionuclide administration. After this time there was little change in nuclide distribution. Twelve out of 20 (5/5 with lymphoma and 7/13 with carcinoma) had visualization either of the tumor or metastasis. Six of the 18 had no uptake in the tumor.

Although Ga^{67} localized in a wide variety of tumors, this localization frequently occurred in only some of the lesions present in a patient. The variation in normal uptake in joints, pelvis, and spleen made it difficult to evaluate the presence of tumors in these regions. In addition, gallium 67 accumulated in the breast of a patient with gynecomastia; and in the vagina and uterus of a patient without demonstrable pelvic pathology.

The reason for the accumulation of gallium 67 in tumors, and the variation in accumulation in several lesions in the same patient is uncertain. Whether this is due to lower intracellular pH causing dissociation of the gallium-chelate (due to increased metabolic activity) or to a specific binding is unknown.

All imaging devices had good resolution of the lesions which were detected.

The tomographic scanner had the additional advantage of indicating the depth of the tumor. Gallium 67 was found to give information about tumors that was not found by other means, suggesting that it may be useful in evaluating the presence and distribution of malignant neoplastic lesions in human subjects.—*H. William Strauss, M.D.*

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CALVARIAL THICKENING AFTER DILANTIN MEDICATION*

By KENNETH R. KATTAN, M.D.
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DILANTIN (diphenylhydantoin) is widely used and extremely effective in the management of patients with epileptic convulsions. The complications are legion: neurologic, gingival, hematologic, reticuloendothelial, metabolic, dermal, and cardiac involvement have been reported.[8,10–12] These were discussed in an excellent review by Sparberg in 1963.[11]

We have observed the development of widening of the calvaria, mainly due to thickening of the diploic space, in several patients who have received dilantin. A search of the literature indicates that this effect of dilantin has not been previously reported. Porosis at the metaphysis of the growing long bones has been reported as a rare complication of dilantin medication,[7] but none of the patients in our study showed this change.

MATERIAL AND METHOD

The hospital charts of 83 epileptic patients were reviewed, and the patients were classified into 4 groups (Table 1).

Group I included patients who received at least 200 mg. of dilantin regularly for 3 years or more. They may also have received other anticonvulsant medications.

Group II consisted of those patients who received dilantin for less than 3 years, or who did not take the drug regularly, or whose records were incomplete.

Group III was made up of patients who received anticonvulsant medication other than dilantin or none at all.

Group IV were nonepileptic patients, all with some kind of mental disorder.

In order to exclude any patient with sickle cell trait from the study, only white patients were included.

REPORT OF CASES

The following 3 patients are reported in more detail because they are the only ones with calvarial thickening in whom early roentgenograms prior to medication were available.

CASE I. This 14 year old boy has been epileptic since he was 5. In 1965, at the age of 11, he was admitted to Longview State Hospital because of incorrigible behavior at home and at school. He was started on 100 mg. of dilantin twice daily, and this regimen has been continued to the present.

Skull roentgenograms on admission were normal (Fig. 1*A*); however, 3 years later, there has been remarkable calvarial thickening (Fig. 1*B*). The diploë is wide and porotic, although the inner and outer tables remain unchanged. There is slight thickening of the posterior clinoids, again mainly in the diploë. The vertebral bodies and pelvis show questionable increased density and coarsened trabeculation.

The skull changes simulate those seen in the hemolytic anemias, but many blood examinations during the entire time of hospitalization showed no sign of anemia. His I.Q. was 83 and 69 (high dull normal) on two different occasions.

CASE II. This 17 year old boy was been epileptic since the age of 6, when he contracted

TABLE I

SUMMARY OF PATIENTS

	No. of Patients	Calvarial Thickening		
		Present	Questionable	Absent
Group I	22	7	3	12
Group II	46	4	0	42
Group III	15	1	1	13
Group IV	19	1	1	17

* Presented at the Seventieth Annual Meeting of the American Roentgen Ray Society, Washington, D.C., September 30–October 3, 1969, under the title "Bone Changes After Dilantin Treatment."
From the Department of Radiology, University of Cincinnati College of Medicine and Longview State Hospital, Cincinnati, Ohio.

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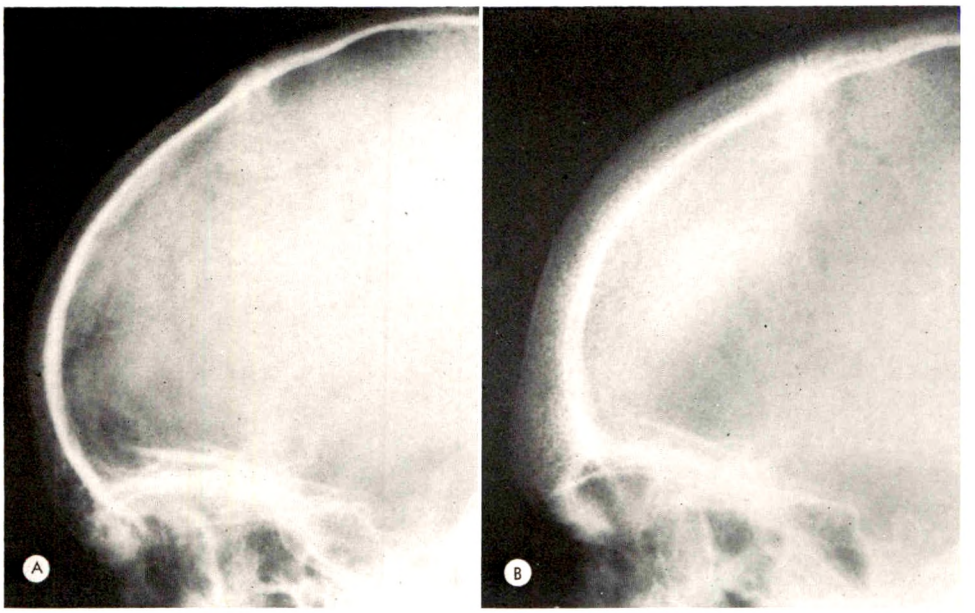


Fig. 1. Case 1. (*A*) Skull roentgenogram on admission showing normal calvaria. (*B*) Same area after 3 years of medication showing marked widening of the diploë. Note the thickening of the posterior clinoids.

encephalitis. Since then he has had epileptic seizures and behavioral changes. In 1963, at the age of 11, the seizures became more frequent and he was admitted to Longview State Hospital. A daily dose of 300 mg. of dilantin was begun. It was later increased to 400 mg. daily. He also received other tranquilizers and anticonvulsant drugs during the time of hospitalization.

Comparison of the admission skull roentgenograms with those taken 5 years later (Fig. 2, *A–D*) shows marked increase in the width of the diploë. Blood cell counts during his hospitalization showed no anemia. Platelet counts on two occasions were 92,500 and 77,500. Bone marrow examination done especially for this study in 1968 was normal. His I.Q. at this time was 57.

Case III. This white woman, now 22 years old, has been epileptic since the age of 12. She was admitted to Longview State Hospital in 1962 at the age of 16 because of the convulsive disorder and behavioral reaction. A dilantin regimen of 300 to 400 mg. daily was started and has been continued regularly since that time. Other medications were also prescribed, namely tranquilizers and anticonvulsants.

Roentgenograms of the skull in 1962 and 1968 show an evident increase in the thickness of the calvaria (Fig. 3, *A* and *B*).

Red and white blood cell counts are normal. Her I.Q. on admission was 89 (mild mental deficiency).

DISCUSSION

A random study was attempted, but was not successful. Most of the patients were in a mental institution, and some were mentally retarded. That severe epileptics are usually hospitalized and receive medication regularly cannot be disregarded; nor can it be ignored that some of the patients received other medications in addition to dilantin.

Therefore, to make the study as impartial as possible, calvarial thickness in the 4 groups was evaluated without knowledge of the patient's history. The criteria set by Hansman[4] for skull thickness were not strictly followed because they are for thickness at only one point, namely the lambda in the lateral projection.

However, the high percentage of thickened calvaria in Group 1, compared with the other 3 groups, is striking. (It is especially striking in Case 1.) The age at which medication is given probably plays a contributing role to the presence and de-

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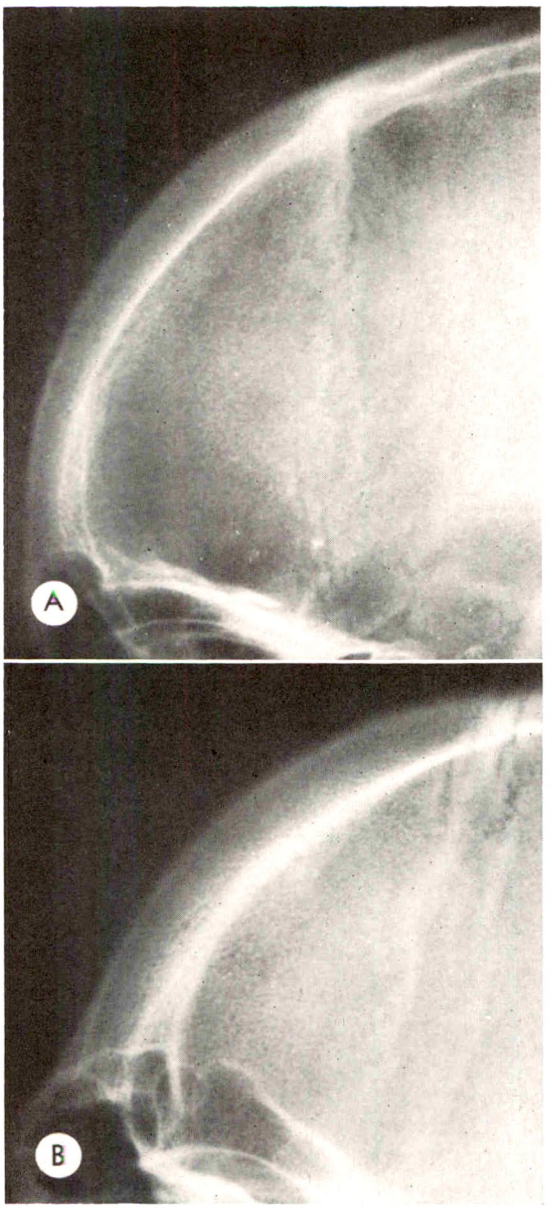


FIG. 2. Case II. (*A*) Normal lateral skull roentgenogram on admission. (*B*) After 5 years of dilantin medication. Note the increased calvarial thickening.

gree of the thickening.[5] Of the 7 patients in Group I who showed calvarial thickening, at least 4 were started on dilantin in their teens; in another, the age was not known. On the other hand, of the 12 patients in Group I who showed no changes in the calvaria, all but one had been put on dilantin after the age of 20, although epileptic seizures had started before that time in 6.

There are many causes of calvarial thickening. The chronic hemolytic anemias are the most common culprits;[2,9] however, in early childhood iron deficiency anemia[1] and secondary polycythemia are sometimes implicated.[9] The mechanism of the thickened calvaria in these conditions is the bone marrow hyperplasia, which widens the diploë. This was discussed in an excellent review by McAfee.[9]

The same report describes some of the other diseases that accompany hyperostosis of the skull. The healing stages following prolonged rickets, renal osteodystrophy, hypoparathyroidism, and microcephaly are only a few of those cited. Abnormal thickening of the calvaria associated with mental retardation due to microcephaly was discussed by Dorst.[3] In these patients, the digital markings are usually lacking, the sutures are narrow, and the mastoid air cells and frontal sinuses are large and hyperpneumatized. Paget's disease and osteogenesis imperfecta[6] are among the other reported causes of calvarial thickening.

The I.Q. of the patients with thickened calvaria after dilantin medication (Group I) was found to be comparable to that in the other groups. Therefore, if microcephaly plays any role, it is not the only one. Obviously, neither anemia nor microcephaly are the cause of the calvarial thickening in these patients.

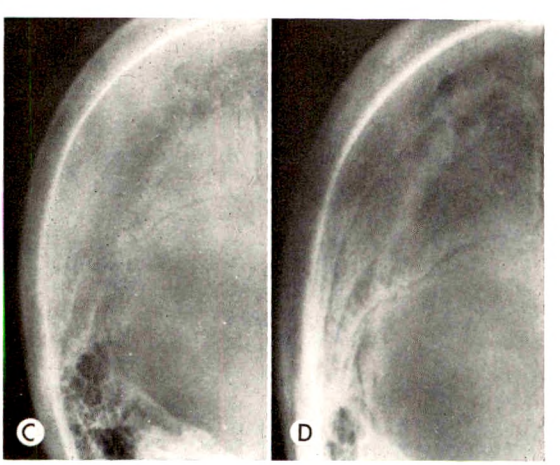


FIG. 2. (*C*) Parietal area in the Towne view on admission. (*D*) Five years later.

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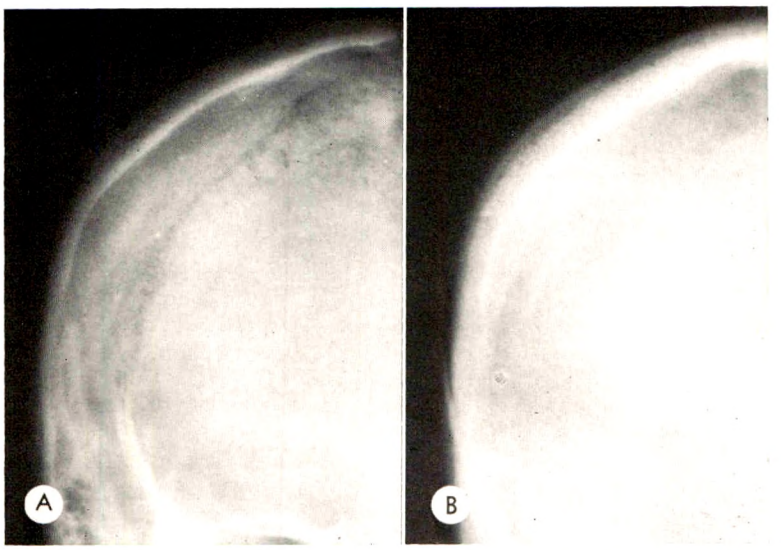


Fig. 3. Case III. (*A*) Anteroposterior skull roentgenogram on admission. (*B*) Thickened calvaria after 6 years of dilantin.

SUMMARY

Increase in the thickness of the calvaria, mainly in the diploic space, was noticed in epileptic patients receiving dilantin for prolonged periods. The younger the patient, the more prominent were the changes. Three white teenage patients are reported with skull roentgenograms before and 3 or more years after the institution of dilantin medication. An additional 4 patients on prolonged dilantin therapy showed a thick calvarium.

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THE CONTRACTING SKULL*

INWARD GROWTH OF THE INNER TABLE AS A PHYSIOLOGIC RESPONSE TO DIMINUTION OF INTRACRANIAL CONTENT IN CHILDREN

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ALTHOUGH thickening of the cranium has been described by several writers[2,8,9] in cases of cerebral atrophy or relieved hydrocephalus, the fact that the internal dimensions of the brain case sometimes diminish appreciably has received little attention.[1] Perhaps this is because we are not accustomed to think that an organ, particularly a more or less solid bony box, actually may grow smaller. Nevertheless, in certain circumstances, inward growth of the inner table of the skull does indeed occur.

The 5 children described in Table 1 were found among 140 cases of hydrocephalus with operative shunting and longterm follow-up. All 5 were severely hydrocephalic when first seen. Their skulls were quite large. In each case cerebrospinal fluid pressure was well above normal initially. Their ages at that time ranged from 9 months to 7½ years. All had had cerebrospinal fluid shunting operations and most had had more than one shunt procedure. All thereafter developed distinct contraction of their internal skull measurements and calvarial

TABLE I

LESSENING OF INNER CALVARIAL DIMENSIONS AND SELLAR VOLUMES AFTER SHUNTING FOR HYDROCEPHALUS

Case	Type or Cause of Hydrocephalus	Internal Anteroposterior Diameter of Skull				Volume of Sella Turcica, $V=\frac{1}{2}(L\times H\times W)$[4] (Fig. 4)	
		Age of First Shunting (yr.)	Diameter at First Shunting	Diameter at Last Examination	Age Then (yr.)	Before Shunting	At Last Examination
1	Aqueductal stenosis (Fig. 1)	9/12	20.3 cm.	18.2 cm.	12 3/12	Length and height of the sella grew smaller. Sellar width could not be measured, and volume could not be computed	
2	Arachnoid cyst of posterior fossa	1 5/12	18.2 cm.	17.0 cm.	6 6/12	250 mm.^{3}	180 mm.^{3}
3	Communicating hydrocephalus (Fig. 2)	2 3/12	21.9 cm.	19.6 cm.	6 6/12	610 mm.^{3}	250 mm.^{3}
4	Atresia of foramina of Luschka and Magendie	2 7/12	22.5 cm.	21.4 cm.	9 11/12	720 mm.^{3}	280 mm.^{3}
5	Cystic astrocytoma of vermis	7 7/12	18.7 cm.	17.5 cm.	20 1/12	560 mm.^{3}	310 mm.^{3}

L = length; H = height; W = width.

* From the Departments of Radiology of Children's Hospital Medical Center and Harvard Medical School, Boston, Massachusetts.

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volumes. In 2 cases the internal anteroposterior diameter shrank more than 2 cm. The superoinferior and lateral dimensions also invariably grew smaller. In Case 1 and Case 3 about 25 per cent of the intracranial volume was lost; volume loss in the other 3 cases was less striking. This shrinkage in calvarial capacity occurred over periods of 4 to 13 years. It may still be slowly progressing. By visual estimation, internal calvarial capacity at the time of the last examination was slightly above normal for the age in 2 of these 5 cases, about normal in 1 case, and slightly below normal in 2 cases. How much of the cranium was then occupied by brain and how much merely by widened cerebrospinal fluid spaces is not known. The cerebrospinal fluid pressures during this period were normal or low on the occasions when they were measured. Some of these children are neurologically impaired (Fig. 1, *A*, *B* and *C*; and 2, *A*, *B* and *C*), as would be expected from the initial severity of their hydrocephalus.

Despite the moderate diminution in internal diameters of the skull, the diameters of the outer tables were still somewhat above normal at the time of the last skull roentgenograms in all cases.[6] External dimensions had slightly increased in the interval in some cases and had remained stable in others. Similarly, head circumference remained the same or increased slightly. Increase in diploic thickness, of course, accounts for this discordance between the outer table and the inner table. This thickening (Table II) naturally is reflected in outer table diameters as measured from skull roentgenograms and in head circumference as measured clinically. External measurements are therefore only first approximations of internal calvarial changes, especially in cases of chronic reduction in intracranial pressure and volume.

Some of the physiologic steps in the shrinkage of the skull capacity illustrated here are clear. Relief of intracranial hypertension has been shown to be accompanied by the laying down of laminated layers of new bone along the inner table.[3,7] Removal of part of the brain in growing rats is followed, at maturity, by smallness and thickness of the skull, especially on the side of the removal.[12] Endocranial osteoclasts (the cells resorbing bone at the dura to allow for brain growth) are found to be less numerous after brain removal, and ectocranial osteoclasts are more numerous.. Resorption by osteoclasts and bone formation by osteoblasts are, of course, the classic mechanisms whereby bone remodels itself to fit new surroundings and circumstances. Such remodeling must be one of the steps in

Table II

SKULL FILM MANIFESTATIONS OF LOW OR DECREASING INTRACRANIAL TENSION OR CONTENT

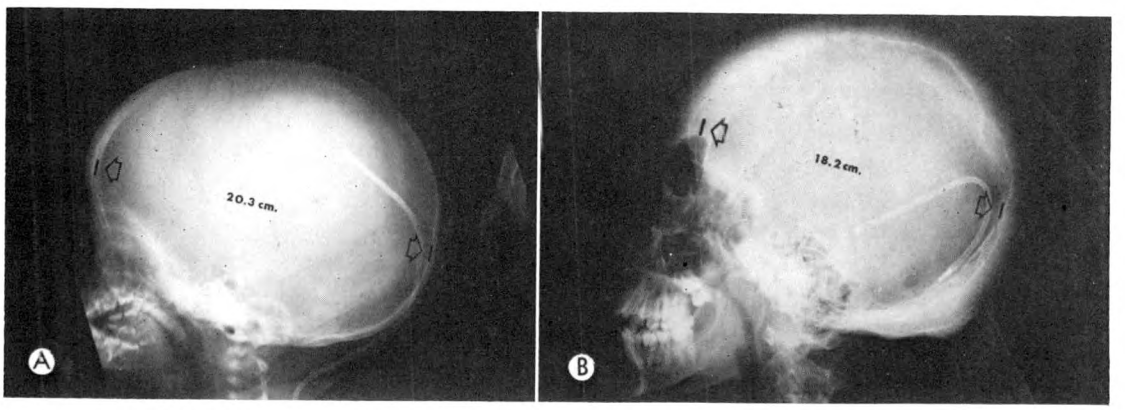
The responses listed first appear more promptly	
1. Concave Fontanelle	
2. Depressed Craniotomy Bone Flap	
3. Approximation or Overlapping of the Edges of a Previously Diastatic Fracture	
4. Abnormal Sutures	Too narrow for the age; narrower than before; premature closure of sutures or fontanelle; dense parasutural bone; persistent neonatal overriding or molding
5. Smooth Inner Table	Few convolutional or endocranial vascular landmarks; rounded, globular, featureless appearance; smooth, round orbital roofs
6. Thick Cranial Bones	Endocranial laminated new bone; early appearance of definable diploe; widened diploic space; diminution of internal diameters of the skull and calvarial capacity
7. Small Cranium	Smaller than normal for the age; low cranium-to-face ratio; small, sloping forehead
8. Small Lateral Sellar Profile, Small Sellar Volume, and Massive Dorsum and Posterior Clinoids	
9. Hyperpneumatized Sinuses and Mastoids, Elevated Petrous Ridges	
10. Supraorbital Ridge	
11. Unilateral or Local Development of One or More of the Above as a Response to Unilateral or Local Loss of Content	

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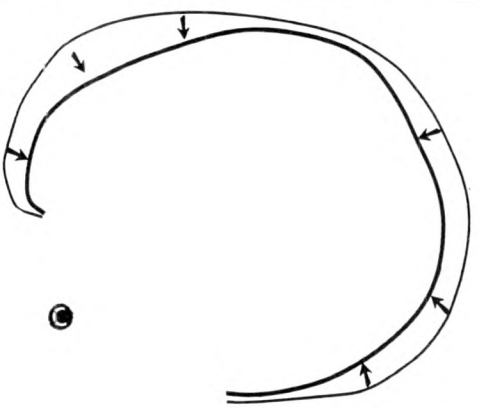
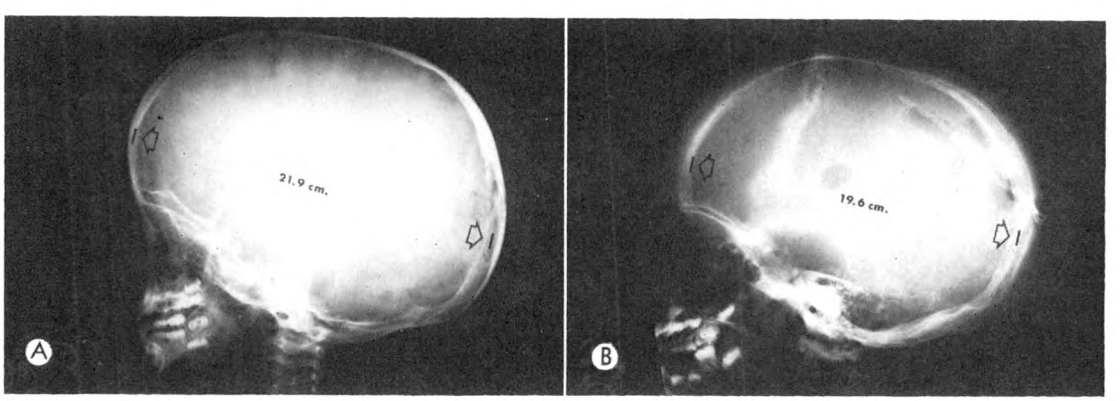


FIG. 1. Case 1. Aqueductal stenosis. (*A*) Lateral view of the skull at age 9 months, just after the first shunt procedure. The internal anteroposterior diameter is 20.3 cm. (*B*) Lateral view of the skull at age 12 years and 3 months. The internal anteroposterior diameter is 18.2 cm. (*C*) Tracings of the contracting inner table, from *A* and *B*. The patient is severely retarded.



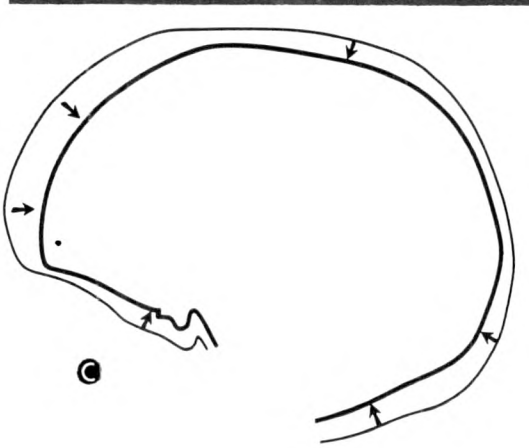


FIG. 2. Case 3. Communicating hydrocephalus. (*A*) Lateral view of the skull at age 2 years and 3 months, before the first shunt procedure. The internal anteroposterior diameter is 21.9 cm. (*B*) Lateral view of the skull at age 6 years and 6 months. The internal anteroposterior diameter is 19.6 cm. (*C*) Tracings of the shrinking inner table, from *A* and *B*. The patient is somewhat retarded and has occasional seizures.

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inward growth of the cranium. This inward growth involves bones formed in cartilage (sphenoid, occipital bone) as well as those formed in membrane. The vault remodels itself readily, the cranial base less readily.[12] Analogous instances of inward growth of bone are: (1) the restoration of spinal canal contours toward normal after the removal of an intraspinal tumor (Fig. 3, *A–D*); (2) the diminution in size of an orbit after enucleation;[10] (3) the return of the sella to normal after the relief of the pituitary hyperplasia that accompanies childhood hypothyroidism;[11] and (4) normalization of the sella after irradiation of a pituitary tumor.[11]

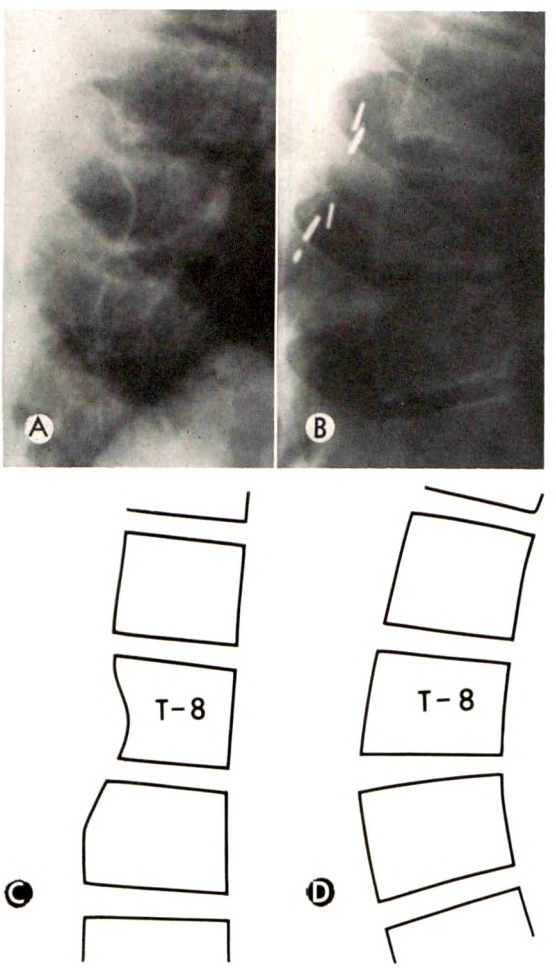


Fig. 3. Restoration of spinal canal contours toward normal after laminectomy and removal of an intraspinal extradural neurilemmoma. (*A* and *B*) Lateral views of the mid-dorsal region at the age of 14 years, and its appearance 8 months after the removal of the tumor. (*C* and *D*) Tracings of *A* and *B*. The pedicles similarly returned toward normal during this period. The laminae and spinous processes of the seventh and eighth dorsal vertebrae were removed during the operation at the site of the clips.

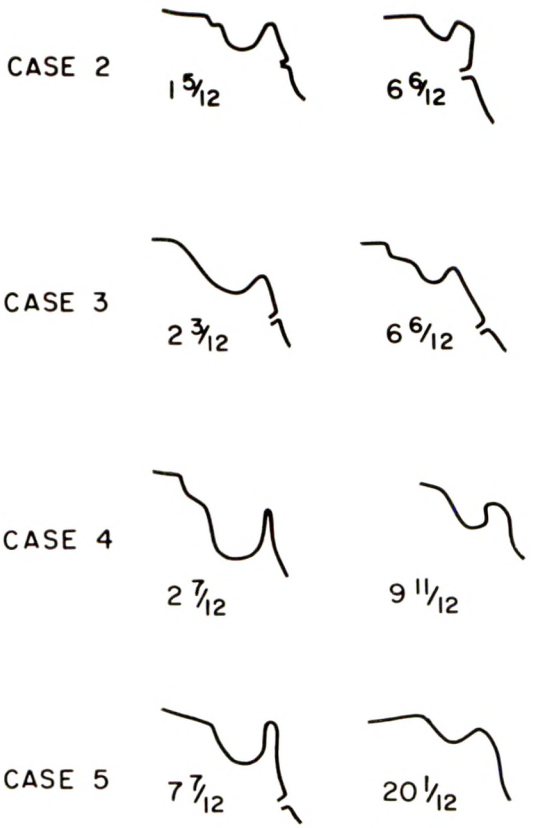


Fig. 4. Reduction in the lateral profiles of the sellae in Cases 2, 3, 4, and 5 after shunting for hydrocephalus. See Table 1. Although heights and lengths diminished as shown, widths increased slightly. Sellar volumes nevertheless grew much smaller, falling to or below the lower limits of normal.[4]

Inward growth of the inner table of the skull with actual loss of intracranial volume is therefore an uncommonly seen but physiologically reasonable accompaniment of relief of childhood hydrocephalus. Table II includes this phenomenon in the signs of low or lowered intracranial content or pressure. Some of the signs listed there, such as concavity of the fontanelle and depression of a craniotomy bone flap, obviously are acute manifestations of diminution in intracranial content and tension. Most of the others require months or years to develop.

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Those signs which are slow to develop tend to be slow to reverse themselves when circumstances change. The repertory of cranial responses to changes in content and tension is greatest in fetal life and infancy. Rapidity of response is also greatest in early life. The capacity of the skull to respond is progressively limited as adulthood approaches but is never completely lost.

One of the other signs given in Table II has received little attention heretofore. Upon review of these cases of relieved hydrocephalus, it became evident that the sella turcica in lateral profile was often remarkably small (Fig. 4). In each of the cases shown, the sella was shorter from front to back and shallower from top to bottom than before. But in each case the sella had grown slightly wider. The increased width of the sella may be a reflection of outward growth of the central mass of the sphenoid bone to take up slack in the middle fossae. Despite this increased width, the sellae were so short and shallow that total volume was distinctly less than before. In fact, sellar volume had fallen to or below the lower limits of normal (Table I),[4] although the sellae were sometimes so cramped and misshapen that accurate measurement was difficult. This diminution in sellar volume is proportionately greater than the general loss of intracranial volume described above. Perhaps feebleness of pulsation of the anterior recesses of the third ventricle is a local factor further promoting sellar shrinkage. Children with primary microcephaly also tend to have sellae that are small in lateral profile; in such cases, the posterior clinoids and dorsum may be quite large and massive.[5]

SUMMARY

One of the responses of the skull to diminution in content or pressure is thickening of the cranium. In a few cases new bone slowly laid down along the inner table of the skull, after shunting for severe childhood hydrocephalus, diminishes internal calvarial dimensions and capacity appreciably. The inner table actually grows inward toward the retreating brain and intradural contents. This inward growth of the inner table may proceed in spite of stability or slow increase in outer table dimensions and head circumference.

Measurement of outer table diameters and head circumferences in such cases fails to reflect internal volume changes.

The sella sometimes becomes quite small.

The probable physiologic basis of this shrinkage of calvarial capacity is presented briefly.

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LAMINOGRAPHY OF THE MASTOID IN THE BASAL PROJECTION*

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SELECTED projections to show specific anatomic structures are just as important in tomography as in routine roentgenography of the mastoid. The basal projection has received very little attention[2,3] due to technical difficulties, but nevertheless it is an excellent method for viewing the internal auditory canal and meatus as well as the semicircular canals.

The purpose of this paper is to review the normal roentgen anatomy as shown by laminography in the basal projection and also some of the clinical uses of the projection.

TECHNIQUE

Most patients are unable to retroflex the head far enough to assume the basal projection for any prolonged period of time. In order to overcome this difficulty, we have utilized a second table to be used with a tilting table laminographic unit capable of hypocycloidal movement (Fig. 1). The angle between the 2 tables is so arranged to suit the needs of the individual patient. Reid's baseline is positioned parallel to the laminographic table top and assurances are then taken to be certain that the sweep of the tube clears the patient's body.

Similar results can be obtained by using a rotating-type pneumoencephalographic chair and placing the laminographic device in an upright position to obtain the necessary angulation.

Exposure factors for laminography of the mastoids are: 68–75 kv., 240 mas., using a 0.3 mm. or 0.6 mm. focal spot tube. The central beam of the x-ray tube should be directed 0.5 cm. anterior to the external auditory canal when the patient is in the basal projection. Scout roentgenograms have been of limited value because the patient frequently moves. Sections, 1 mm. thick, are made at 1 mm. intervals for a total of 6 exposures, starting 0.5 cm. cephalad to the external auditory canal and moving caudally. Adjustments in positioning and technique are made following review of the laminograms in the initial projections. The right and left sides should be recorded separately to lessen scatter.

NORMAL ANATOMY

The roentgen anatomy is correlated with selected histologic sections of an autopsy specimen for better visualization of the structures. Roentgenograms were obtained by placing a patient in the basal projection and making 1 mm. sections at 1 mm. intervals with a hypocycloidal movement of the laminographic device.

The roentgenograms demonstrated were chosen to give an introduction to important structures. Comparison was then made to decalcified histologic sections representing a composite that corresponds to the roentgen examination.

Figure 2, *A* and *B* is a section through the inferior-most portion of the petrous pyramid. The intracranial opening of the cochlear aqueduct is consistently visualized in the basal projection and should not be mistaken for a portion of the internal auditory canal. It lies immediately adjacent to the jugular foramen. The carotid canal is immediately adjacent to the hypotympanum. The thin tympanic membrane, which is well demonstrated on the ana-

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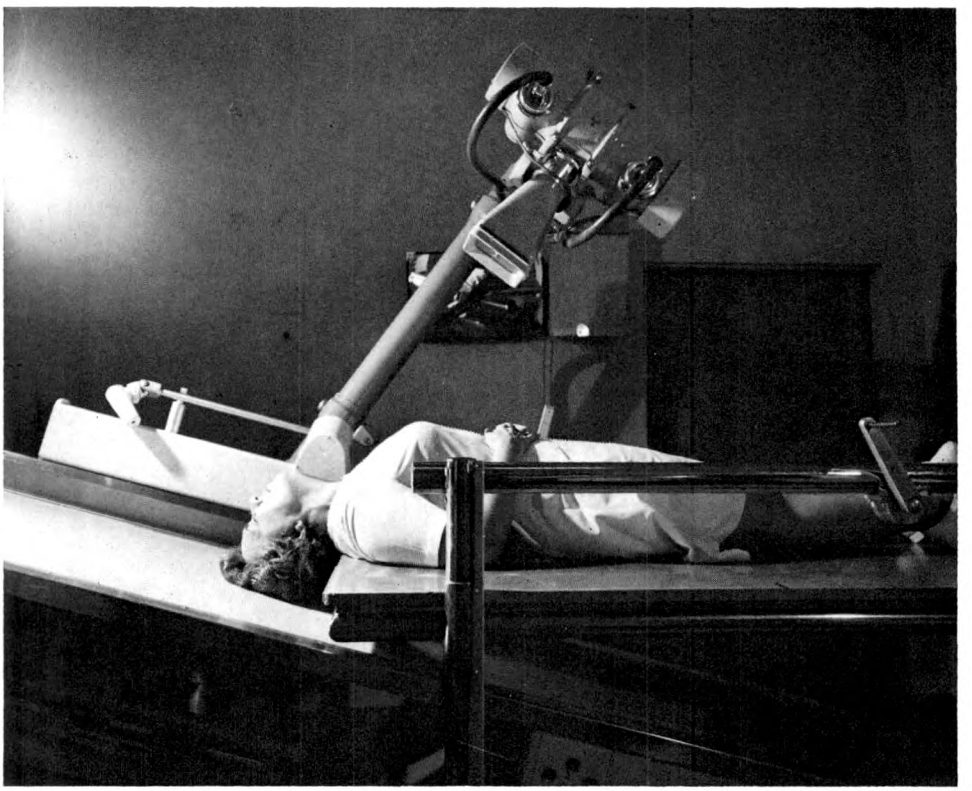


FIG. 1. Two tables are used to support the patient, with the head in the extended position. The angle that the laminographic table makes with the patient support table is arranged to give maximum comfort to the patient and yet allow the x-ray tube to clear the body during its sweep while maintaining Reid's baseline parallel to the x-ray film. A similar arrangement can be obtained with the x-ray table vertical and utilizing a rotating chair for patient support. The magnification factor is approximately 1.3 and a 0.6 mm., or a 0.3 mm. focal spot high speed rotating anode tube is used for exposure.

tomic specimen, cannot be visualized roentgenographically.

Section taken 4 mm. more cephalad (Fig. 3, *A* and *B*) than the previous laminogram shows that the inferior portion of the cochlea has been entered. The basal turn of the cochlea can be traced into the vestibule. Approximately 2 mm. medially and inferior to the vestibule is the round window of the cochlea. Its exact position can only be approximated on the roentgenogram. The laminographic cut is now cephalad to the external auditory canal and passes through the mesotympanum. The jugular foramen is no longer in sharp focus. Due to a slight difference in angulation of the histologic section as compared to the roentgenogram, the oval window niche is not seen on the histologic section (Fig. 3*A*).

Section 1 mm. cephalad (Fig. 4, *A* and *B*) to the previous cut passes through the cochlea and vestibule. The oval window, together with the footplate of the stapes, can be seen. The malleus and incus are visible in the epitympanic recess. A faint structure, possibly representing the short process of the incus, is seen directed posteriorly.

Figure 5, *A* and *B*, taken at 1 mm. cephalad, reveals the horizontal semicircular canal coming into view. Immediately adjacent are the mastoid antrum and the aditus leading anteriorly from the antrum into the epitympanic recess. The internal auditory canal and meatus are in focus, and

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the exit of the facial nerve canal from the lateral extremity of the internal auditory canal is visible.

Figure 6, *A* and *B*, at 3 mm. more cephalad, shows the internal auditory canal fading out of focus. The anterior limb of the superior semicircular canal is seen in cross section. The crus commune, which is formed by the posterior limb of the superior semicircular canal and the superior limb of the posterior semicircular canal, is in sharp focus.

MATERIAL

Initially patients were examined in the basal projection in a random fashion until the details of the anatomy became clear. The roentgen findings were sufficiently revealing that basal laminography was added as the projection of choice when the question of acoustic neuroma was to be resolved. Sixty consecutive patients were then investigated in an attempt to define the limits of normal and the specific findings of acoustic neuroma. We were successful in every instance in obtaining satisfactory basal laminography. As one might expect, most of these patients were in the older age group.

FINDINGS

Measurements of the internal auditory canals at the level of the porus acusticus were made on the basal as well as the anteroposterior laminograms. In addition, the length of the posterior wall was measured in both the basal and anteroposterior laminograms. The range of normal is so great from individual to individual as to make specific measurements useless.[1,4] Of considerably greater importance was the sharpness of definition and the exact position of any erosion of the internal auditory canal and/or porus acusticus as seen in any projection. The general configuration could be considered as "funnel-shaped" or the canal could have parallel walls assuming a "tube shape" in the basal projection. Sixteen instances of parallel wall were found, while the remainder of the 60 patients had funnel-shaped canals (Fig. 5, *A* and *B*).

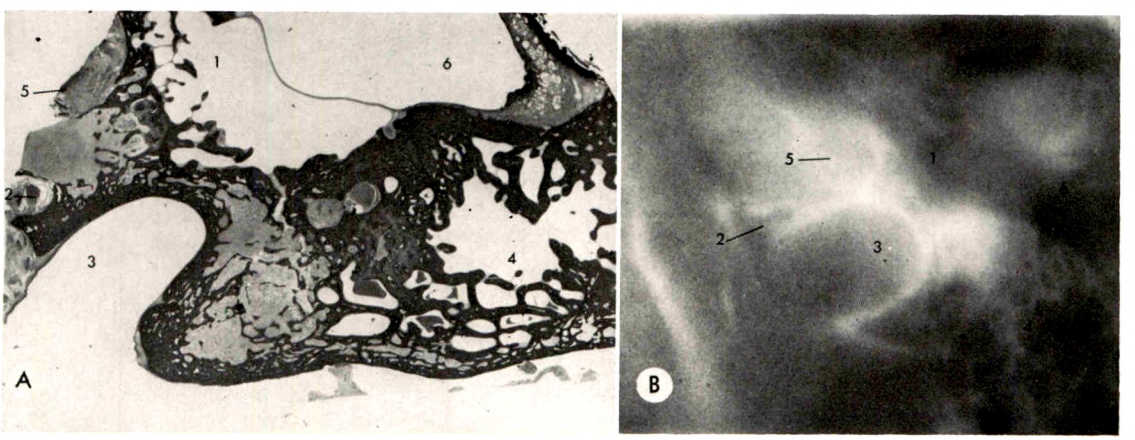


Fig. 2. (*A*) Histologic section and (*B*) basal laminogram of a normal right mastoid. Anterior is at the top of the section and posterior is at the bottom. The histologic section is not of the same living patient that was roentgenographed, but merely chosen from a group of decalcified specimens as a matched pair of comparable levels. This level is at the inferior-most portion of the hypotympanum.

Key to illustrations: 1. hypotympanum; 2. cochlear aqueduct; 3. jugular foramen; 4. mastoid tip; 5. carotid canal; 6. external canal; 7. cochlea; 8. hook of cochlea; 9. tympanic cavity; 10. ossicles; 11. niche of oval window; 12. footplate of stapes; 13. otic capsule anterior to oval window; 14. malleus; 15. incus with short process; 16. epitympanic recess; 17. facial canal; 18. aditus; 19. mastoid antrum; 20. posterior semicircular canal; 21. horizontal semicircular canal; 22. internal auditory canal; 23. facial hiatus; 24. vestibule; 25. anterior limb of the superior semicircular canal; 26. crus commune.

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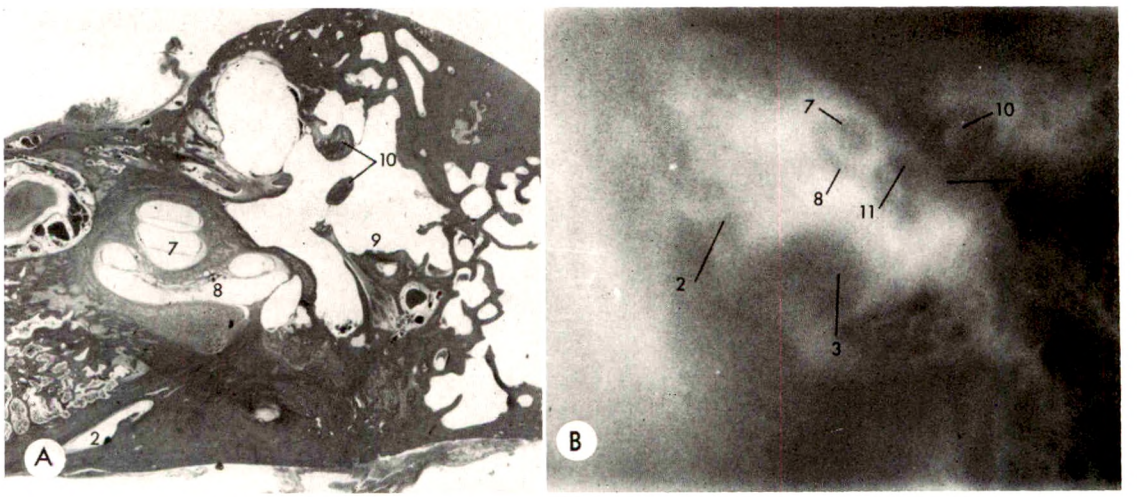


FIG. 3. (*A* and *B*) Four millimeters cephalad to Figure 2 through the level of the cochlea (7). On the histologic section and on the laminogram the terminal portion of the basal turn of the cochlea (8) can be seen entering the vestibule. Due to slight differences in angulation of the sections, the oval window niche (11) is not clearly shown on the histologic section. The slender cochlear aqueduct (2) can be traced into the hook portion of the cochlea on the histologic section. Due to the dense surrounding bone and the small size of this aqueduct, the entire course of this channel cannot be demonstrated roentgenographically.

Several of the patients had internal auditory canals that are oval in cross section such that in one mastoid the long axis of the porus acusticus is in the vertical plane, while on the opposite side the long axis would be in an anteroposterior direction. Thus, the total diameters as measured on the anteroposterior laminograms and basal laminograms would be equal. If one had obtained only the anteroposterior or basal projection, a spurious impression of acoustic neuroma could occur (Fig. 7, *A–D*).

The variation of normal length of the posterior canal wall was about the same in

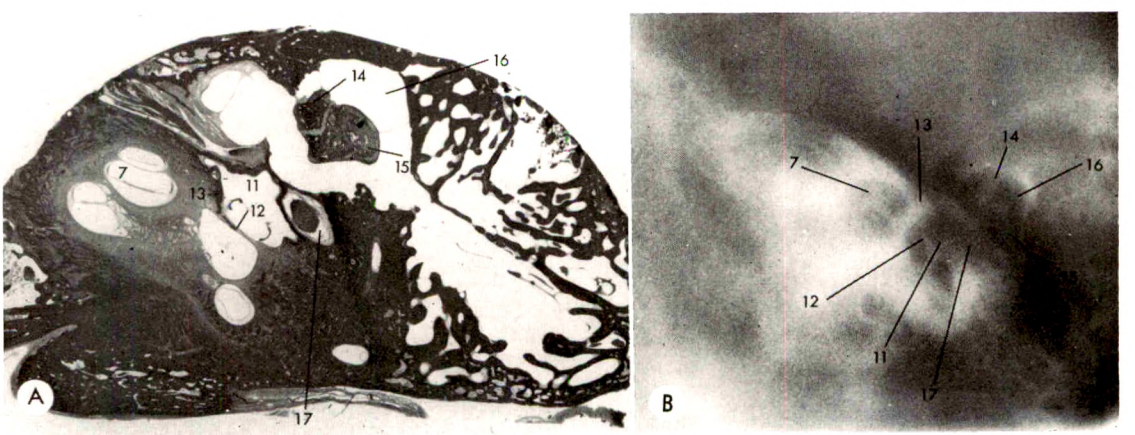


FIG. 4. (*A* and *B*) One millimeter cephalad to Figure 3. The layer passes through the oval window, which is more of a potential foramen since it is completely filled by the footplate of the stapes (12). The thickness of the footplate cannot be consistently visualized roentgenographically. Of considerably more interest, however, is the reliable demonstration of the otic capsule anterior to the oval window (13). This promontory of bone is at the anterior border of the oval window and is the most frequent site of early otosclerotic involvement. The joint space between the malleus (14) and body of the incus (15) is consistently visible, but does not reproduce well for illustration.

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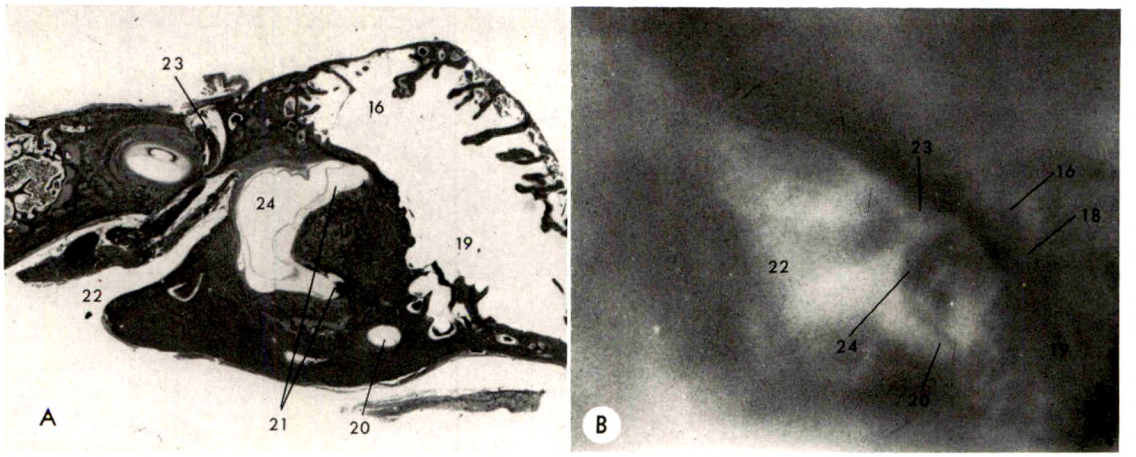


Fig. 5. (*A* and *B*) One millimeter cephalad to Figure 4. The most important feature of this section is the internal auditory canal (22), with its sharp demonstration of the posterior wall. The posterior semicircular canal (20) should lie 3–4 mm. beneath the posterior cortical margin of the petrous bone. Any increase in this thickness indicates that the laminographic section is on an oblique plane rather than a true basal projection. In the histologic section, the ossicles dropped from the section; however, they can be identified in the laminographic cut. The hour-glass configuration of epitympanic recess (16), narrow aditus (18), and mastoid antrum (19) are clearly shown.

the basal laminograms as in the anteroposterior laminograms. Approximately 35 per cent of each group showed variations of 1.0–1.5 mm. between the 2 sides. Similarly, the vertical diameter of the internal auditory canal on the anteroposterior laminograms showed about a 15 per cent variation of greater than 1 mm. between the 2 sides. A similar percentage was encountered when one measured the anteroposterior diameter of the canal on the basal laminograms and compared the 2 sides.

DISCUSSION

Basal laminography should not be considered as a competitive procedure with any other procedure in mastoid radiologic investigation, but merely as another ad-

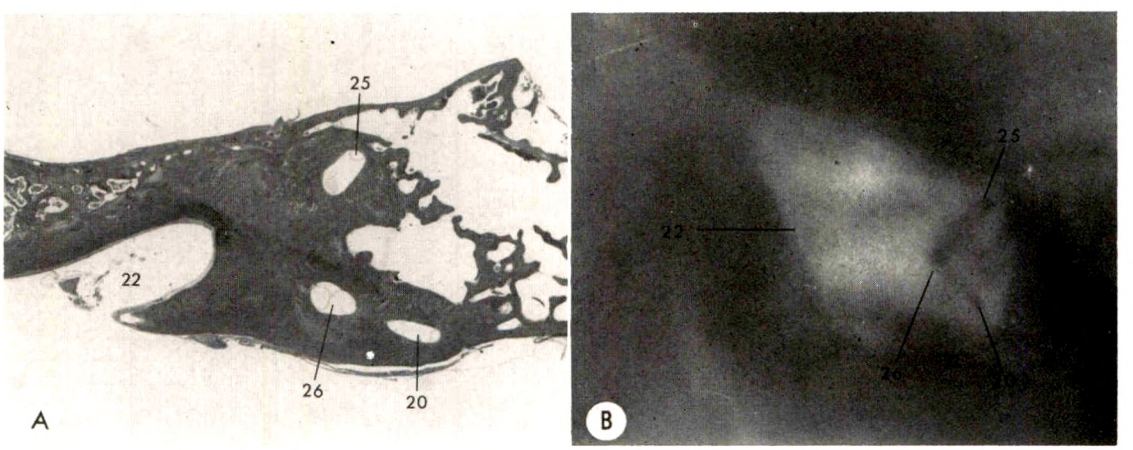
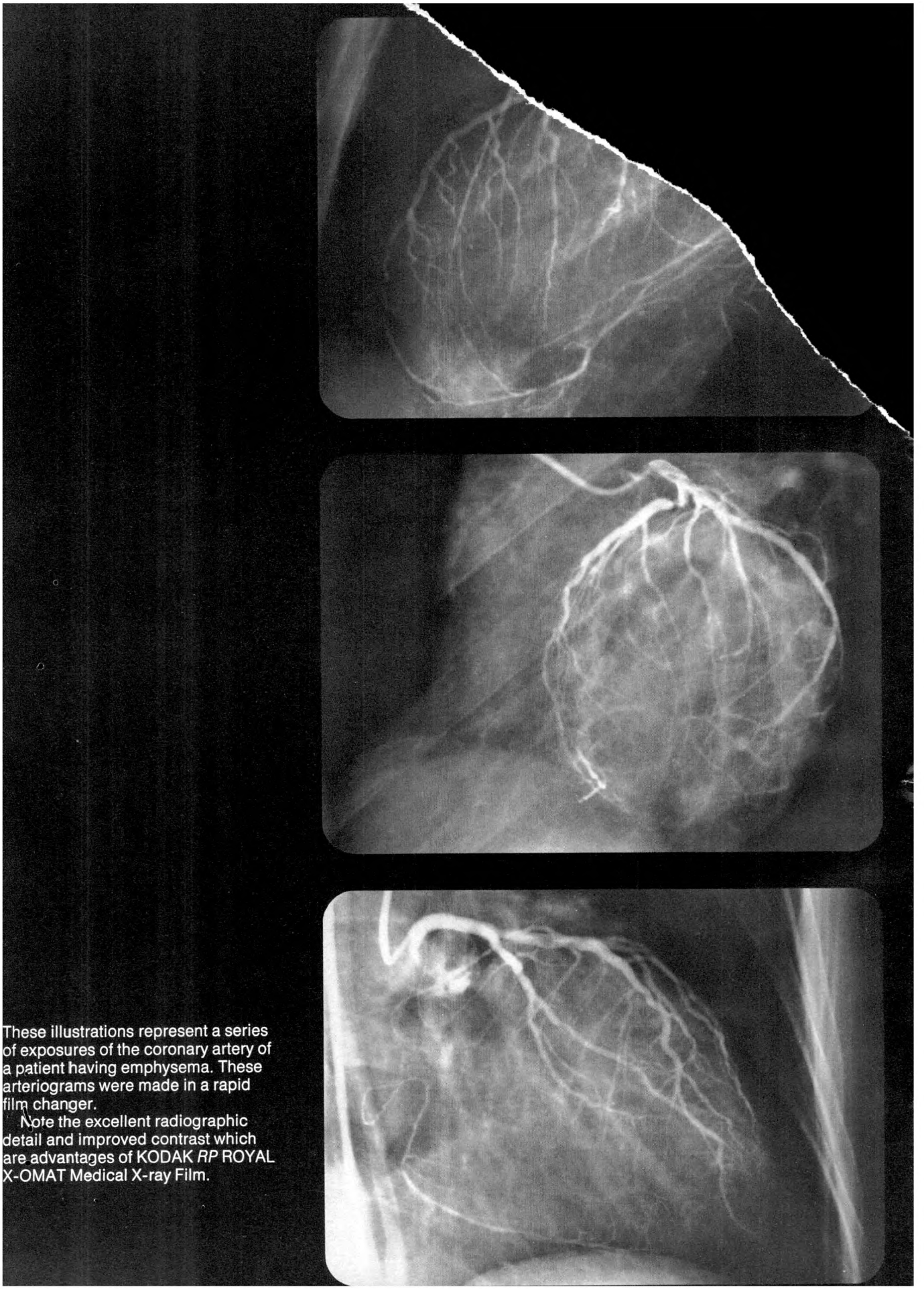


Fig. 6. (*A* and *B*) Three millimeters more cephalad than Figure 5. The histologic section as well as the laminogram show the air cells contained in the angle between the plane of the limbs of the superior semicircular canals and posterior semicircular canals. The internal auditory canal is fading from focus in the laminographic section. The air cells that extend laterally and posteriorly into the sinodural angle can be identified in the lower right hand corner of both *A* and *B*.



These illustrations represent a series of exposures of the coronary artery of a patient having emphysema. These arteriograms were made in a rapid film changer.

Note the excellent radiographic detail and improved contrast which are advantages of KODAK *RP* ROYAL X-OMAT Medical X-ray Film.

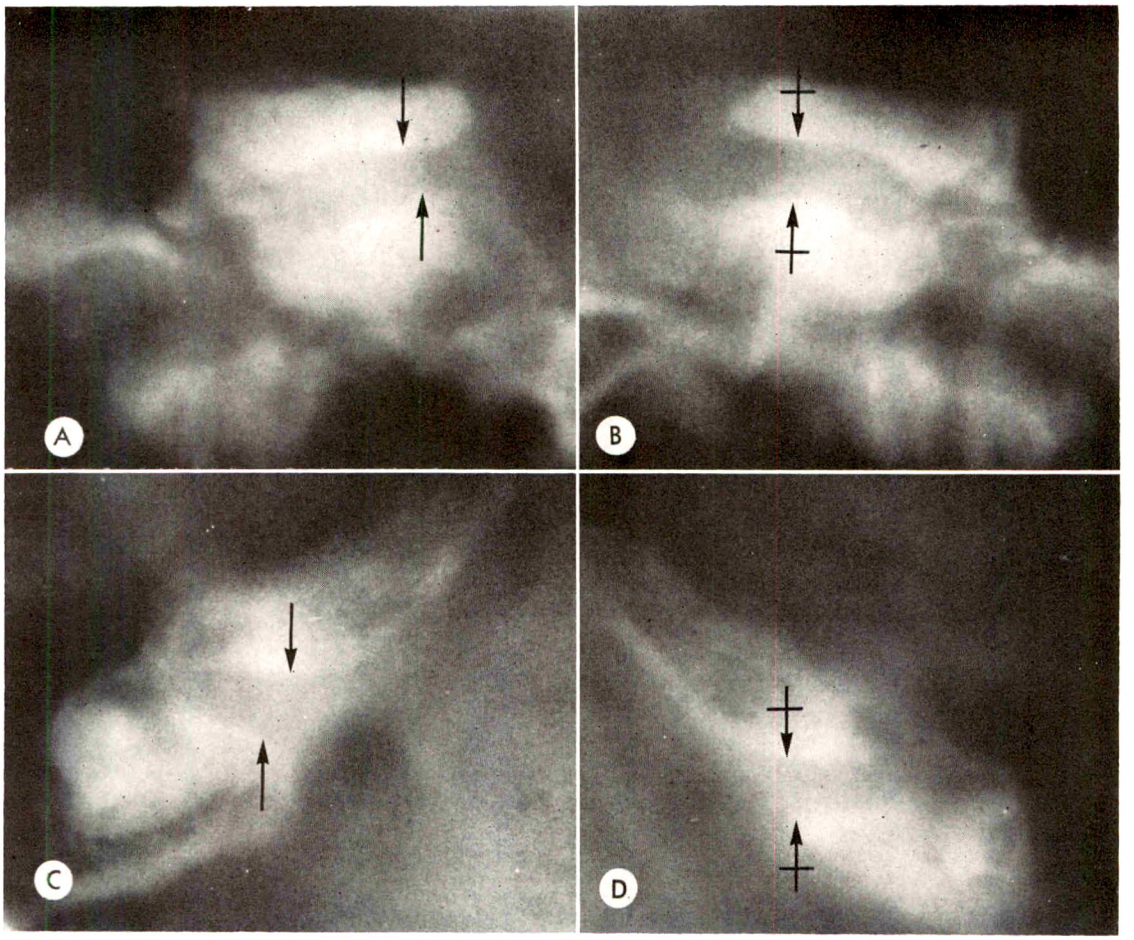
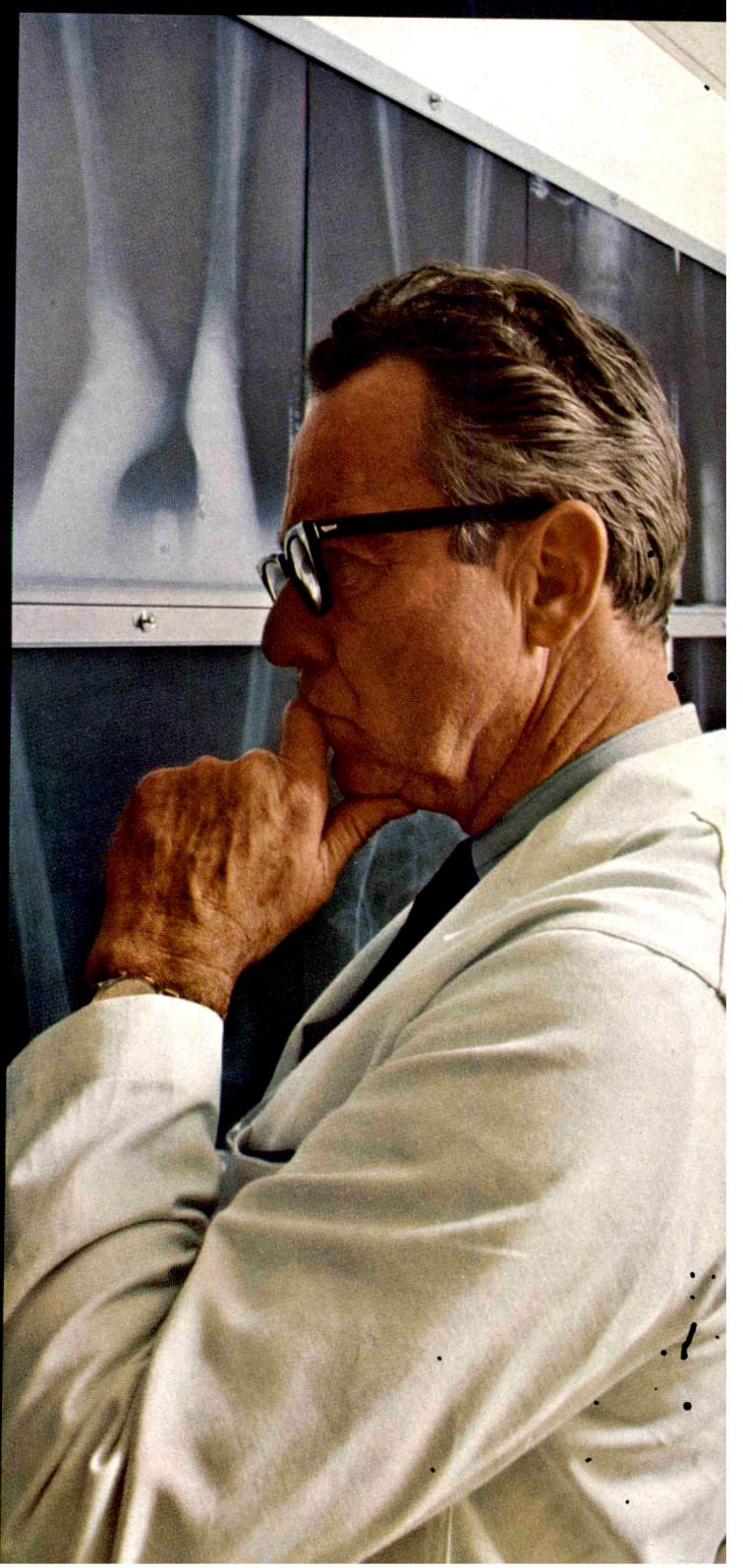


FIG. 7. (*A*) On the right the internal auditory canal gives a spurious impression of erosion in the basal projection (arrows). (*C*) In the anteroposterior projection the canal is slender. (*B*) By contrast, the left internal auditory canal in the basal projection has parallel walls. (*D*) This canal appears widened in the anteroposterior projection.

junct in the diagnosis of certain pathologic conditions. The otologist is striving for the early diagnosis of acoustic neuroma, while the lesion is still within the canal (Fig. 8, *A–D*). Recognizing the minute lesions that are producing the earliest possible bone erosion tests the diagnostic acumen of any radiologist. Basal laminography has been of considerable assistance in identifying the anatomic variations and shows considerable promise in elucidating the problems of the acoustic neuroma (Fig. 9, *A–D*).

Care must be taken to get a sufficient angulation for a true basal projection; otherwise, the laminograph will pass in an oblique plane. Instead of the usual sharp posterior wall of the internal auditory canal, the laminographic section will pass through the posterior inferior wall of the petrous pyramid. The porus acusticus assumes an exaggerated funnel shape with an ill defined posterior margin. Faulty technique can be suspected by comparing the relationships of the posterior semicircular canal to the porus acusticus. Anatomically, the posterior semicircular canal parallels the posterior surface of the petrous pyramid immediately beneath a 2–3 mm. layer of cortical bone (Fig. 5, *A*

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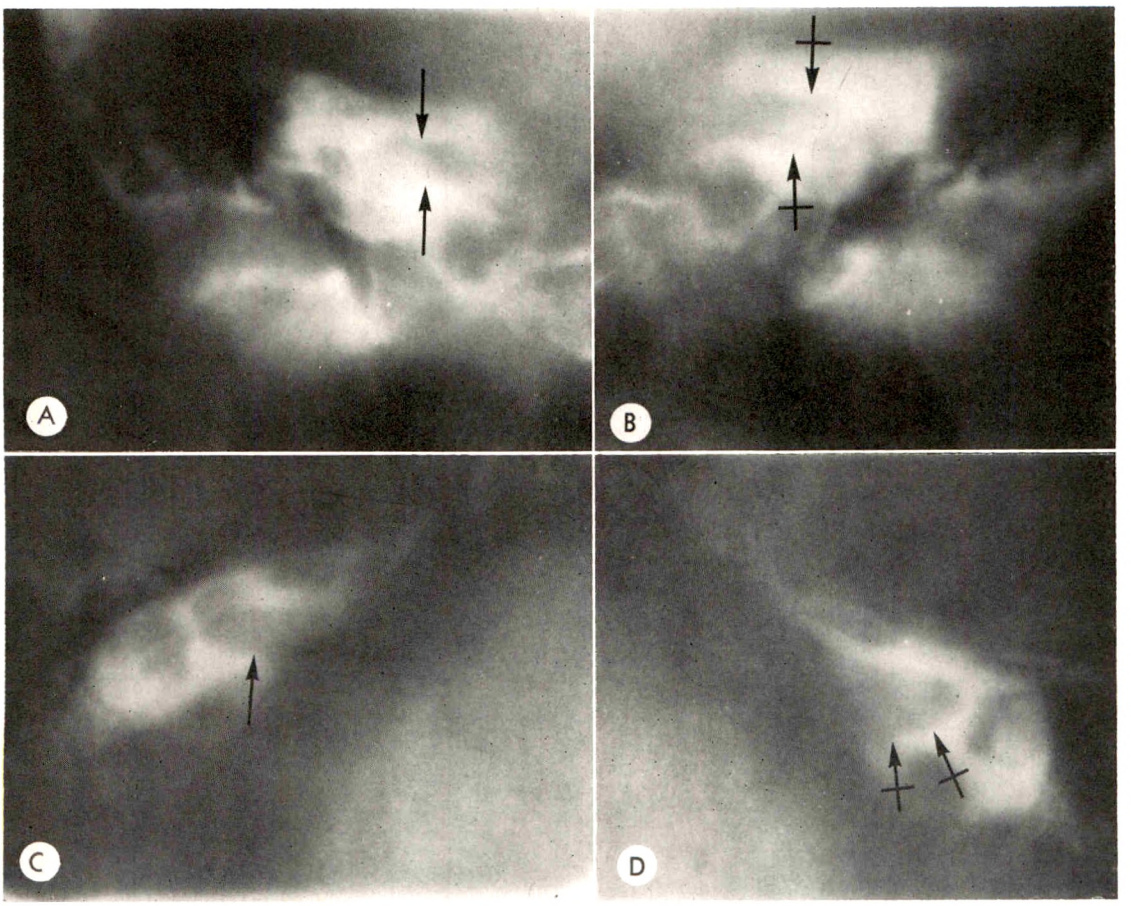


Fig. 8. An acoustic neuroma was confirmed to be present on the right. The lesion was confined to the interna auditory canal. (*A* and *C*) Relatively normal appearance of the internal auditory canal in the anteroposterior laminograms. (*B* and *D*) In the basal projections the erosive changes of the posterior wall (crossed arrows) are a distinct finding as compared to the normal side (single arrow in *C*).

and *B*). The examiner can, by drawing a line parallel to the posterior semicircular canal about 2–3 mm. posterior to the canals, identify the true location of the porus acusticus and medial limit of the posterior wall of the internal auditory canal.

Some future uses of this projection could well be in better delineation of the inflammatory processes in the epitympanic recess-aditus-antral area. The joint space between the head of the malleus and incus can clearly be delineated and may prove the projection of choice for identifying ossicular dislocations. We are also curious about its use with sharper coning in better delineating the optic capsule anterior to the oval window, since this is the earliest and most frequent site of otosclerotic involvement of the oval window.

CONCLUSIONS

1. By the simple mechanism of a second table, basal laminography of the mastoids and base of the skull becomes readily obtainable.

2. In our hands, this projection becomes the method of choice for visualizing early acoustic neuroma.

3. The projection shows the anatomy in a fashion that is familiar to the otologic

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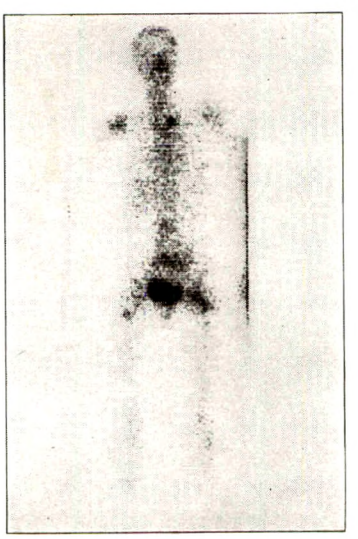
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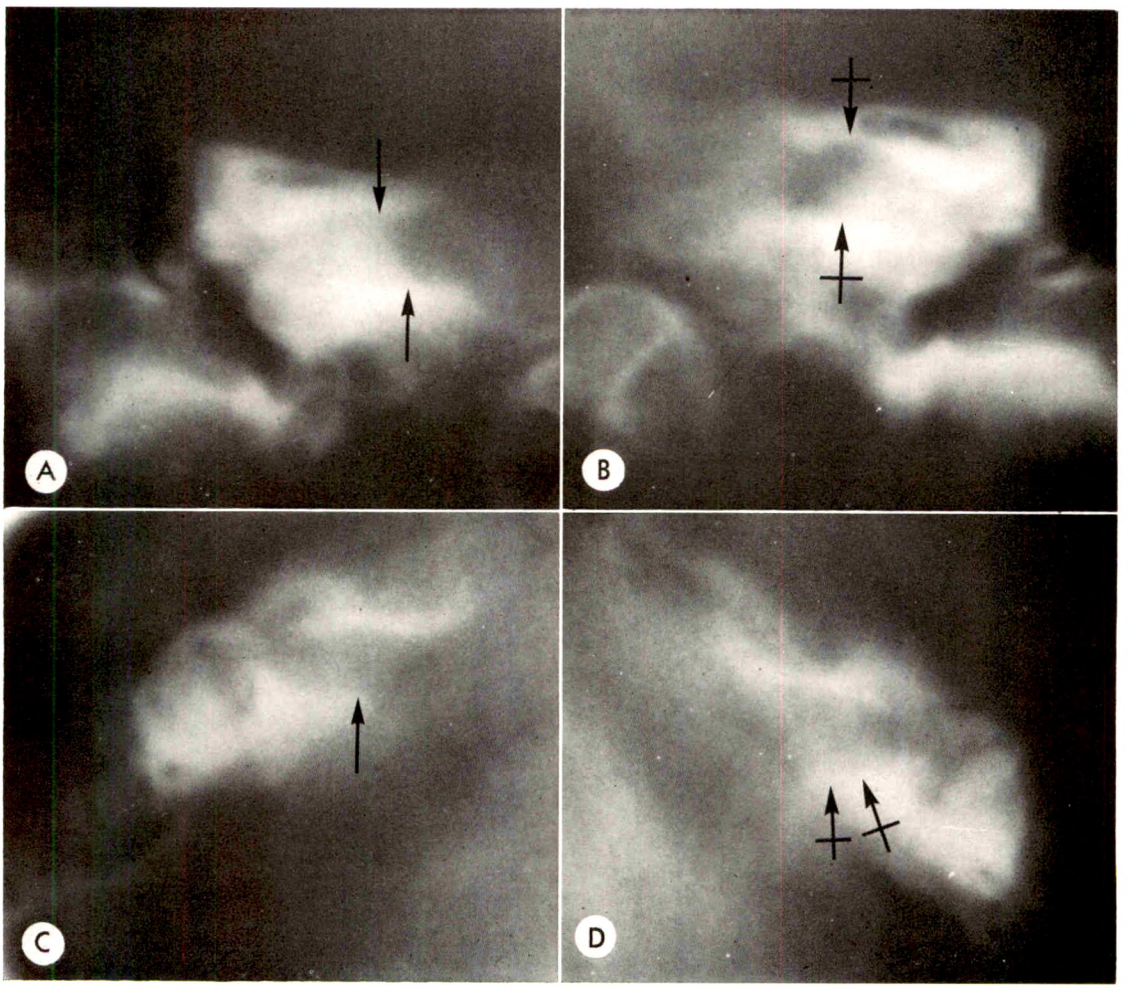


FIG. 9. (*A* and *C*) Normal. (*B*) The acoustic neuroma in this patient bulged from the internal auditory canal into the pontocerebellar angle. The anteroposterior laminogram shows erosion of the superior margin of the porus acusticus (crossed arrows) as compared to the normal side in *A*. (*D*) In the basal projection the entire posterior wall of the internal auditory canal and porus acusticus is markedly eroded (crossed arrows).

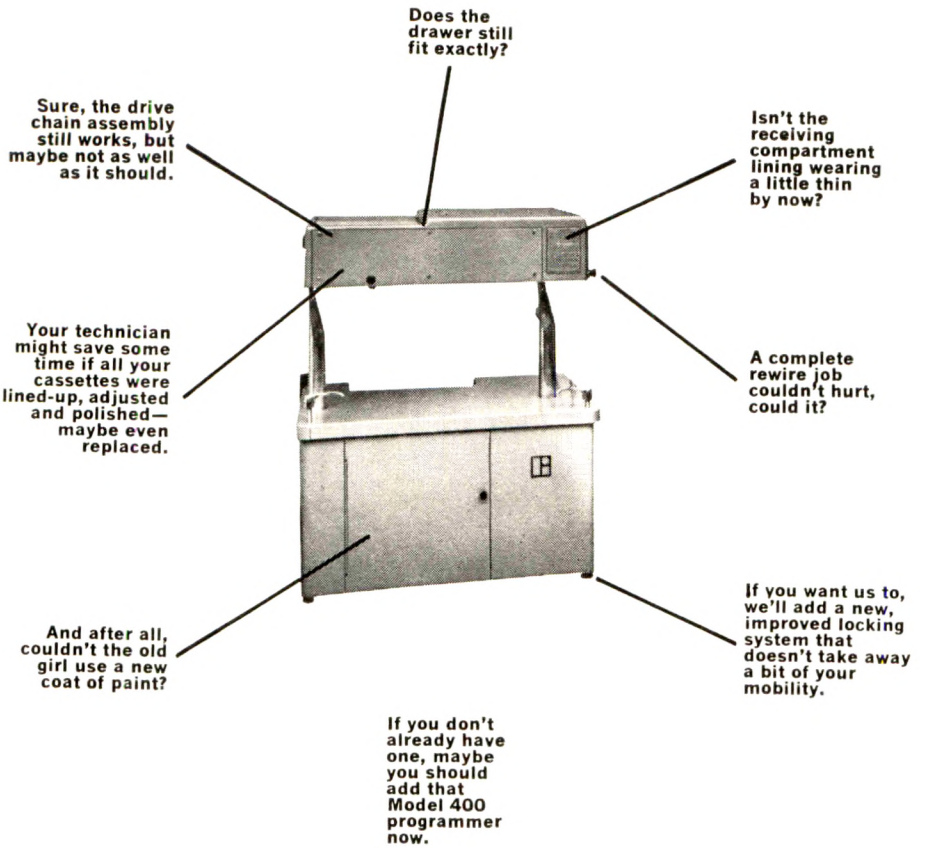
pathologist and allows better delineation of the inner ear structures for pathologic correlation.

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OCULAR HYPERTELORISM (GREIG'S SYNDROME) ASSOCIATED WITH SPRENGEL'S DEFORMITY*

By THEODORE E. KEATS, M.D.
CHARLOTTESVILLE, VIRGINIA

GREIG'S syndrome consists of ocular hypertelorism with a variety of associated anomalies. Among those recognized is the unusual association with Sprengel's deformity of the shoulders.

This paper presents 2 additional cases of this manifestation of the syndrome.

REPORT OF CASES

CASE 1. W.M., a 12 year old white boy, has been confined to a state training school since the age of 4 because of mental retardation. His facies was unusual in that he had striking ocular hypertelorism and depression of the root of the nose and maxilla. His head was abnormally small and a bone defect was palpable in the midline of his frontal bone. His palate was high and arched. Both shoulders were held in an elevated position.

Roentgen examination of the skull showed a small cranial vault with flattening of the occiput and bulging of the right temporal area. Osseous defects were present in the midline of the frontal bone (cranium bifidum occultum) and in the occipitomastoid sutures. The maxilla was hypoplastic with marked orbital hypertelorism, measuring 0.5 cm. above the maximum normal limit for his age and sex according to the standards of Hansman.[4]

The frontal sinuses were not developed. The hard palate was arched, the maxilla and root of the nose depressed, and the mandible relatively prognathic. The first cervical vertebra has assimilated to the base of the skull and its spinous process was absent (Fig. 1, *A* and *B*)

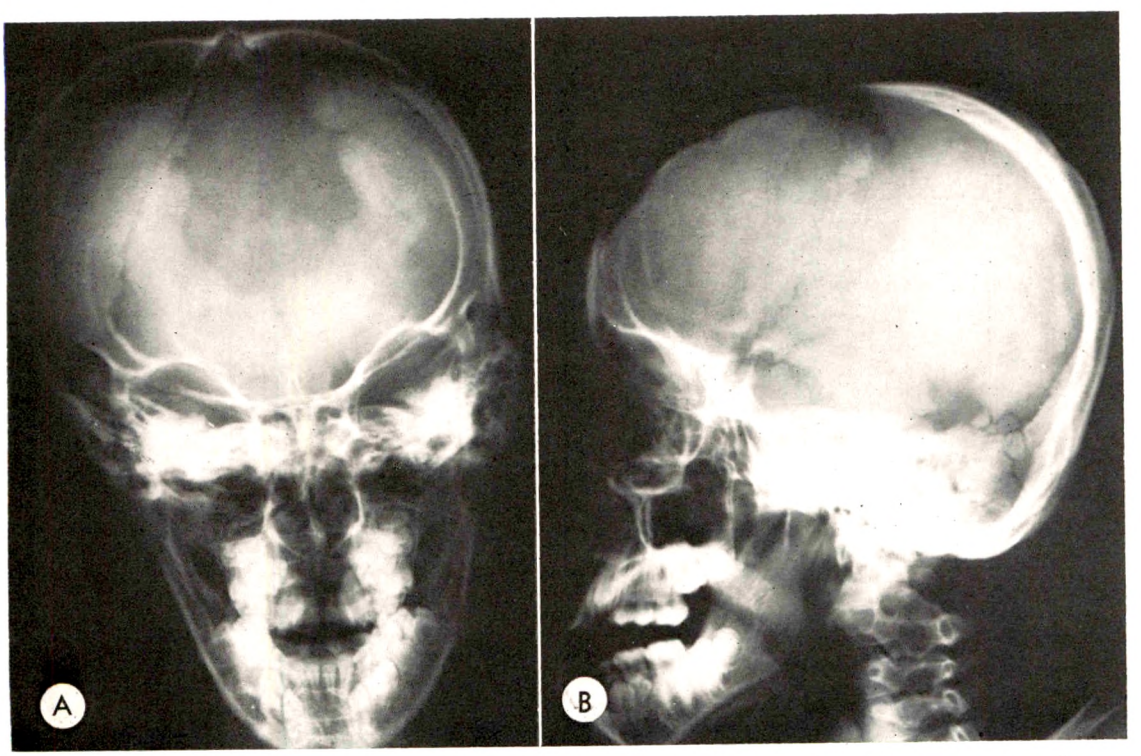


FIG. 1. Case 1. (*A* and *B*) Roentgenograms of the skull show microcephaly, cranium bifidum occultum, hypertelorism, depression of the maxilla, and relative mandibular prognathism.

* From the Department of Radiology, University of Virginia School of Medicine, Charlottesville, Virginia.



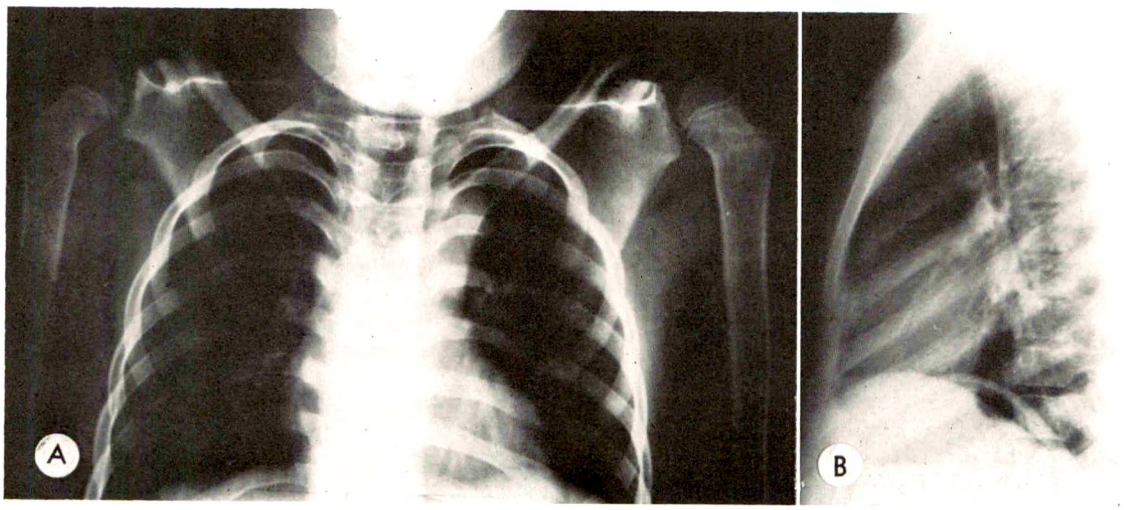


Fig. 2. Case I. (*A* and *B*) Chest roentgenograms show Sprengel's deformity of the shoulders.

Chest roentgenograms showed high position of the shoulders (Sprengel's deformity), non-segmentation of the sternum, narrow antero-posterior diameter of the chest and notch-like depressions on the medial aspects of the humeral necks, particularly on the right (Fig. 2, *A* and *B*).

A roentgen survey of the remainder of the skeleton showed short ulnae with obliquity of the distal radial epiphysis, incomplete closure

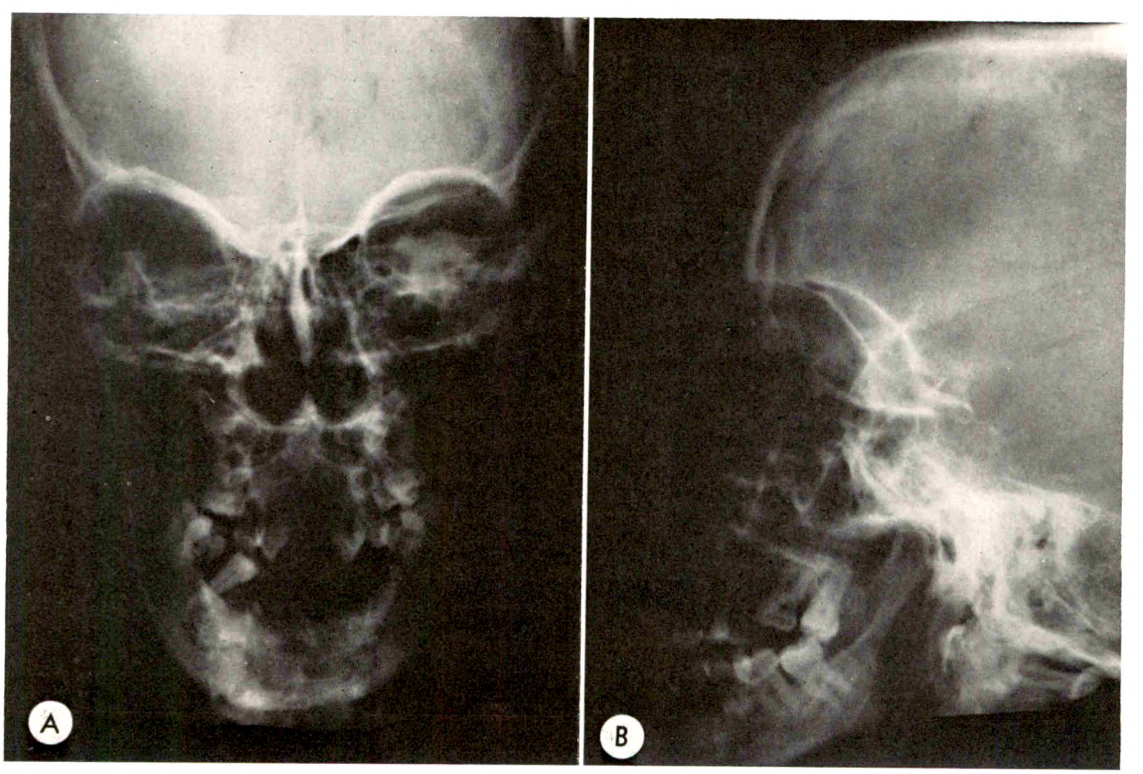


Fig. 3. Case II. (*A* and *B*) Roentgenograms of the facial bones show hypertelorism, depression of the maxilla, and relative mandibular prognathism.

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of the neural arches of the lumbar vertebrae, and marked juvenile epiphysitis of the lumbar spine. Coxa valga deformity of the hips was noted.

Case II. E.J., a 48 year old Negro woman, was admitted for repair of an umbilical hernia. Her mentality was apparently normal. The most striking finding of interest here was marked depression of the mid face with hypertelorism. The palate was high and arched and her shoulders held in an elevated position.

Roentgen examination showed hypoplasia and depression of the maxilla and hypertelorism, measuring 0.7 cm. above the maximum normal for her age and sex according to the standards of Hansman.[4] The root of the nose was depressed and the frontal sinuses undeveloped. The mandible was prognathic (Fig. 3, *A* and *B*).

Chest roentgenograms showed high position of the shoulders with short clavicles (Sprengel's deformity) (Fig. 4, *A* and *B*).

DISCUSSION

The term hypertelorism has become confined to describe excessive distance between the eyes and orbits. Greig's original description of this entity was made in 1924[3] and successive reports have implicated a variety of other inconstant anomalies. Among these are: mental retardation, syndactily, muscular hypotonicity, renal hypoplasia, hypospadias, webbing of the neck, congenital heart anomaly, auditory disturbances, high arched palate, cleft lip and palate, hypoplasia of the maxilla, macroglossia, microdontia, and amelogenesis imperfecta.[2] The association with Sprengel's deformity has been reported previously in only 4 other patients.[5–8]

Hypertelorism may occur in a variety of craniostenoses and craniofacial dysostoses. However, exophthalmos is common in these conditions, but is never encountered in Greig's hypertelorism. The frontal cranial defect seen in our Case 1 was also present in James' patient who also had Sprengel's deformity.[5] The failure of segmentation of the sternum present in our Case 1 has apparently not been described previously.

The cause of the syndrome is not known. Greig suggested that the hypertelorism was due to underdevelopment of the greater wings of the sphenoid and relative underdevelopment of the lesser wings. It is interesting to note that there is an association of mental retardation with both hypertelorism[3] and with Sprengel's deformity.[1]

The facies of patients with this syndrome

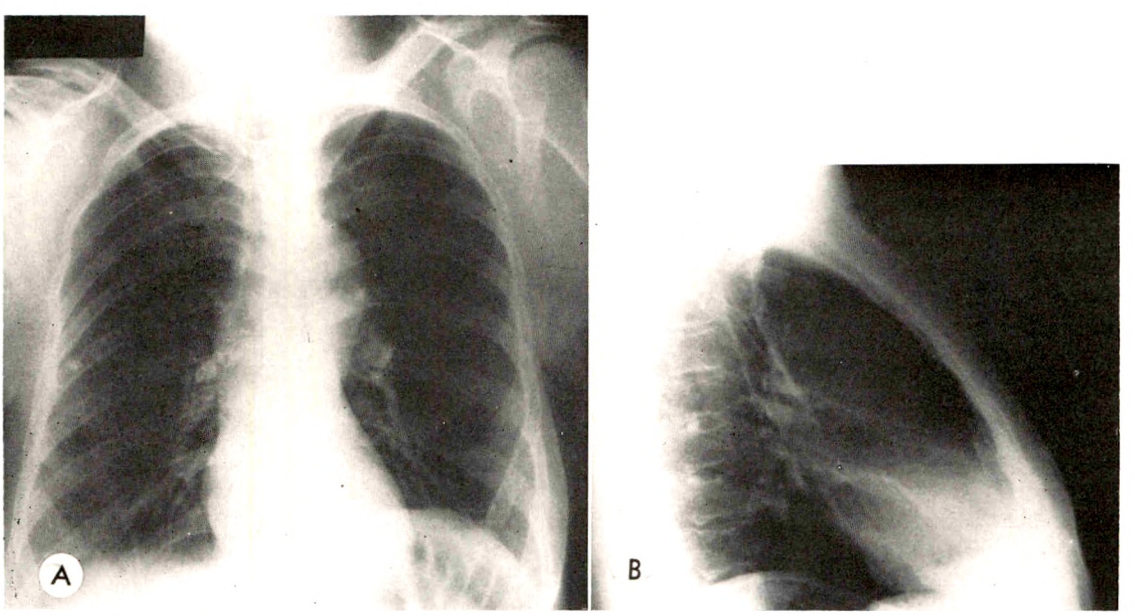
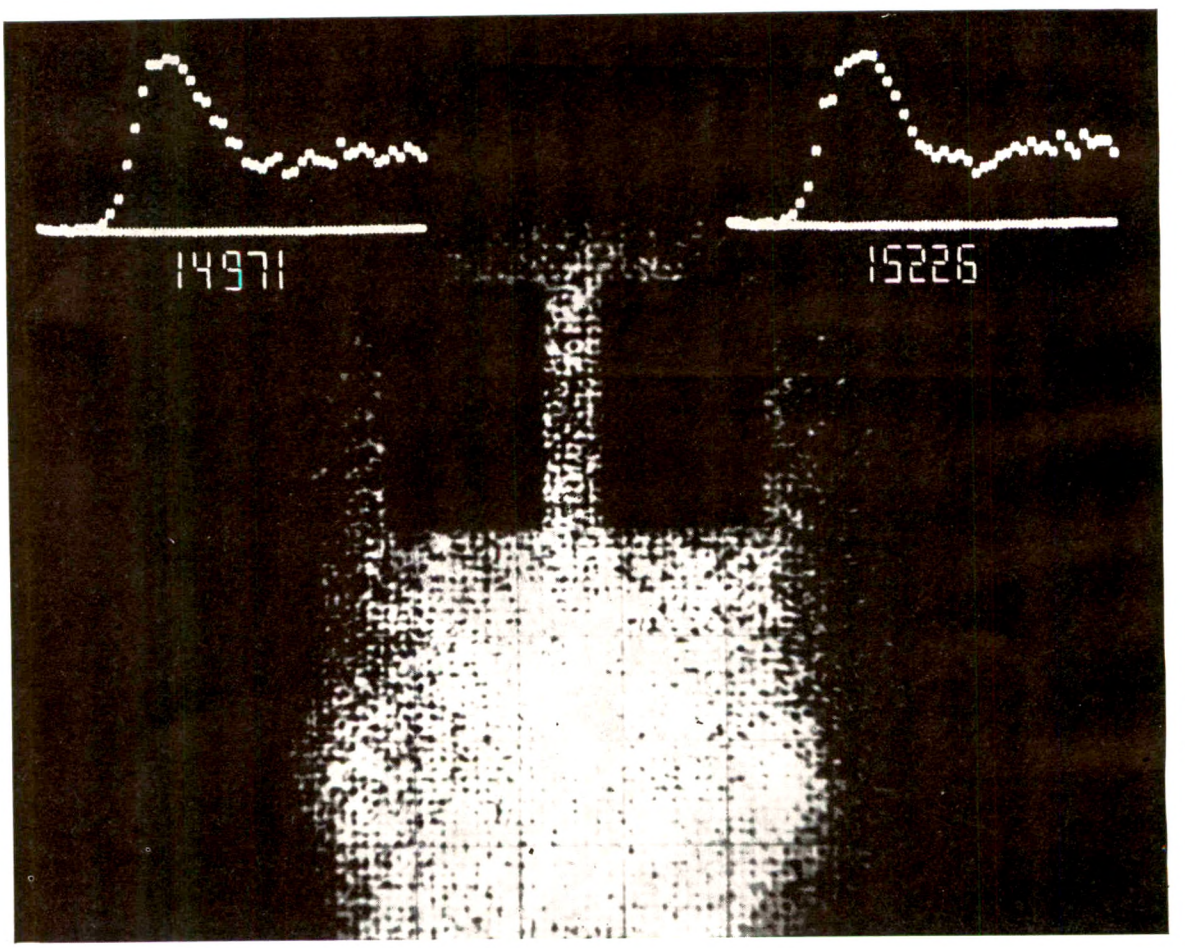


Fig. 4. Case II. (*A* and *B*) Chest roentgenograms show Sprengel's deformity of the shoulders.

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is common to all, consisting of separation of the eyes, depression of the root of the nose and the maxilla. Roentgenologic confirmation of hypertelorism is simple and the standards for determination reported by Hansman[4] are useful in this regard. Once hypertelorism is detected, the radiologist should lead a search for the variety of associated anomalies, some of which are consistently present.

SUMMARY

Ocular hypertelorism (Greig's syndrome) occurs in association with a wide variety of associated anomalies, many of which are detectable roentgenologically.

This report describes 2 patients with this syndrome in association with Sprengel's deformity.

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ORBITAL SCLEROSIS IN HISTIOCYTOSIS X*

By MARK E. NESBIT, JR., M.D.,† JUSTIN J. WOLFSON, M.D.,‡
STEPHEN A. KIEFFER, M.D.,‡ *and* HAROLD O. PETERSON, M.D.‡
MINNEAPOLIS, MINNESOTA

ORBITAL involvement in histiocytosis X is most often manifested clinically as exophthalmos. Masses originating in the bones of the orbit displace the globe in the direction of least resistance. Roentgenograms typically reveal single or multiple irregularly marginated lytic lesions usually involving the roof or lateral wall of the bony orbit.

Recently, we have observed 3 children with biopsy-proven histiocytosis X who presented with orbital sclerosis. The roentgenographic appearance was most unusual and was felt to be more compatible with that of fibrous dysplasia or other processes which more typically produce sclerosis in bone.

A review of 59 additional children with biopsy-proven histiocytosis X revealed that 11 had orbital lesions at some time during the course of their illness. Two of these 11 cases demonstrated orbital sclerosis, while the remaining 9 were examples of the classical osteolytic lesion.

CLINICAL FINDINGS

In the group of children who had lytic orbital lesions with histiocytosis X, there were 4 boys and 5 girls. Their clinical findings are summarized in Table 1A. The age at onset of the symptoms due to the underlying disease ranged from 3 months to 48 months. Three of the 9 children had involvement of the skeletal system alone, while the remaining 6 had skeletal and visceral disease. Orbital manifestations of the disease occurred at times ranging from the onset of the disease to 29 months later. However, with the exception of 2 cases, orbital involvement occurred within 12 months of the original diagnosis. Four of the 9 children received chemotherapy, and 4 received courses of local irradiation.

The clinical data on the 5 patients with orbital roof sclerosis and thickening are summarized in Table 1B. There were 3 boys and 2 girls. All but 1 had generalized disease. When one compares the groups with lytic and sclerotic lesions as to age of onset, organ involvement, onset of orbital symptoms, and length and type of therapy, there apparently is no difference between the 2 groups. The only significant difference between the 2 groups is that in those cases with sclerosis, all lesions except 1 were bilateral, while in the group with purely lytic orbital lesions 7 of the 9 cases were unilateral.

ROENTGENOGRAPHIC FINDINGS

The osteolytic lesion of histiocytosis X appears roentgenographically as an irregularly marginated radiolucent defect (Fig. 1, *A* and *B*). There is little or no surrounding sclerosis. The lesions vary in size and are often asymmetric in shape and distribution. Eight of the 9 cases with purely lytic lesions demonstrated involvement of the orbital roof. The lateral wall of the orbit and lesser and greater wings of the sphenoid were less commonly involved (Table 11). Healing associated with local radiotherapy or by spontaneous regression was usually complete; however, minimal or moderate residual flattening of the orbital roof was noted in 4 patients (Fig. 1*C*). In addition, 2 patients who had extensive involvement of the bony orbit eventually demonstrated decreased size of the orbital cavity. Both of these patients were treated entirely with chemotherapy and received no irradiation to the involved orbits.

* Presented in part at the Sixth Annual Meeting of the American Society of Neuroradiology, New Orleans, Louisiana, September 27–28, 1968.
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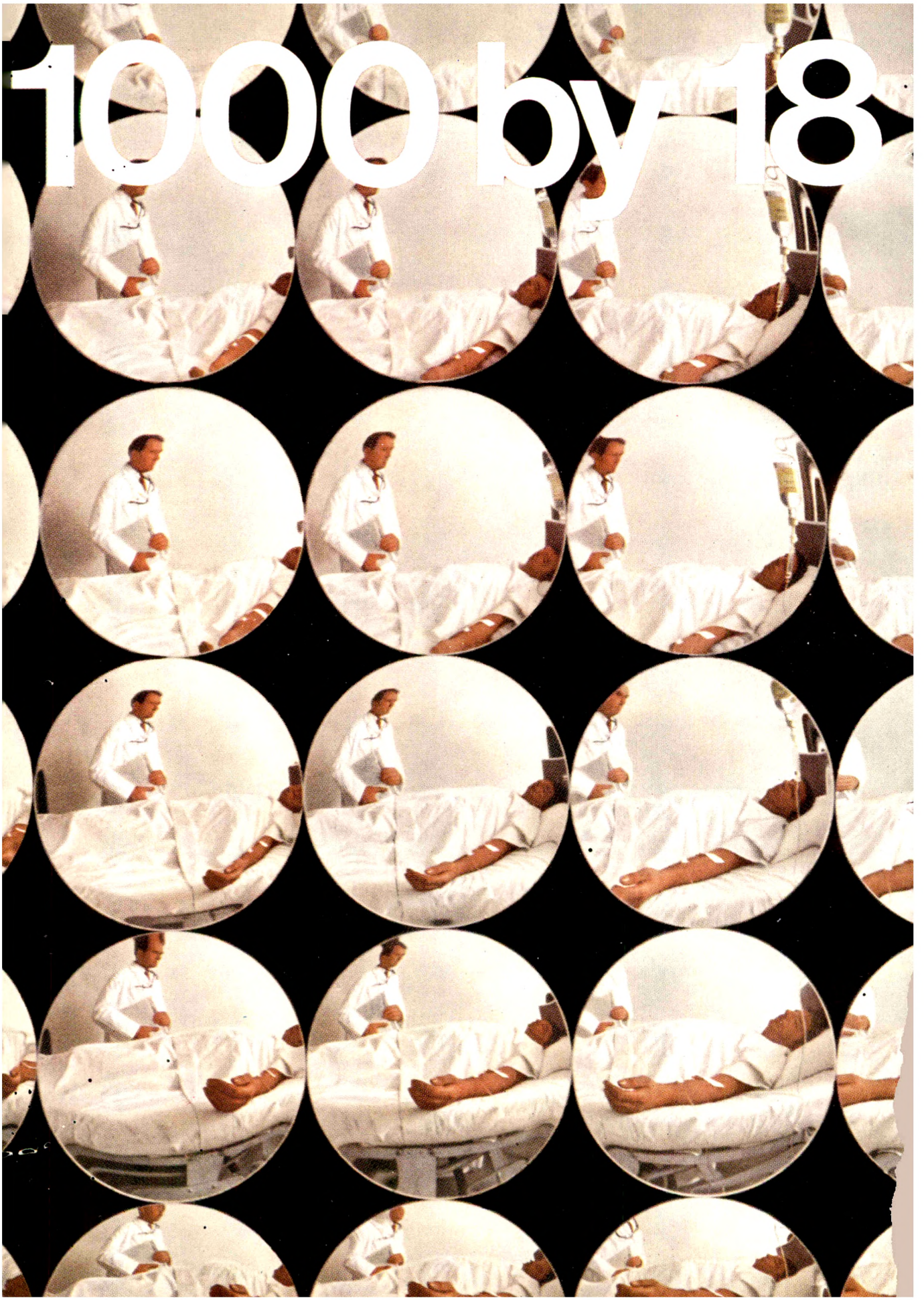

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TABLE I

ORBITAL INVOLVEMENT IN HISTIOCYTOSIS X—CLINICAL SUMMARY

Patient	Sex	Age at Onset	Organ Involvement	Interval to Onset of Orbital Involvement	Systemic Therapy	Local Radiotherapy to Orbit
A. *Without Sclerosis*						
1. B.L.	M	48 mo.	Skeleton	Onset	None	Co^{60} (450 rads/3 days)
2. V.J.	F	9 mo.	Skeleton, lung, skin	25 mo.	Tetracycline	None
3. V.B.	M	9 mo.	Skeleton, lung, skin	11 mo.	Prednisone, velban, methotrexate	3 courses over 7 months: Co^{60} (800 rads/6 days), Co^{60} (800 rads/7 days), 250 kvp. (800 rads/9 days)
4. D.M.	F	36 mo.	Skeleton	Onset	None	Co^{60} (450 rads/3 days)
5. K.C.	F	6 mo.	Skeleton, liver, central nervous system, spleen, skin, lymph nodes	8 mo.	Methotrexate	None
6. D.L.	M	5 mo.	Skeleton, liver, skin, lung	7 mo.	Prednisone, methotrexate	250 kvp. (1,800 rads/30 days)
7. B.B.	F	42 mo.	Skeleton	3 mo.	Prednisone, velban	None
8. C.R.	F	6 mo.	Skeleton, lung, skin, central nervous system	5 mo.	None	250 kvp. (500 rads/7 days)
9. A.L.	F	3 mo.	Skeleton, lung, skin, spleen, lymph nodes, central nervous system	29 mo.	ACTH, 6 mercaptopurine	None
B. *With Sclerosis*						
10. D.H.	M	24 mo.	Skeleton, liver, skin, spleen, lungs	Onset	Prednisone velban, methotrexate	250 kvp (1,000 rads/14 days) Age 3 yr.: Co^{60} (1,000 rads/12 days)
11. C.D.	F	4 mo.	Skeleton, lung	20 mo.	None	2 courses over 2 months: 250 kvp. (600 rads/5 days), (300 rads/2 days)
12. N.H.	F	16 mo.	Skeleton, lung, skin, lymph nodes	12 mo.	Prednisone, methotrexate	None
13. M.S.	M	18 mo.	Skeleton	Onset	None	None
14. T.T.	M	30 mo.	Skeleton, lymph nodes, skin	Onset	Prednisone, velban, methotrexate	250 kvp. (1,800 rads/14 days)

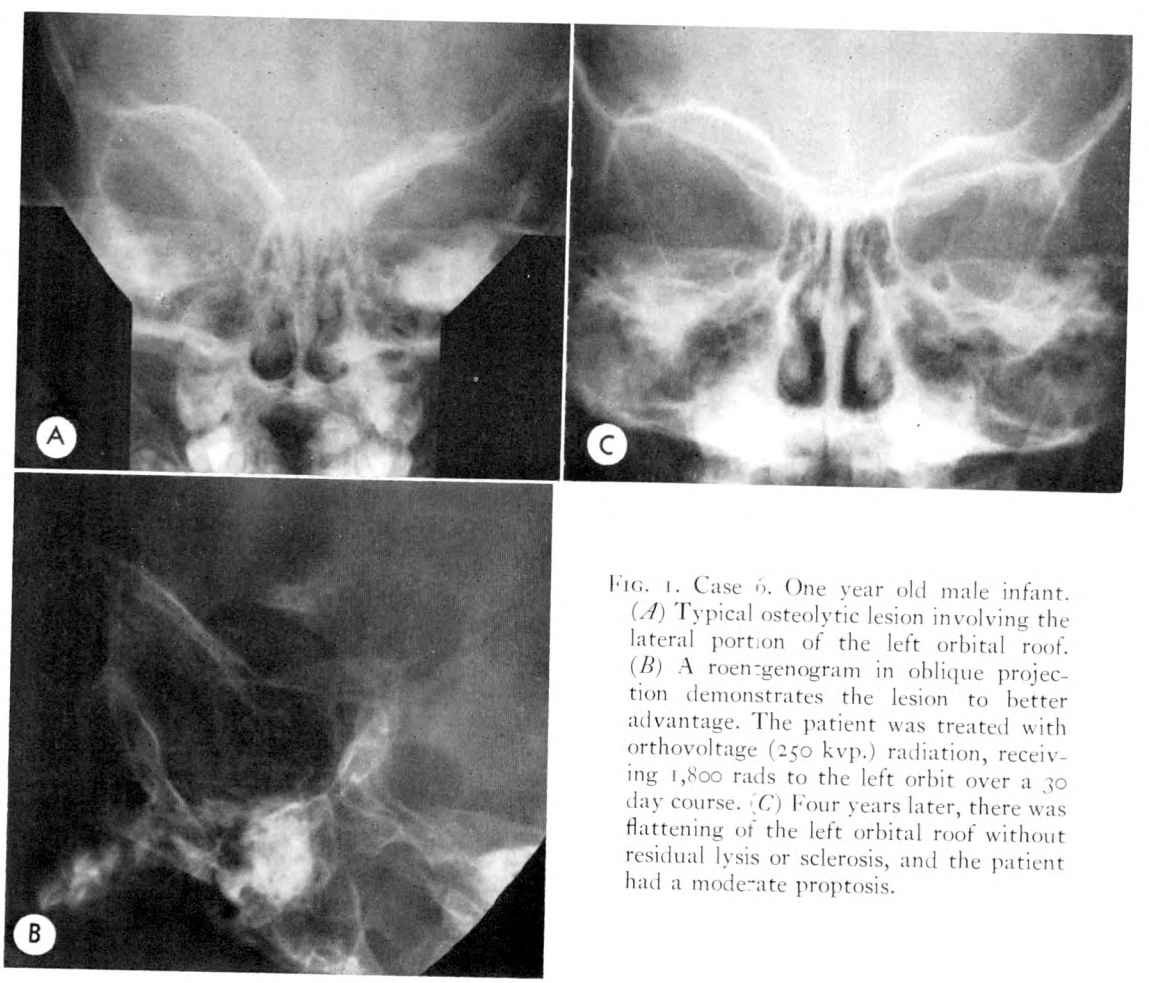
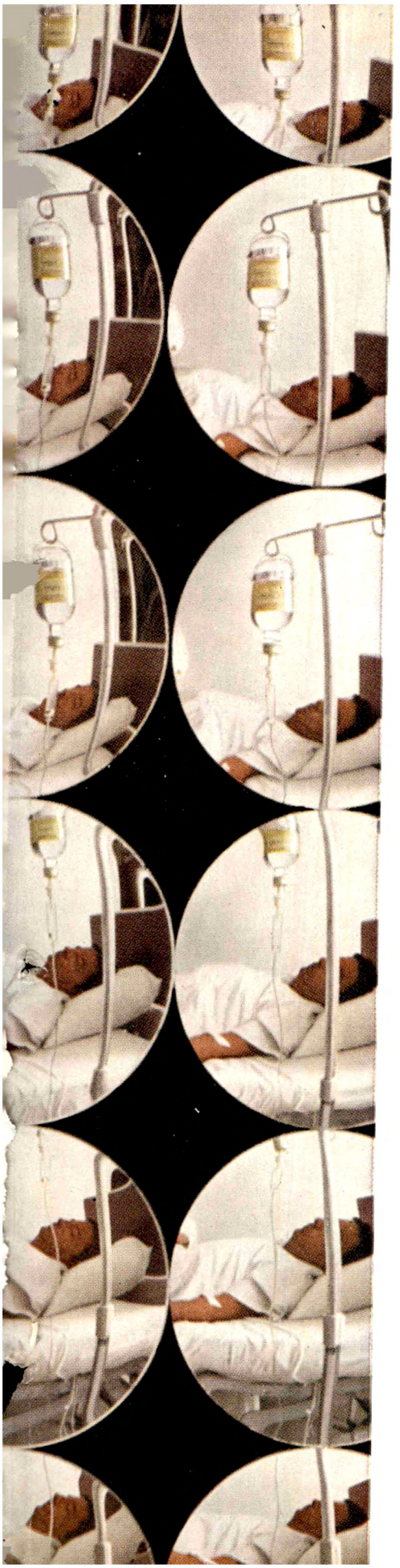


FIG. 1. Case 6. One year old male infant. (*A*) Typical osteolytic lesion involving the lateral portion of the left orbital roof. (*B*) A roentgenogram in oblique projection demonstrates the lesion to better advantage. The patient was treated with orthovoltage (250 kvp.) radiation, receiving 1,800 rads to the left orbit over a 30 day course. (*C*) Four years later, there was flattening of the left orbital roof without residual lysis or sclerosis, and the patient had a moderate proptosis.



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1. Data on file at The Squibb Institute for Medical Research.

See next page for brief summary.

TABLE II

ORBITAL LESIONS IN HISTIOCYTOSIS X: ROENTGENOGRAPHIC–CLINICAL CORRELATION

Patient	Clinical Features	Extent of Orbital Involvement				Residual Roentgenographic Findings	Residua Proptosis	Length of Follow-up
		Orbital Roof	Lateral Wall of Orbit	Lesser Wing of Sphenoid	Greater Wing of Sphenoid			
A. *Without Sclerosis*								
1. B.L.	Mass lateral to rt. eye	—	Lytic	—	—	None	—	1 yr.
2. V.J.	Proptosis, mass, lt. eye	Lytic	Lytic	Lytic	Lytic	Decreased size of orbital cavity	Moderate	8 yr.
3. V.B.	Proptosis, lt. eye	Lytic	—	—	—	Minimal flattening of orbital roof	—	1 yr.
4. D.M.	Proptosis, mass, lt. eye	Lytic	Lytic	—	—	Minimal flattening of orbital roof	—	2 yr.
5. K.C.	None, lt. eye	Lytic	—	Lytic	Lytic	No follow-up, patient died one month later	—	—
6. D.L.	Proptosis, lt. eye	Lytic	—	—	—	Moderate flattening of orbital roof	Moderate	4 yr.
7. B.B.	Proptosis, rt. eye	Lytic	Lytic	Lytic	Lytic	Moderate flattening of orbital roof	—	6 yr.
8. C.R.	Proptosis, mass, rt. eye	Lytic	Lytic	—	Lytic	No follow-up, patient died	—	—
	Proptosis, mass, lt. eye	Lytic	Lytic	—	Lytic		—	—
9. A.L.	Proptosis, rt. eye	Lytic	—	—	—	Decreased size of orbital cavity	—	6 yr.
	Proptosis, lt. eye	Lytic	Lytic	—	—	Decreased size of orbital cavity	—	6 yr.
B. *With Sclerosis*								
10. D.H.	Proptosis, rt. eye	Sclerotic	Sclerotic	—	—	Sclerosis and minimal flattening of orbital roof	Marked	2 yr.
	Proptosis, lt. eye	Lytic and sclerotic	Sclerotic	—	—	Sclerosis and minimal flattening of orbital roof	Marked	2 yr.
11. C.D.	None, rt. eye,	Lytic	—	Lytic	—	Sclerosis	—	8 mo.
	Proptosis, mass, lt. eye	Lytic	Lytic	Lytic	Lytic	Sclerosis and minimal flattening of orbital roof	Moderate	8 mo.
12. N.H.	None, rt. eye	Sclerotic	—	—	—	Sclerosis	—	3 yr.
	Proptosis, lt. eye	Lytic and sclerotic	Sclerotic	—	—	Sclerosis and minimal flattening of orbital roof	Moderate	3 yr.
13. M.S.	Proptosis, rt. eye	Sclerotic	Sclerotic	—	—	Sclerosis and minimal flattening of orbital roof	Minimal	7 yr.
	Proptosis, lt. eye	Sclerotic	Sclerotic	—	—	Sclerosis and minimal flattening of orbital roof	Minimal	7 yr.
14. T.T.	Proptosis, mass, rt. eye	Lytic and sclerotic	Lytic	—	—	Sclerosis and minimal flattening of orbital roof	—	2 yr.

Sclerosis of the orbital roof was noted in 5 of the 14 cases in this series. The findings in this group typically consisted of poorly marginated thickening and sclerosis of one or more often both orbital roofs. This process typically involved the orbital plate of the frontal bone. Extension into the zygomatic process of the frontal bone and across the frontozygomatic suture was frequent. Involvement of the medial wall of the orbit superiorly and anteriorly was noted in 3 instances. However, the posterior margin of the sclerosis and thickening was at the frontosphenoidal suture (between the orbital plate of the frontal bone and the lesser wing of the sphenoid bone) in every case.

Three patients when first seen had combined destructive and sclerotic involvement of the orbital roofs which eventually progressed to pure sclerosis (Fig. 2, *A* and *B*). Three patients exhibited sclerosis without destruction at the time of initial roentgenographic demonstration of orbital involvement in this hospital; however, 1 of these patients had received prior chemotherapy for systemic disease while another had received previous courses of radiotherapy to the skull and orbit in other institutions (Fig. 3, *A–C*). One patient who initially presented with bilateral lytic lesions of the orbital roofs showed progressive thickening and sclerosis with eventual stabilization into a sclerotic phase (Fig. 4, *A* and *B*). In this series there was never any regression of the sclerosis once it developed, and all of these patients demonstrated flattening of the orbital roofs with

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Contraindications: Contraindicated in persons hypersensitive to salts of diatrizoic acid. Urography is contraindicated in patients with anuria.

Warnings: A definite risk exists with the use of contrast agents in excretion urography in patients with multiple myeloma. There has been anuria with progressive uremia, renal failure and death. This risk of the procedure in these patients is not a contraindication; however, partial dehydration in preparation for study is not recommended since it may predispose for precipitation of myeloma protein in renal tubules. No therapy, including dialysis, has been successful in reversing this effect. Myeloma should be considered in persons over 40 before undertaking urographic procedures.

In cases of known or suspected pheochromocytoma, if the physician feels that the possible benefits outweigh the considered risks, radiopaque materials should be administered with extreme caution; however, an absolute minimum of material should be injected, the blood pressure should be assessed throughout the procedure, and measures for treating a hypertensive crisis should be available.

Contrast media may promote sickling in homozygous individuals when injected I.V. or intra-arterially. Although a history of sensitivity to iodine *per se* or to other contrast media is not an absolute contraindication, administration of meglumine diatrizoate requires extreme caution in such cases. Meglumine diatrizoate should be used in pregnant patients only when the physician deems its use essential to the welfare of the patient since safe use during pregnancy has not been established. Perform thyroid function tests prior to administration of meglumine diatrizoate since iodine-containing contrast agents may alter the test results. Perform urography with extreme caution in persons with severe concomitant hepatic and renal disease.

Precautions: Diagnostic procedures involving use of contrast agents should be performed under the direction of personnel with prerequisite training and a thorough knowledge of the particular procedure. Appropriate facilities should be available for coping with situations which may arise as a result of the procedure and for emergency treatment of severe reactions to the contrast agent itself; competent personnel and emergency facilities should be available for at least 30 to 60 minutes after I.V. administration since delayed reactions have been known to occur. These severe life-threatening reactions suggest hypersensitivity to the contrast agent. A personal or family history of asthma or allergy or a history of a previous reaction to a contrast agent warrants special attention and may predict more accurately than pretesting the likelihood of a reaction although not the type nor severity of the reaction in the individual. The value of any pretest is questionable. The pretest most performed is the slow injection of 0.5—1.0 cc. of the preparation which may be given through the needle to be used for the full dose. If no reaction occurs within 15 minutes, the full dose may be given; however, this does not preclude the possibility of reaction. Should the test dose produce an untoward response, the necessity for continuing the examination should be reevaluated. If deemed essential, examination should proceed with all possible caution. In rare instances, reaction to the test dose may be extremely severe; therefore, close observation and facilities for emergency treatment are indicated.

Renal toxicity has been reported in a few patients with liver dysfunction who were given oral cholecystographic agents followed by urographic agents; therefore, if known or suspected hepatic or biliary disorder exists, administration of meglumine diatrizoate should be postponed following the ingestion of cholecystographic agents. Consider the functional ability of the kidneys before injecting meglumine diatrizoate.

The recommended rate of infusion should not be exceeded. The diuretic effect of the drip infusion procedure may hinder an assessment of residual urine in the bladder. Adequate visualization may be difficult or impossible in uremic patients or others with severely impaired renal function (see Contraindications).

Adverse Reactions: Reactions most frequently encountered with drip infusion pyelography are nausea and urticaria. Chills, metallic taste, vomiting, dizziness, a rise or fall in blood pressure, itching, flushing, or generalized feeling of warmth, sneezing, etc. may occur and, rarely, may be severe enough to require discontinuation of dosage. Severe reactions which may require emergency measures (see Precautions) are a possibility and include cardiovascular reaction characterized by peripheral vasodilatation with hypotension and reflex tachycardia, dyspnea, confusion, and cyanosis progressing to unconsciousness. An allergic-like reaction ranging from rhinitis or angioneurotic edema to laryngeal or bronchial spasm or anaphylactoid shock may occur. Temporary renal shutdown or other nephropathy may occur. Intravenous injection of meglumine diatrizoate in a more concentrated formulation has produced a few instances of a burning or stinging sensation and of venospasm or venous pain.

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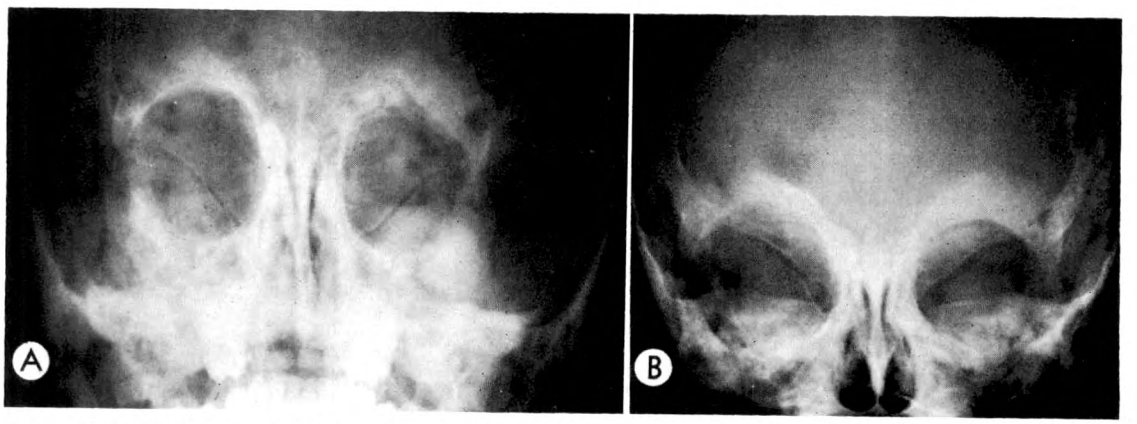


FIG. 2. Case 12. Two year old female with widespread osteolytic lesions throughout the skeleton presented with left-sided proptosis. (*A*) The roof and lateral wall of the left orbit show combined lytic and sclerotic involvement. There is also mild sclerosis of the medial portion of the right orbit. The patient was treated with systemic chemotherapy but received no radiation to either orbit. (*B*) One year later, there is marked sclerosis and thickening of the left orbital roof and lateral wall, and similar but less marked change is seen in the right orbital roof. The left proptosis was still present.

residual proptosis at the most recent follow-up examination.

DISCUSSION

Orbital involvement in histiocytosis X[7] has been emphasized historically through the classical triad of diabetes insipidus, exophthalmos, and lytic lesions of the cranial vault known as Hand-Schüller-Christian disease. When all patients with histiocytosis X are reviewed, however, this triad usually accounts for less than 10 per cent of patients.[3] Although direct invasion of the globe is rarely seen, a displacement of the eye is most often directly related to pressure produced by granulomas occurring in the surrounding orbital wall. Rarely is exophthalmos seen without the typical osteolytic lesions being demonstrated roentgenographically. These findings were noted in 9 of the children (Cases 1–9).

A more unusual finding is that of sclerosis and thickening of the orbital walls. Avery, McAfee, and Guild[1] noted this appearance in several instances of orbital involvement reviewed from their total experience with histiocytosis X. Thannhauser[13] commented that sclerosis occasionally occurs as a residual to treated eosinophilic granuloma in other skeletal sites. Five of our 14 cases of orbital involvement did demonstrate extensive orbital sclerosis. The data gathered concerning the patterns of disease, age of onset, and therapy of this group of patients did not reveal any reasons why these children had roentgenographic changes which were different from the more classical lytic lesions.

It would appear from these 5 cases that there is a progression in some patients from either mixed lytic and sclerotic (Cases 10, 12, and 14) or purely destructive lesions (Case 11) to a healing phase which consists of sclerosis and thickening. One patient (Case 12) had calvarial lesions which showed evidence of surrounding sclerosis. The remaining children did not have other sclerotic lesions elsewhere in the skeleton.

Residual proptosis following resolution of clinically active disease in the orbit was noted in all 5 patients with sclerosis and thickening of the orbital roof. The sclerotic thickened bone produced a depression of the roof of the involved orbit in these cases. It seems likely that the resultant contraction in orbital volume is responsible for the residual proptosis in these individuals. Proptosis also persisted in 2 patients with osteolytic lesions (Cases 2 and 6) following resolution of the acute process, late roentgenograms demonstrating depression of the orbital roof. Kennedy[5] has demon-

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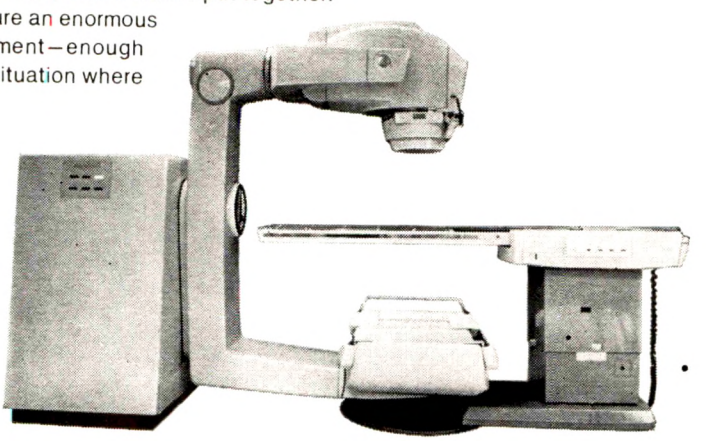
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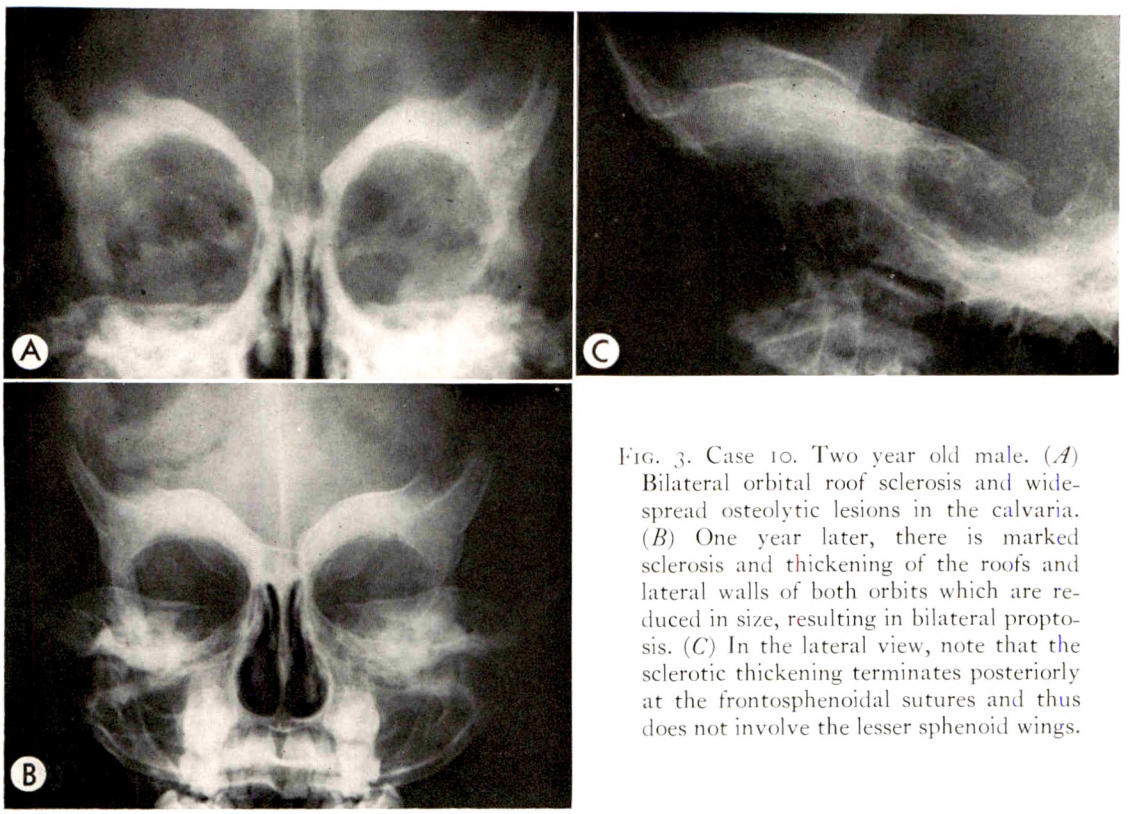


Fig. 3. Case 10. Two year old male. (*A*) Bilateral orbital roof sclerosis and widespread osteolytic lesions in the calvaria. (*B*) One year later, there is marked sclerosis and thickening of the roofs and lateral walls of both orbits which are reduced in size, resulting in bilateral proptosis. (*C*) In the lateral view, note that the sclerotic thickening terminates posteriorly at the frontosphenoidal sutures and thus does not involve the lesser sphenoid wings.

strated that heavy dose irradiation in the first 3 years of life will arrest orbital growth and result in a markedly contracted orbit. However, 2 of our patients (Cases 2 and 9) with extensive lytic lesions who had received no orbital irradiation and were treated only with systemic chemotherapy, nevertheless developed contraction in size of the involved orbits.

The differential diagnosis of sclerosis and thickening of the orbital roof includes fibrous dysplasia,[6] meningioma,[4] dermoid and epidermoid tumors,[8] lacrimal gland carcinoma,[11] osteoblastic metastases from

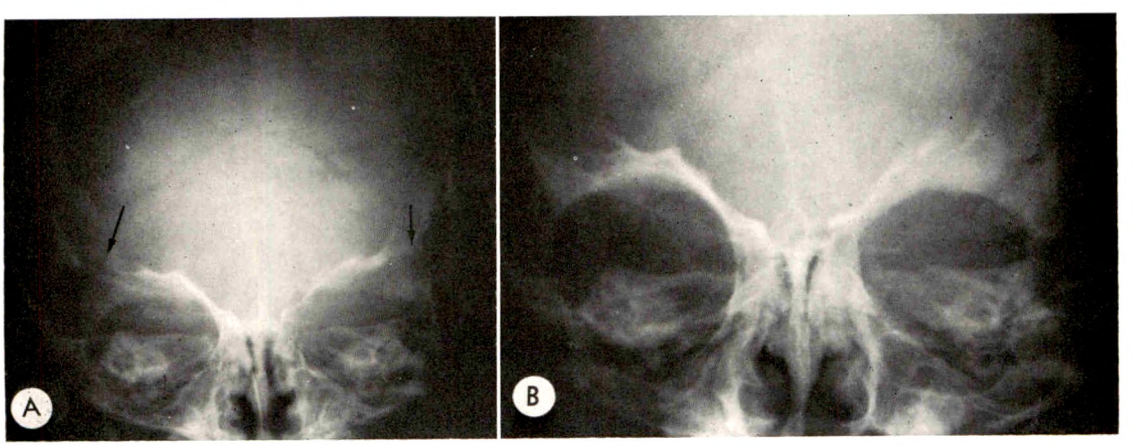


Fig. 4. Case 11. Two year old female. (*A*) Bilateral osteolytic lesions at the lateral margins of the orbital roofs. (*B*) Re-examination 5 months later reveals sclerosis of both orbital roofs with flattening and depression on the left side.

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carcinoma of the breast and prostate,[12] lymphoma,[10] sarcoid,[11] osteitis secondary to chronic sinusitis,[2] and Caffey's disease.[9] The majority of the neoplastic processes listed above occur almost exclusively in adults. Fibrous dysplasia may represent a rather difficult differential, for this process occurs occasionally in children and affects the base of the skull and the orbital roofs, causing either sclerosis or a mixed lytic-sclerotic appearance. The sclerosis, however, is typically much more widespread (often bilateral and involving the greater and lesser sphenoid wings) than was exhibited in our group of patients in whom the hyperostosis did not extend posteriorly beyond the frontosphenoidal suture. Perhaps most important in differentiating sclerosis and thickening of the orbital roofs in histiocytosis X from fibrous dysplasia, chronic sinusitis, or Caffey's disease is the presence of destructive lesions elsewhere in the skeleton in the former process.

SUMMARY

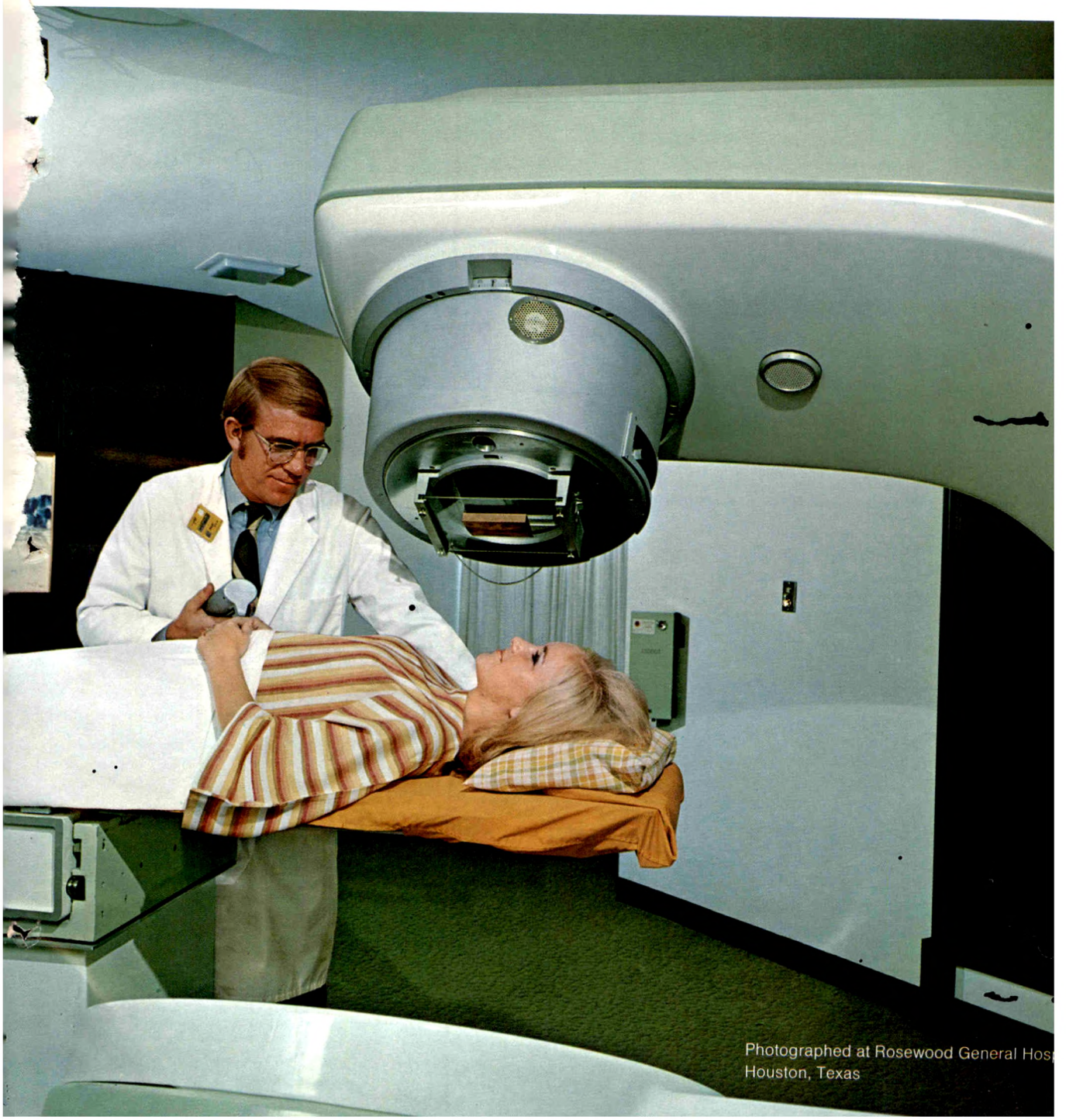
A review of roentgenographic studies of the skull in 62 patients with histiocytosis X revealed 14 with orbital involvement. Five of the 14 have shown increased thickness and sclerosis of the orbital roofs, all with a similar pattern. The remaining 9 cases showed more typical lytic lesions of the roof and zygomatic arch. The occurrence of orbital sclerosis was not related to the type of therapy employed. Serial studies suggest that the sclerosis and thickening are related to healing of destructive areas within the orbital roofs.

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CHEMODECTOMAS OF THE HEAD AND NECK*

By ENRIQUE PALACIOS, M.D.†
KANSAS CITY, KANSAS AND LUND, SWEDEN

EXTENSIVE discussions of chemodectomas reveal some unanswered questions concerning their diagnosis and treatment. The characteristic slow growth of these tumors, and their anatomic location adjacent to critical structures, result in surgical difficulties and considerable morbidity. There are however, relatively few references, stressing the importance of angiography and tomography in the detection and evaluation of these uncommon tumors. The observation of a number of these lesions stimulated our interest in reviewing the chemoreceptor system and its pathology, and in reporting 24 chemodectomas, 21 of which were located in the tympanojugular area.

The term "chemodectoma" was first proposed by Mulligan in 1950[39] to describe a neoplasm arising from the chemoreceptor system. "Glomus tumor" and "nonchromaffin paraganglioma" are synonyms. The lesions are named according to their location in the body.

The chemoreceptor system represents a number of histologically similar structures located in various parts of the body (Fig. 1). They consist of groups of cells with sensory innervations which respond reflexly to changes in the chemical composition of the arterial blood.[48]

Histologically chemodectomas have cellular characteristics, essentially the same as normal chemoreceptor tissue. LeCompte[32] describes two less common variants of the normal pattern: one with an agiomatous pattern where the vascular elements are profuse, and the other with an adenoma-like pattern. Marshall and Horn[35] in a comparative study divide the chemodectomas into 3 types: the predominantly vascular tumor, the mixed type or "usual type," and the predominantly cellular tumors.

Chemodectomas are slow growing and extremely variable in size, ranging from few mm. to 10 or more cm. They are usually encapsulated, but may sometimes be intimately bound to vessels and other adjacent structures.[9] Although the vast majority of chemodectomas are histologically benign, they may invade locally or recur, and may infrequently metastasize.[25,41,42,49,50]

The chemodectomas are much more common in females than in males, particularly the intratympanic and glomus jugulare tumors. The majority of these lesions are unilateral, but bilateral tumors are occasionally found.[5,9,12,13,48] Other combinations of simultaneous occurrence of chemodectomas, although rare, have been reported: carotid body tumor and glomus jugulare tu-

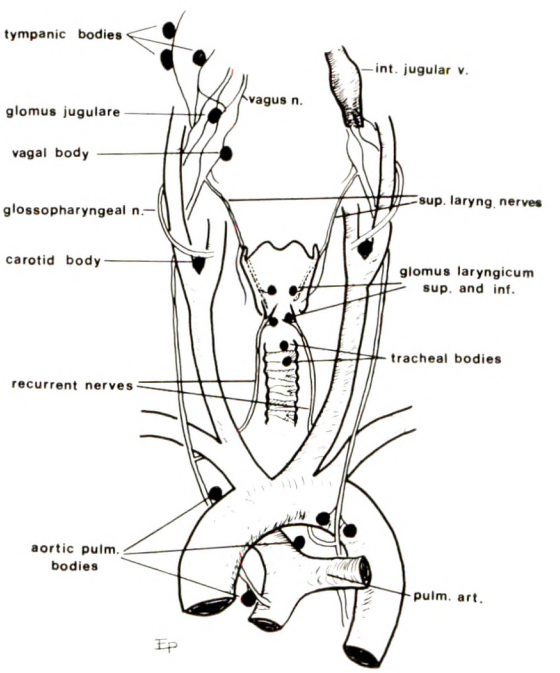


FIG. 1. Diagram illustrating the sites in which chemoreceptor bodies (glomera) are known to occur.

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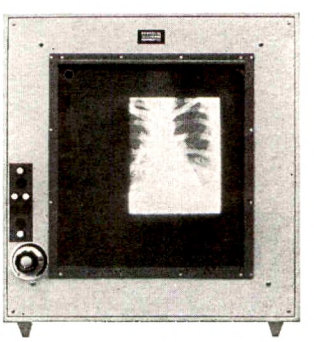
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mors;[11,13,24,28,45] carotid body tumor and vagal tumors;[34] carotid body tumor and chemodectoma of the orbit.[30]

The rare feature of heredofamilial tendency has been shown. Desai and Patel[12] described 5 cases of carotid body tumors occurring in one family. Two members of this family had bilateral tumors. Borden[5] reported 2 cases of tympanojugular tumors occurring in father and son, one of whom had bilateral tumors.

Chemodectomas of the ciliary ganglion of the eye[15,30] and of the mandible[7] have been reported. However, glomus or chemoreceptor tissue of the ciliary ganglion has not been found in man[6] and no definite alveolar or mandibular glomus has yet been described. There are reports of tumors arising from chemoreceptors located in regions other than the head and neck: in the mediastinum, from the aortic-pulmonary bodies;[13,31,38] in the retroperitoneal; and in the femoral areas.[3,9,40]

CLINICAL FEATURES

I. CAROTID BODY TUMORS

The best known chemoreceptor is the carotid body, situated at the bifurcation of each of the common carotid arteries. It is a small mass measuring about 5 mm. in greatest diameter. The carotid bodies are highly vascularized, containing islands of chemoreceptor cells surrounded by vascular sinusoids. Their nerve supply is predominantly sensory, the nerve fibers ascending to the medulla oblongata via a branch of the glossopharyngeal nerve.[1,46] Their blood supply comes from the external carotid artery.

The carotid body tumor manifests itself as a painless mass in the lateral aspect of the neck. Due to its slow growth and the absent or minimal symptoms, it is not usually discovered until it has grown for some time. The duration of the growth may range from months to many years before detection.

When the lesion is large, it may produce pressure on the surrounding structures causing dysphagia, hoarseness, cough and nausea. Despite the intimate topographic relationship between the carotid sinus and the tumor, carotid sinus symptoms are remarkably rare.

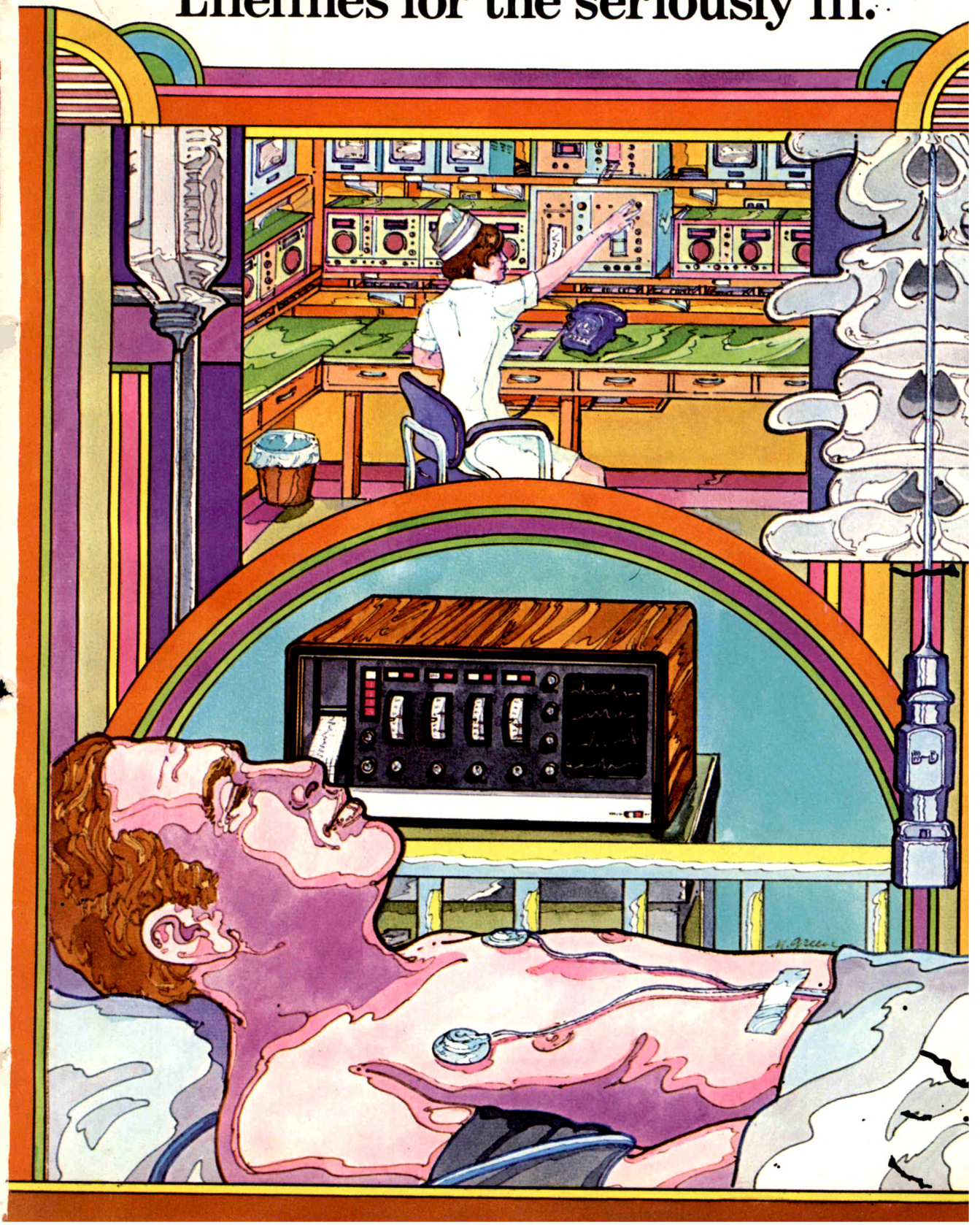
Physical examination, as a rule, reveals only a tumor mass at the carotid bifurcation, usually movable laterally but not vertically. The mass may be pulsatile and may be associated with a thrill or bruit, making its differentiation from an aneurysm difficult.

II. INTRATYMPANIC AND GLOMUS JUGULARE TUMORS

A second important group of chemoreceptors is the glomus jugulare complex located in the jugular and middle ear areas. It was identified for the first time by Guild[16] in 1941. In 1953 he expanded the description of these structures after examining 88 temporal bones in 44 subjects.[17] These glomera are situated mainly along the structures of the glossopharyngeal nerve: by the superior ganglion, in the adventitia of the bulb of the internal jugular vein; along the tympanic nerve (Jacobson's nerve); and in the tympanic plexus (tympanic bodies) located in the mucosa of the cochlear promontory (Fig. 2). Glomera are also found along the auricular branch of the vagus nerve (Arnold's nerve), sometimes as far as the facial canal. Each ear contains from 1 to 3 glomera. They tend to be bilateral and similar in location on both sides. They are ovoid in shape and measure from 0.1 to 1.5 mm. in greatest diameter. Histologically they are similar to the carotid body and other chemoreceptors. They are innervated by the glossopharyngeal nerve. The blood supply is mainly derived from the external carotid artery via the tympanic branch of the ascending pharyngeal artery.

Recently, electron microscopic studies of the glomus jugulare tumor have been reported.[20] Chemoreceptor units and specialized cytoplasmic expansions, as described in the normal chemoreceptor organ, were not identified. The authors were unable to compare these findings with a normal che-

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moreceptor organ since no electron microscopic studies of a normal glomus jugulare were published at the time. Further investigations are needed to substantiate these limited observations.

A. *Intratympanic glomus tumor* was first described by Rosenwasser in 1945.[43] The tumor produces a variety of symptoms determined by the location, direction of spread of the tumor, and the stage at which the patient is seen.[10] The symptoms may be aural, neurologic or both. The usual symptoms are progressive decrease in hearing acuity, tinnitus (often pulsatile in nature), vertigo, pain, bloody discharge, and secondary infection after rupture of the tympanic membrane. The tumor occasionally manifests itself, as an isolated facial paralysis. A combination of otologic and neurologic symptoms may occur when extension of the tumor involves the petrotympanic and jugular areas. The lesion may penetrate through the dura into the middle and posterior cranial fossae.[14]

These tumors are usually detected when a polypoid mass breaks through the tympanic membrane or wall of the external auditory canal. Otologic examination in the early lesions reveals a slight bluish discoloration[10] and a suggestion of a mass behind the tympanic membrane.

B. *Glomus jugulare tumor* arises from the adventitia of the bulb of the internal jugular vein in the jugular fossa. It may cause aural and neurologic symptoms similar to those produced by lesions arising in the tympanic area depending on the direction of spread. A long history is characteristic. The vein may become compressed and there may be extension of the tumor into its lumen. The lesion destroys the roof of the jugular fossa extending either into the middle ear or into the external auditory canal, or posteriorly and medially into the jugular foramen producing paresis of the cranial nerves passing through it (9th, 10th, and 11th). The latter may be the only clinical manifestation of the lesion. Further extension medially may involve the 12th cranial nerve as it passes through the hypoglossal canal. Occasionally the trigeminal and abducens nerves are affected when the lesion reaches the middle cranial fossa through the tip of the petrous bone.[24]

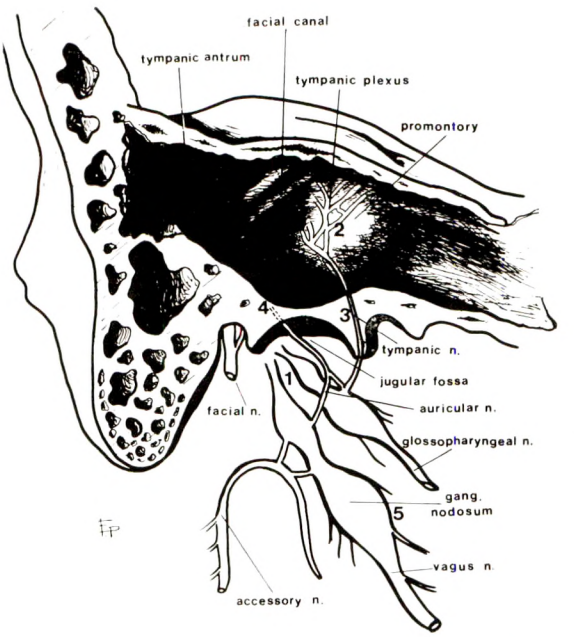


Fig. 2. Schematic representation of the sites of glomera in the middle ear and jugular fossa: (1) superior ganglia, in the adventitia of the internal jugular vein (glomus jugulare); (2) tympanic plexus (tympanic bodies); (3) tympanic nerve; (4) auricular nerve; (5) ganglion nodosum (vagal bodies).

III. VAGAL TUMORS

The third important site for chemoreceptor tissue to occur is the vagus nerve in its cervical portion: around the inferior ganglion or ganglion nodosum (vagal bodies); and along the rest of the cervical portion (glomus intravagale). Up to now 13 cases of vagal tumors have been reported in the literature.[4,22,31,34] The usual clinical manifestation of these tumors is a painless mass behind the angle of the mandible which frequently bulges into the pharyngeal wall producing dysphagia. Since the mass lies in close relation to the carotid vessels, it may be pulsatile mimicking an aneurysm. Chemoreceptor hyperactivity and vagal dysfunction have been present in 3 of the reported cases and involvement of the

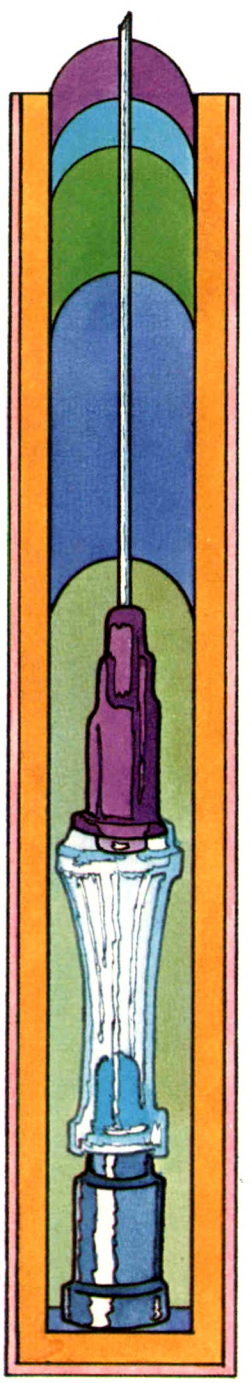
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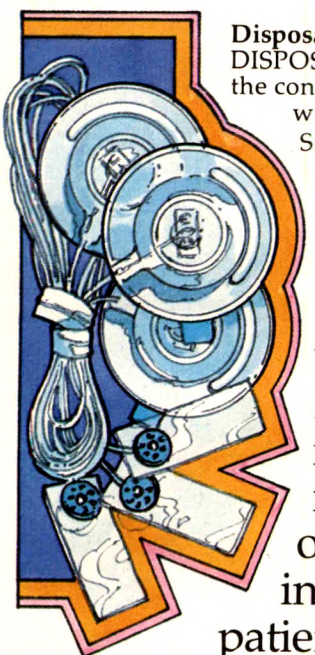
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TABLE I

ANALYSIS OF CASES OF CHEMODECTOMAS OF THE HEAD AND NECK

Case No.	Sex	Age	Clinical Information	Roentgenographic Information	Surgical and Pathologic Information
1	F	45	Pulsatile tinnitus on the right for 3 years, mild loss of hearing; redness on the posteroinferior portion of the tympanic membrane	Slight erosion of the right jugular foramen on plain roentgenograms and tomograms; arteriography showed a vascular tumor at the jugular fossa (Fig. 3, *A* and *B*); phlebography revealed complete obstruction at the jugular bulb by the tumor (Fig. 4)	Glomus jugulare tumor removed; the mass was protruding into the lumen of the internal jugular vein
2	F	64	Pulsatile tinnitus on the right for several years, moderate conductive hearing loss; red polypoid mass protruding through the right tympanic membrane	Plain roentgenograms and tomograms revealed a large bone destruction of the right jugular fossa (Fig. 5)	Large glomus jugulare tumor removed; not attached to the internal jugular vein
3	F	54	Paresis of the 9th, 10th, 11th and 12th cranial nerves on the right, tinnitus for 1 year, intermittent vertigo; a glomus jugulare tumor had been removed on the same side 14 years before	Arteriography revealed a slight vascular tumor in the right jugular area; phlebography showed extrinsic pressure at the bulb of the jugular vein and sigmoid sinus with partial obstruction (Fig. 6)	Large glomus jugulare tumor (recurrence) involving the cranial nerves and internal jugular vein; partially removed
4	M	49	Hearing loss on the right for many years, intermittent dizziness; bluish mass seen through the tympanic membrane	Major destruction in the right jugular fossa extending into the middle ear (Fig. 7; and 8); phlebography showed extrinsic pressure on the jugular bulb	Partial removal of a large glomus jugulare tumor, extending into the middle ear
5	F	66	Bilateral loss of hearing and progressive tinnitus for 1 year, intermittent bleeding and vertigo; bilateral intratympanic polypoid masses, protruding through the tympanic membrane	Plain roentgenograms and tomograms revealed clouding of the petrotympanic areas bilaterally; external carotid angiogram on the right showed a large vascular intratympanic mass (Fig. 9.); common carotid angiography on the left revealed a vascular intratympanic mass (Fig. 10); bilateral jugular phlebography was normal	Bilateral, large intratympanic glomus tumors removed
6	F	60	Pulsatile tinnitus on the right for 15 years, progressive hearing loss and intermittent bleeding; pulsatile mass protruding through the right ear, bruit audible over the right mastoid area	Plain roentgenograms and tomograms showed large destruction of the jugular fossa and middle ear; arteriography revealed a large vascularized mass in the jugular area extending into the middle ear and down in the neck (Fig. 11)	Partial removal of a large glomus jugulare tumor, involving the middle ear and extending down into the neck
7	F	41	Tinnitus on the left for several years, intermittent discharge, pain and bleeding; bluish mass protruding through the tympanic membrane; father had a carotid body tumor removed	Plain roentgenograms and tomograms revealed clouding of the petrotympanic area; arteriogram showed a small vascular mass in the left jugular fossa and hypotympanum	Glomus jugulare tumor removed from the left hypotympanic area; internal jugular vein was intact
8	F	70	Pulsatile tinnitus on the left, chronic discharge and progressive hearing loss; tumor protruding through the left ear, congestive heart failure	Chronic inflammatory changes in the left petrotympanic area and slight enlargement of the jugular foramen, seen on plain roentgenograms and tomograms	Impression, intratympanic glomus tumor; surgery deferred
9	F	39	Bluish discoloration on the right tympanic membrane on routine examination and suggestion of a mass behind the membrane; asymptomatic	Plain roentgenograms and tomograms showed slight clouding of right tympanic area	Small intratympanic glomus tumor removed
10	F	42	Tinnitus on the left; a red cherry spot seen in the posteroinferior portion of the tympanic membrane	Normal plain roentgenograms and tomograms	Small intratympanic glomus tumor removed
11	F	38	Progressive decrease of hearing acuity and tinnitus for 5 years, pain and discharge; red polypoid mass protruding through the left tympanic membrane	Clouding and sclerosis of the petromastoid cells and tympanic cavity, seen on plain roentgenograms and tomograms on the left	Large intratympanic glomus tumor removed from the hypotympanum
12	F	40	Progressive tinnitus and hearing loss on the left; small bluish mass bulging through the tympanic membrane	Slight clouding of the middle ear on the left seen on plain roentgenograms and tomograms	Small intratympanic glomus tumor removed
13	F	29	Slight hearing impairment and tinnitus of the left; bluish discoloration and increased vascularity of the tympanic membrane	Plain roentgenograms and tomograms showed clouding of the left tympanic cavity and suggestion of a soft tissue mass	Intratympanic glomus tumor removed
14	F	39	Tinnitus for 2 years, intermittent discharge and decreased hearing on the left	Slight clouding of the left tympanic cavity on plain roentgenograms and tomograms	Intratympanic glomus tumor removed
15	F	33	Persistent tinnitus on the left for several years, slight hearing impairment; small bluish mass seen through the left tympanic membrane	Slight clouding of the left tympanic cavity on plain roentgenograms and tomograms	Small intratympanic glomus tumor removed
16	F	58	Chronic otitis media on the left, decreased hearing acuity	Chronic inflammatory changes in the middle ear on the left on plain roentgenograms and tomograms	Chronic inflammatory changes; small intratympanic glomus tumor removed
17	F	67	Progressive tinnitus and hearing loss for several years on the left; bluish discoloration on the tympanic membrane	Clouding of the left tympanic cavity on plain roentgenograms and tomograms	Intratympanic glomus tumor removed

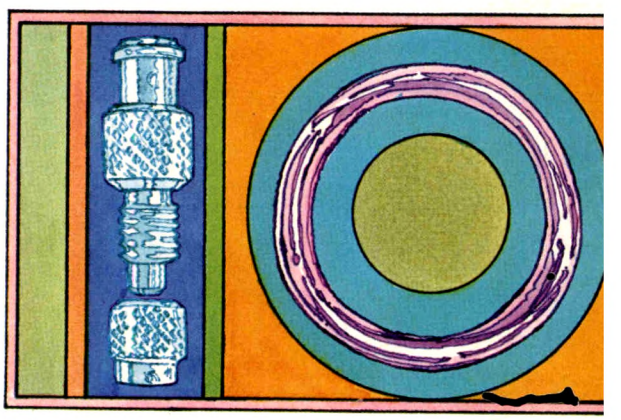
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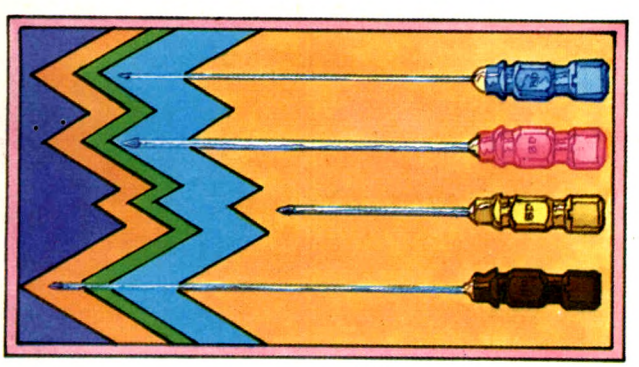
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TABLE I (*Continued*)

Case No.	Sex	Age	Clinical Information	Roentgenographic Information	Surgical and Pathologic Information
18	F	62	Hearing loss and pulsatile tinnitus for several years on the right; occasional discharge and bleeding; mass seen through the right tympanic membrane	Chronic inflammatory changes in the right petromastoid cells and tympanic cavity, seen on plain roentgenograms and tomograms	Chronic inflammatory changes; intratympanic glomus tumor removed
19	F	52	Intermittent tinnitus on the left for several years; slight bluish discoloration and bulging of the left tympanic membrane	Plain roentgenograms and tomograms showed clouding of the left tympanic cavity; normal arteriography and jugular phlebography	Intratympanic glomus tumor removed
20	M	43	Pulsatile tinnitus and pain on the left; small mass in the tympanic cavity was seen through the tympanic membrane	Plain roentgenograms and tomograms revealed clouding of the petrotympanic area on the left	Small intratympanic glomus tumor removed
			Patient readmitted 2 years later because of symptoms of recurrence of the glomus tumor	Carotid angiography revealed no definite evidence of tumor in the tympanojugular area, but a large vascularized tumor was discovered at the carotid bifurcation on the left (Fig. 12)	Recurrence of intratympanic glomus tumor Carotid body tumor (not removed)
21	F	45	Painless pulsatile mass for many years, just below the angle of mandible on the left; bruit heard over the mass; chronic congestive heart failure	Common carotid angiography revealed a large vascularized mass at the carotid bifurcation (Fig. 13)	Carotid body tumor on the left; surgery deferred
22	F	55	Progressive dysphagia and hoarseness for several years; pulsatile mass protruding into the left retropharyngeal wall	Soft tissue mass in the retropharyngeal area seen on plain roentgenograms; common carotid angiography revealed a vascularized mass above and medial to the carotid bifurcation, displacing the external carotid artery lateral and posteriorly (Fig. 14)	Vascularized mass at the left retropharyngeal wall with extensive infiltration of the surrounding tissues, which was considered unresectable; biopsy revealed chemodectoma tissue; diagnosis was vagal tumor

cranial nerves (9th, 10th, 11th, and 12th) in 4 cases. Simultaneous occurrence with other chemodectomas has also been demonstrated. Malignant characteristics have been shown in 5 cases, 1 with extensive metastasis to bone and lungs.[8]

IV. LARYNGEAL AND TRACHEAL CHEMODECTOMAS

Recently, other glomera of chemoreceptor tissue have been demonstrated in the larynx. Watzka[53] reported the discovery of a glomus body next to the internal branch of the superior laryngeal nerve (glomus laryngicum superior). Kleinsasser[29] described similar structures between the anterior and posterior branches of the recurrent laryngeal nerve adjacent to the cricoid and thyroid cartilages (glomus laryngicum inferior). Reports of chemodectomas in the trachea suggest that these lesions might have originated from a misplaced carotid body. However, LeCompte's theory[33] of segmental arrangements of the chemoreceptors (which correspond to the branchial arches) would suggest that these tumors arise from true glomera in the trachea. The 8 reported cases[2,23,26,36,37,54] of laryngeal and tracheal chemodectomas had a subglottic mass with involvement of the vocal cords, laryngeal obstruction and extension into the thyroid area.

ROENTGENOGRAPHIC ASPECTS

I. CAROTID BODY TUMORS

The profuse vascularity of these tumors, makes carotid angiography one of the most valuable methods in the diagnosis. Idbohrn[27] was among the first to make a roentgenographic preoperative diagnosis of these lesions.

Angiography yields important information as regards the size of the lesion and relationship with the carotid vessels and other adjacent structures. The cerebral blood flow from the opposite side of the lesion should be assessed either by performing carotid angiography on the unaffected side or by compression of the uninvolved carotid artery during the injection of contrast medium in the affected side. Knowledge of the patency of the collateral circulation via the circle of Willis or extracranial vessels, is of extreme importance should ligation of the affected artery be-



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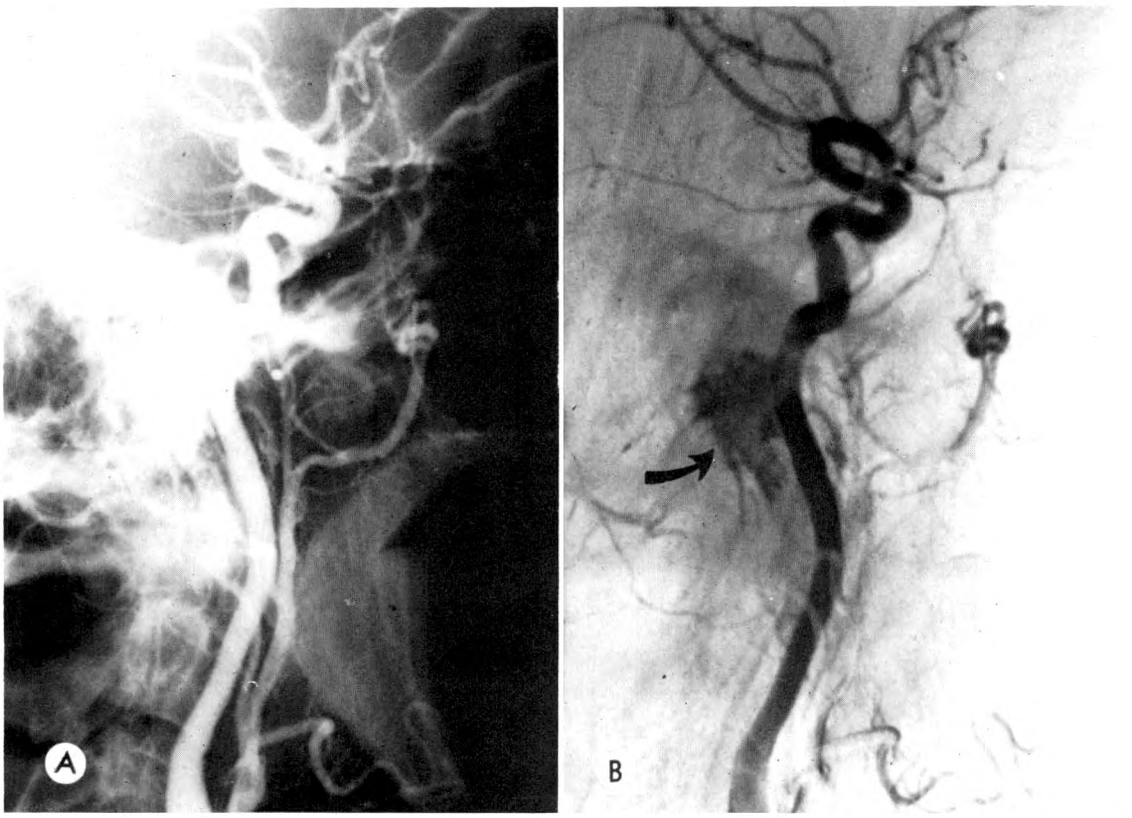


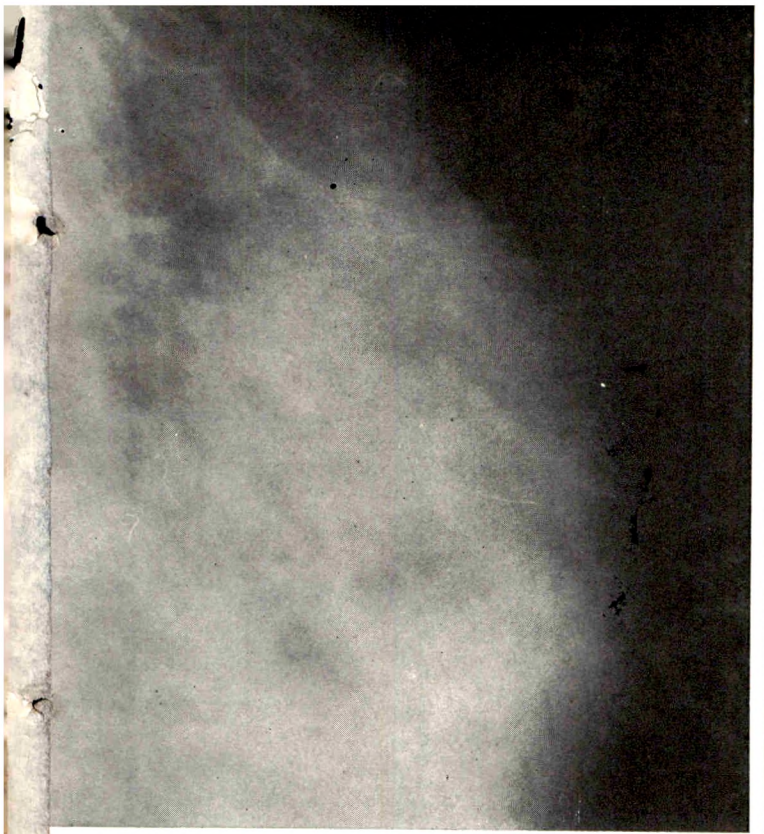
FIG. 3. Case 1. Common carotid angiography illustrating the importance of the use of subtraction technique for better demonstration of vascularized lesions. (*A*) Arteriogram before subtraction. (*B*) Subtraction film demonstrating the exact size and extension of a glomus jugulare tumor (arrow). Note the large feeding vessels.

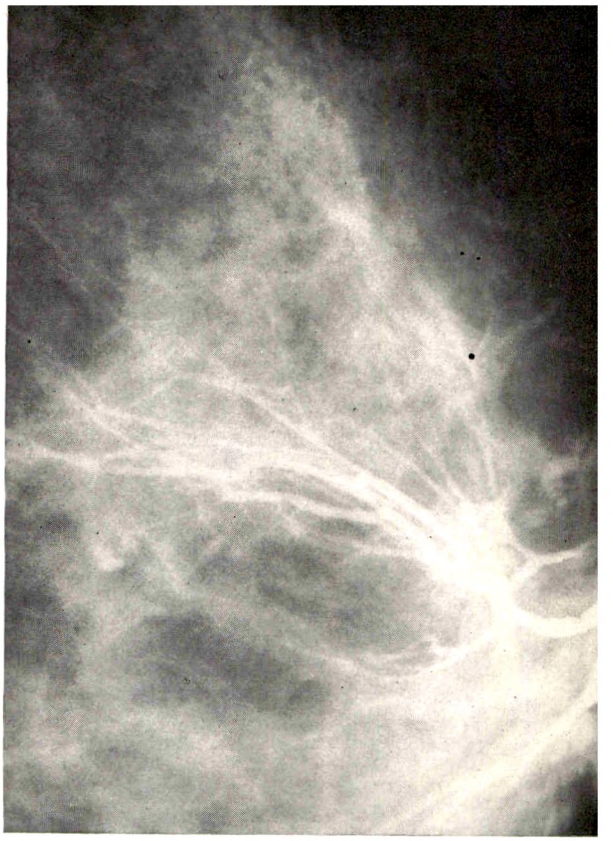
come necessary during the surgical procedure. Carotid angiography is performed either by direct puncture of the common carotid artery or by catheterization from a distal artery, usually the femoral artery. The latter is especially useful when dealing with large masses. Rapid serial filming is important for the best possible appreciation of the tumor's anatomic and physiologic characteristics.

Precise diagnosis is established in the great majority of the cases. The tumor appears to be well circumscribed. Contrast medium accumulates within a network of small irregular vessels; the tumor opacification is not as homogeneously dense as in carotid aneurysm and differentiation is not difficult. Usually the carotid vessels at the bifurcation are separated from each other by the tumor mass. The vessels involved by the tumor may appear slightly narrowed by pressure but are almost never occluded.

II. INTRATYMPANIC AND GLOMUS JUGULARE TUMORS

Early roentgenographic detection and delineation of these tumors requires a high degree of suspicion since the clinical manifestations are frequently so vague. Also, accurate diagnosis necessitates a thorough knowledge and recognition of numerous normal anatomic variations of the bony structures. Mastoid cells frequently grow into the walls of the carotid canal and internal auditory meatus, extending to the apex of the petrous bone. These radiolucencies may simulate bone destruction. The size and shape of the jugular foramina vary considerably and have a direct relationship to the size of the bulb of the in-





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ternal jugular vein. The recognition of a large and asymmetric jugular foramen, as a normal variation, is essential and should not be confused with a pathologic enlargement. The sigmoid sinus may vary in position and shape, and the groove of the retromastoid emissary vein may be enlarged when there is obstruction of the sigmoid sinus or internal jugular vein. However, this should be interpreted cautiously, recognizing the normal variations. Bone destruction of the apex of the petrous bone may be simulated by a large venous lake produced by the junction of the superior and inferior petrosal sinuses.[44]

Initially, intratympanic tumors are manifested by opacification or loss of aeration of the petromastoid cells. However, the commonly superimposed infection, which follows the perforation of the tympanic membrane by these tumors is often indistinguishable from otitis media. As the lesion progresses, bone destruction may be detected in the hypotympanic area, mastoid cells, external auditory canal, and jugular foramen. If the glomus jugulare tumor is large enough, it will destroy the roof of the jugular fossa and expand into the middle ear and adjacent structures, behaving like a large intratympanic tumor. At this stage it is difficult to determine the site of origin of the tumor. Occasionally, the lesion may extend anteriorly involving the posterior portion of the temporomandibular joint.

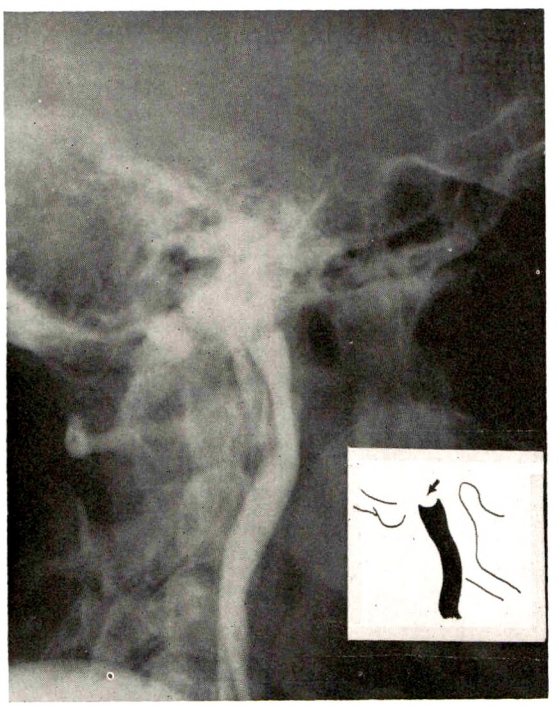


Fig. 4. Case 1. Internal jugular phlebography. Complete obstruction of the vein at the jugular foramen (arrow) by the tumor extending into its lumen.

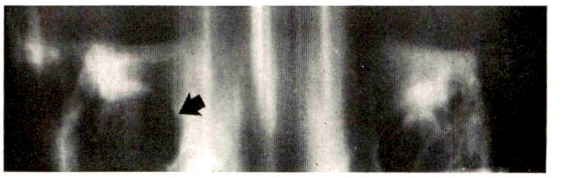


Fig. 5. Case 2. Tomogram revealing extensive bone destruction of the right jugular fossa (arrow), due to a large glomus jugulare tumor.

Plain roentgenograms of the skull and petromastoid areas in standard projections remain the basis of the study. Special views for demonstration of the jugular fossa are very helpful. The latter include: the modified submentovertical view where the roentgen-ray beam enters the midline about 1 inch below the tip of the chin, the beam being aligned with the external auditory meatus; and the transoral view[52] where the roentgen-ray beam is centered to the open mouth between the upper and lower molars and is aligned with the external auditory meatus. From the transoral position, additional oblique views can be obtained by turning the patient's head about 10° to each side, allowing the ray to pass through each jugular foramen.

Tomography is one of the most helpful methods for demonstrating in detail the pathologic changes involving the middle ear, the mastoid and the jugular areas, in the early as well as advanced stages. Tomograms are usually taken in anteroposterior projection; in Stenver's position they may provide additional information. These techniques are needed to demonstrate the hypotympanum and sublabyrinthine areas of the middle ear. Hypocycloidal tomography



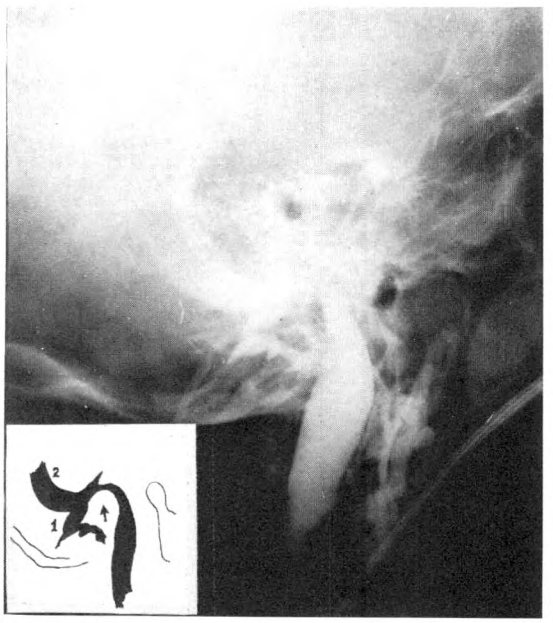


FIG. 6. Case 3. Internal jugular phlebogram. Compression and partial obstruction at the jugular bulb and sigmoid sinus (arrow), produced by a large glomus jugulare tumor; 1 = engorgement of the retromastoid emissary vein, 2 = sigmoid sinus.

will demonstrate these structures with better clarity.

Arteriography provides the best demonstration of the size and extension of highly vascularized tumors and significantly aids in the differential diagnosis. The structures involved are mainly supplied by the tympanic branch of the ascending pharyngeal artery and small meningeal branches of the external carotid artery; however, some meningeal branches from the internal carotid[51] and vertebral arteries may contribute to their supply. Carotid angiography, especially selective external carotid arteriography, will demonstrate vascularized lesions and their blood supply in the majority of the cases. Vertebral angiography should be carried out if there is clinical evidence of middle and posterior cranial fossae involvement. Subtraction techniques demonstrate the size of the lesion more accurately and may actually be the only method for detecting small tumors and arteriovenous shunts. The latter condition is not infrequently seen with these



FIG. 7. Case 4. Tomogram showing a large bone destruction of the right jugular fossa with extension to the hypotympanic area (arrow), produced by a large glomus jugulare tumor.

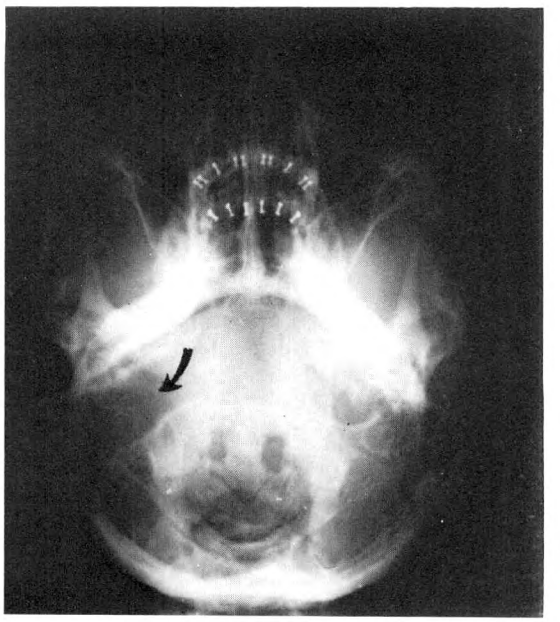


FIG. 8. Case 4. Modified submentovertical view demonstrating the extensive destruction of the right jugular fossa (arrow).

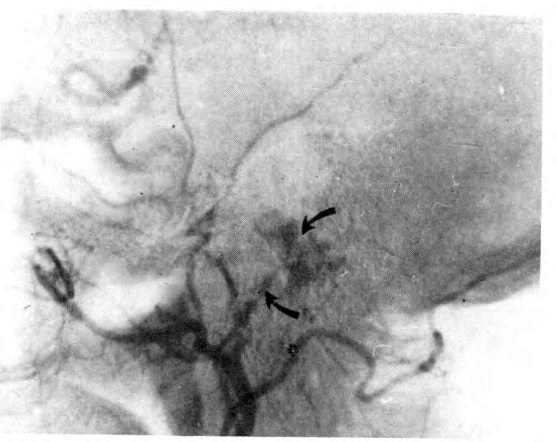


FIG. 9. Case 5. Subtraction film of the right external carotid angiogram. Large vascularized intratympanic glomus tumor (upper arrow). Note the large feeding vessels (lower arrow).

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lesions and is manifested by retrograde filling of the sigmoid sinus during the arterial phase.

Internal jugular phlebography, as described by Gejrot and Lauren,[18,19] is also a very valuable preoperative method in the study of the extension of glomus jugulare tumors. With this technique, known as retrograde jugulariography, it has been possible to detect compression of the jugular vein by the tumor.

Jugular phlebography is carried out by advancing a catheter percutaneously (using Seldinger's technique) as far as the jugular bulb, where contrast medium is injected in retrograde manner with the highest possible manual pressure and with the vein being compressed just distal to the site of puncture. Normally, there is good opacification of the sigmoid sinus and jugular vein of the opposite side as well.[21]

This method for visualization of the jugular vein is used to complement the information of the other studies, especially in those cases where arteriography may have failed to demonstrate the lesion.

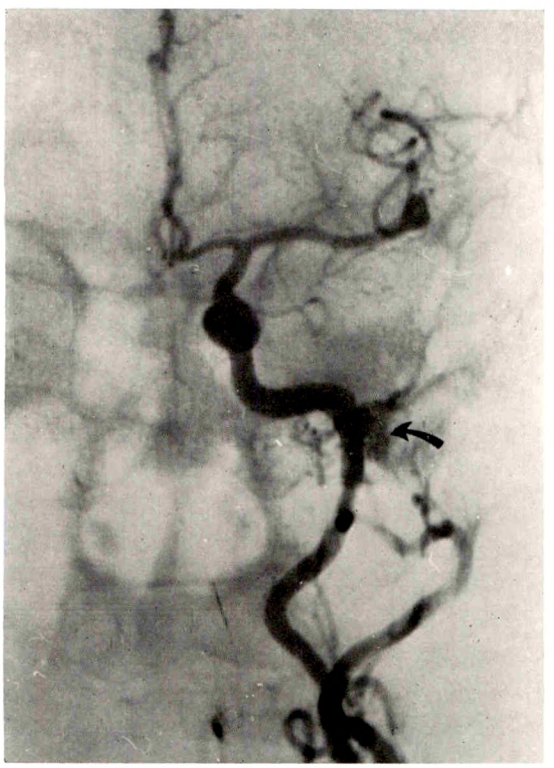


Fig. 10. Case 5. Common carotid angiogram on the left (subtraction film) demonstrating an intratympanic tumor (arrow). Patient had bilateral tumors (see Fig. 9).

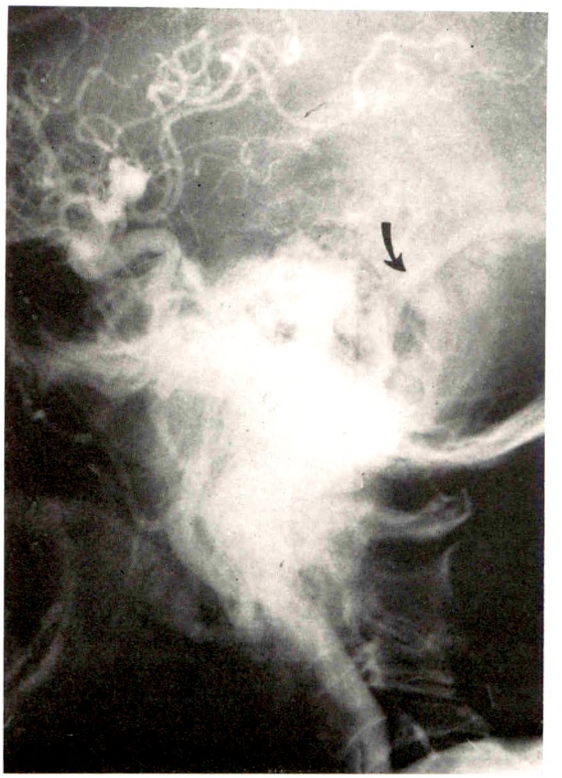


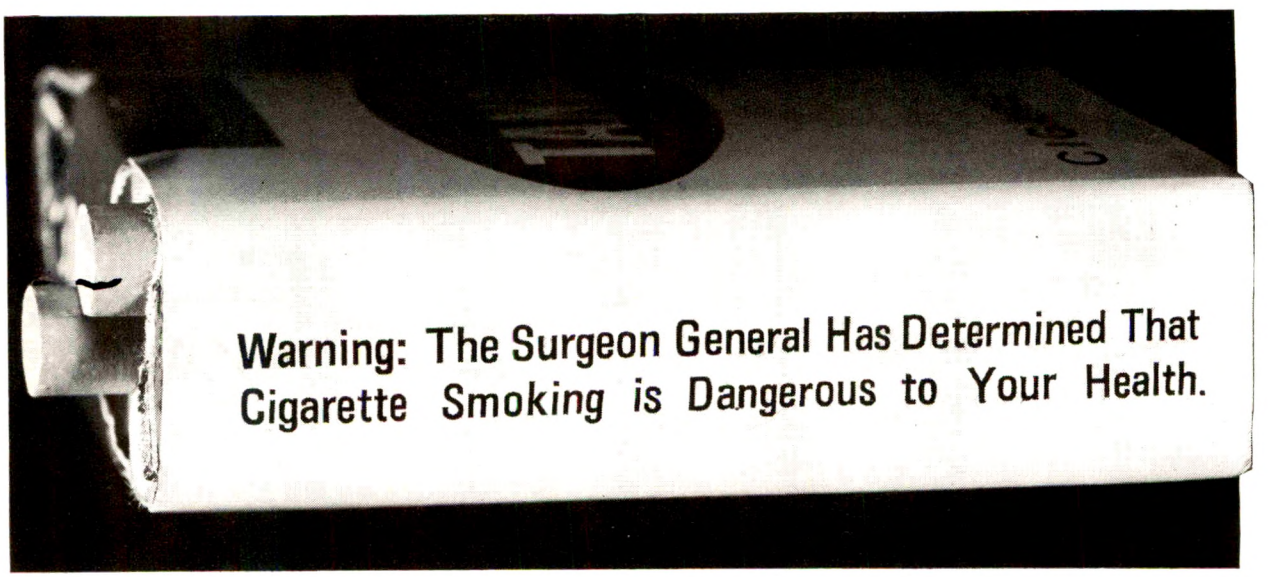
Fig. 11. Case 6. Right common carotid angiogram. Large highly vascularized glomus jugulare tumor which involves not only the base of the skull, but extends down in the neck. Note filling of the sigmoid and transverse sinuses (arrow) due to arteriovenous shunts through the tumor with reversal of blood flow.

III. VAGAL TUMORS

A soft tissue mass encroaching upon the pharyngeal wall can be seen on plain roentgenograms. Arteriography yields more specific information, showing a vascularized mass above the carotid bifurcation. The arteriographic features of vagal tumors are similar to those of the other chemodectomas; but vagal tumor is distinguished by its location. When a mass is located away from the carotid bifurcation and not in close relation to the temporal bone, the most likely diagnosis is a vagal tumor.

Only 3 of the reported cases of vagal tumors appear to have had angiography performed; 2 of them showed marked an-

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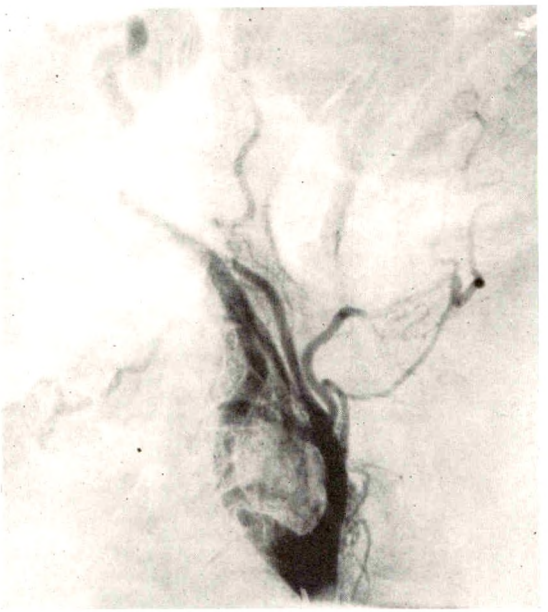


Fig. 12. Case 20. Subtraction film of the common carotid arteriogram. Vascularized carotid body tumor, producing the characteristic separation of the carotid bifurcation.

terior and medial displacement of both carotid vessels.

Since vagal tumors are supplied by the external carotid artery, both common and selective external carotid arteriography should be performed to determine more accurately their blood supply. This knowledge is especially important if the removal of the tumor is contemplated.



Fig. 13. Case 21. Common carotid angiogram revealing a highly vascularized carotid body tumor, separating the carotid bifurcation.

IV. Laryngeal and Tracheal Chemodectomas

Plain film roentgenography and tomography of the larynx and trachea have been used in the diagnosis of these rare tumors. Although no angiographic diagnosis has been reported as yet, the profuse vascularity of these lesions should make arteriography an important method for better preoperative evaluation.

Conclusions

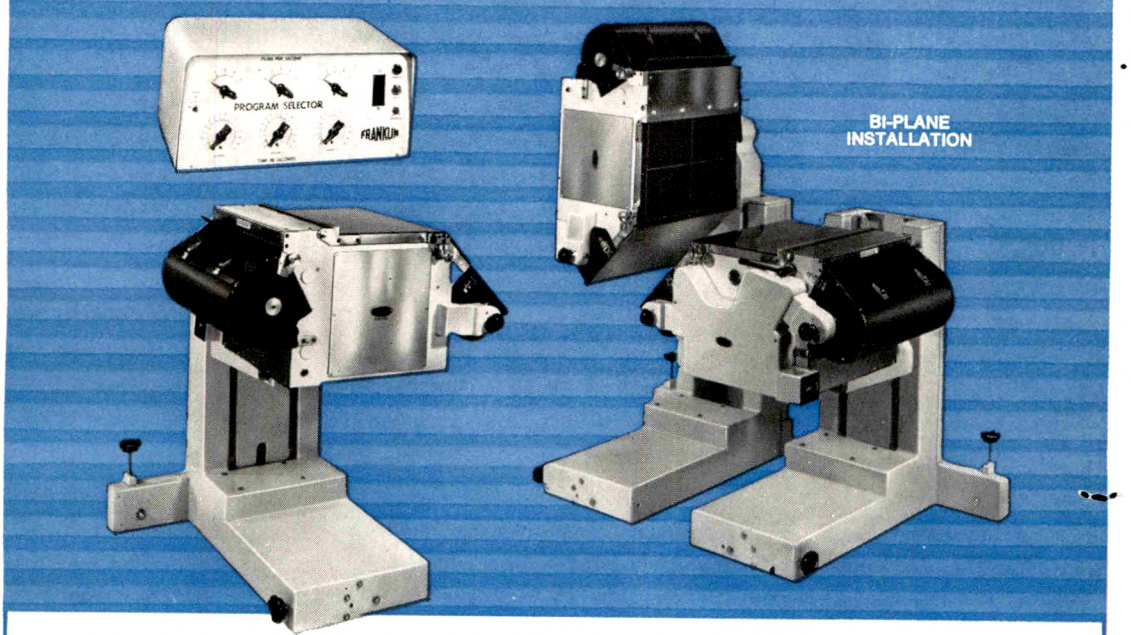
In a series of 24 chemodectomas in 22 patients, the intratympanic glomus tumor was the most common one (15 lesions). Of 6 glomus jugulare tumors, 1 recurred after 14 years involving the 9th, 10th, 11th,



Fig. 14. Case 22. Common carotid angiogram. Large vascularized vagal tumor, located anteriorly, above and medially to the carotid bifurcation. Posterior and lateral displacement of the external carotid artery.

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and 12th cranial nerves, indicating that the lesion spread medially and posteriorly. Glomus tumors located in the tympanojugular area are now regarded as the most common neoplasms involving the middle ear.

Our group of chemodectomas has demonstrated that these lesions occur predominantly in females (20 patients). The age of occurrence ranged from 29 to 70 years. Multicentricity was demonstrated in 2 patients: in one of these, simultaneous intratympanic and carotid body tumors; and in the second case, bilateral intratympanic glomus tumors. Familial tendency was shown in 1 case. Symptoms of chemoreceptor hyperactivity or signs of local or distant malignancy were not detected in any of the cases.

One vagal chemodectoma is presented, it is probably the 14th case reported in the literature and the 4th case diagnosed angiographically. As in most other instances, this case presented with a retropharyngeal mass as the main symptom.

The roentgenographic findings of chemodectomas of the head and neck are quite definite and it should not be difficult to distinguish them from other conditions. In the skull, the lesions to be differentiated are: cholesteatomas of the temporal bone; primary and secondary malignant tumors of the middle ear; and acoustic neuromas. In the cervical area, a number of lesions can simulate chemodectomas clinically but rarely roentgenographically. Among the most common conditions are: enlarged lymph nodes due to benign or malignant processes; aneurysms of the carotid vessels; branchial cysts; neurofibromas; and parotid tumors.

SUMMARY

The anatomic, clinical and roentgenographic features of tumors of the chemoreceptor system of the head and neck are reviewed.

The importance of tomography and appropriate angiography for accurate evaluation and early detection of these tumors is stressed.

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I wish to express my thanks to Dr. A. W. Templeton, Chairman of Department of Radiology, University of Kansas Medical Center, and Dr. S. Cronqvist, Chief of Neuroradiology Section, University Hospital, Lund, Sweden, for their valuable criticism in the preparation of this paper.

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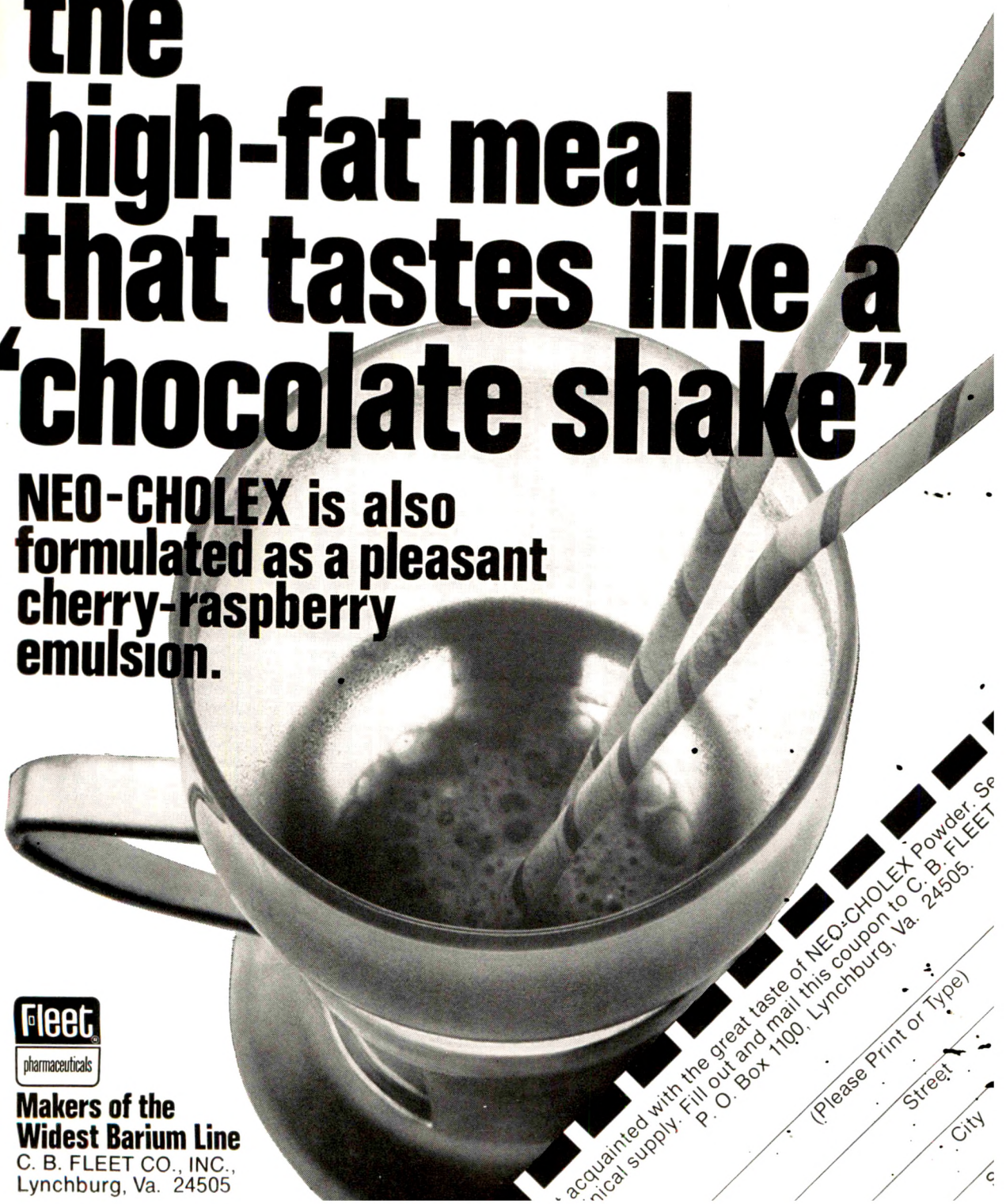
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THE aid of the radiologist is usually sought in the investigation of pain in the shoulder, arm and hand. A careful history may suggest a diagnosis which a plain roentgenogram confirms, but in many, no abnormality will be seen even on close scrutiny, and in a small group, more elegant investigations, such as cervical myelography or arteriography, will be considered This paper concerns the latter.

In addition to roentgenograms of the painful region, a chest roentgenogram is essential. This may reveal pathology in the cervicodorsal region, or even suggest that ischemic heart disease may be responsible for the limb pain.

The value of arteriography in selected patients is illustrated by the following cases.

REPORT OF CASES

Case 1. A 38 year old female accountant noticed coldness and color variations in her left hand, and sought advice after 1 year. The entire arm and forearm had ached towards the end of this time. On examination, a pulsatile swelling was present in the left supraclavicular fossa, the left radial pulse was absent, and the systolic pressure at the left elbow was 80 mm. Hg, blood pressure on right being 140/100 mm. Hg. Neck roentgenography revealed bilateral bony cervical ribs, shown in Figure 1. Operative removal of the left rib and cervical sympathectomy were succeeded by a return of the radial pulse and of warmth to the hand. During the ensuing 6 months the pain returned, and nonhealing ulcers developed in the tips of the thumb and middle finger (Fig. 2). Roentgenograms with soft tissue exposure, as in Figure 3, showed loss of bone density of the shaft of the distal phalanx, wasting of pulp tissue, and nail atrophy. Figure 4 is a left subclavian arteriogram, indicating atheromatous plaque formation in the second part of the artery, with multiple emboli in the distal axillary and brachial arteries, and complete obliteration of the brachial artery in the upper arm. Emboli are seen in the posterior circumflex humeral artery, and the origin of the costocervical trunk is narrowed by atheroma. Later operation confirmed these findings.

Case 11. A 15 year old schoolgirl endured recurrent migrainous right-sided headache for 1 year, and for 3 months had suffered pain in the right upper arm and forearm, with pallor and numbness of the right thumb, index and middle fingers. Two weeks previously an ulcer had developed at the tip of the thumb, and a history

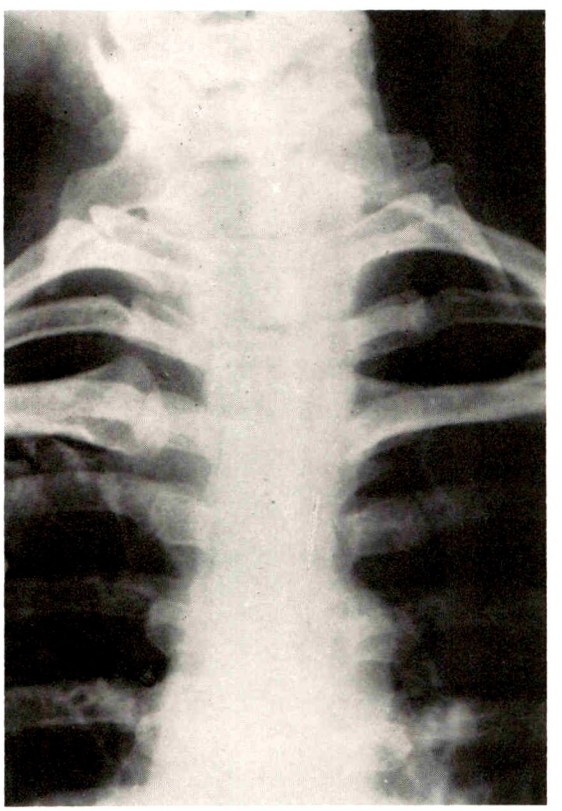


Fig. 1. Large bilateral ribs on C7.

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FIG. 2. Nonhealing ulcers on tips of thumb and middle finger.

of intermittent discolored patches in the hand was obtained. A sudden weakness of the left side of the body and transient loss of consciousness occurred on the day of admission. The examination revealed left hemiparesis, with absent pulse in the right arm below the subclavian artery which felt very pulsatile and gave a systolic bruit. Blood pressure was unrecordable in the right arm, but was 110/60 mm. Hg in the left. Figure 5 is a right carotid arteriogram demonstrating complete occlusion of the origin of the middle cerebral artery, presumably embolic. After 10 days of clinical improvement, arteriography, and later surgery, disclosed narrowing with thrombus formation of the right subclavian artery between the first rib and the lateral fibers of the scalenus anterior, with much post-stenotic arterial dilatation. A friable vegetation 1.25 cm.×0.5 cm. was removed from the artery, and a good neuro-



FIG. 3. Phalangeal osteoporosis, soft tissue and nail atrophy of left thumb.



FIG. 4. Left subclavian arteriogram. Narrowing of the 2nd part with thrombus formation. Multiple distal emboli to axillary and brachial arteries and branches, and complete block of brachial artery in upper arm. Narrowed origin of costocervical trunk.

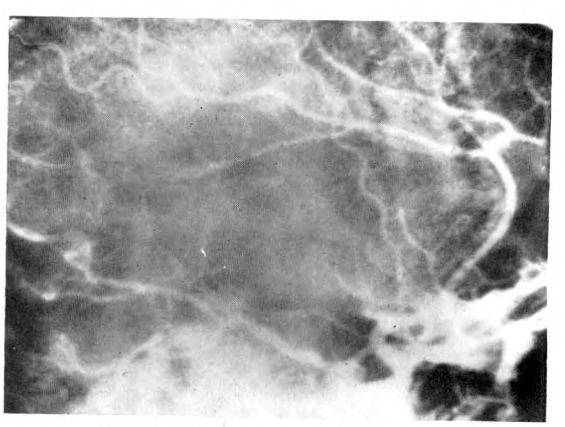
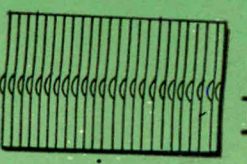
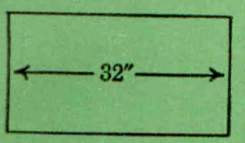
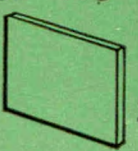
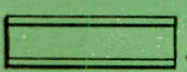
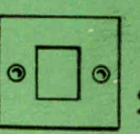


FIG. 5. Right carotid arteriogram. Late arterial roentgenogram showing blockage of entire middle cerebral artery, with good filling of anterior and posterior cerebral arteries and their branches.

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logic recovery occurred. A diagnosis of acute retrograde embolization to the right middle cerebral artery from a thrombus in the subclavian artery, and recurrent embolization to the small arteries of the hand causing early cutaneous necrosis, was made.

CAUSES AND RESULTING SYNDROMES

Arterial emboli of insidious onset traveled to the upper limbs of both patients.

A review of reports of emboli to the upper extremity reveals a variety of causes. These are:

(A) Association with cervical rib and variants.

(B) Atheromatous. Billig, Hallman and Cooley[1] suggest that the subclavian, axillary and brachial arteries account for 20 per cent of peripheral arterial emboli, most arising in the heart after myocardial infarction.

(C) Post-traumatic.
  (a) Contusion of limb, with thrombosis of artery, as in Figure 6.
  (b) Following transaxillary aortography.[1]
  (c) Arising from thrombus at the site of brachial arteriotomy used for cardiac catheterization.[9]

(D) Arising from the wall of an aneurysm, the latter complicating:
  (a) Penetrating wound.
  (b) Local degenerative arterial disease. Persaud[15] reports an 8 cm. diameter subclavian artery aneurysm due to cystic medionecrosis in a 22 year old female.
  (c) Syphilis, contiguous tuberculous lesions.[5,8]

(E) Mycotic. Emboli due to subacute bacterial endocarditis have been described in the brachial artery by Mentha,[13] and in the ulnar artery by Klein and Crowell.[11]

(F) Foreign body. Schloffer[19] instances a case of a metal embolus to the axillary artery following a bullet wound to the ankle.

Group A merits attention. Bramwell[2] first described neurovascular compression at the thoracic outlet in 1903. The community of disorders leading to constriction at the outlet are illustrated in Figure 7 and are as follows:

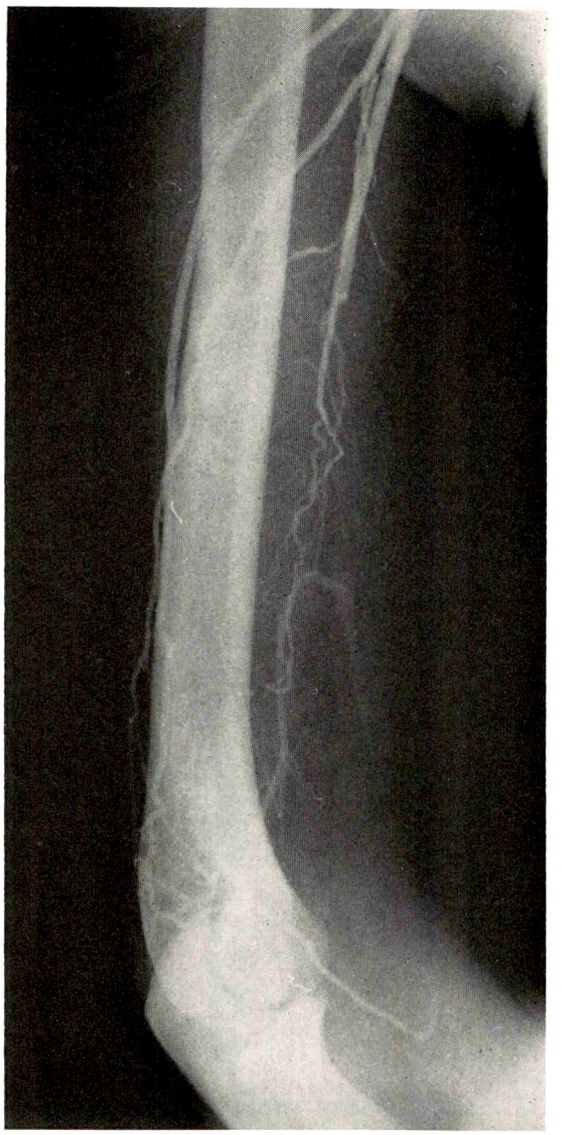


Fig. 6. Thrombosis of upper brachial artery in a 24 year old physician due to contusion of arm from car accident; a possible source of embolus.

(1) Cervical rib syndrome. This is the most frequent form and the easiest to diagnose. The incidence of the bony rib varies with the series[17,20] from 1 in 200 to 1 in 625. No sex difference in occurrence is noted, but compression is much

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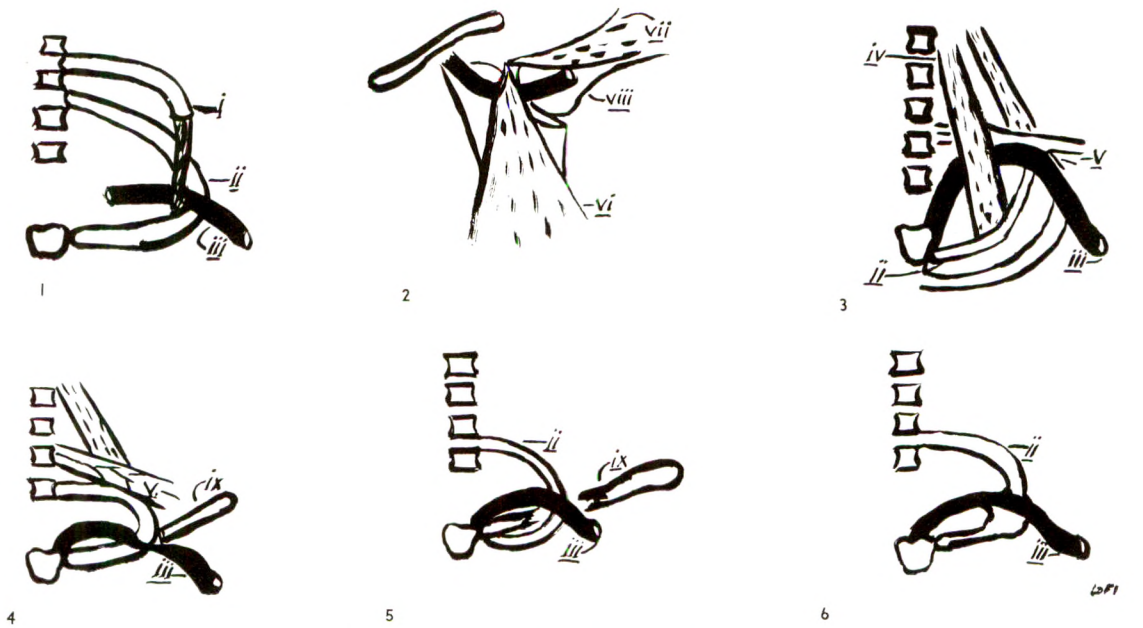


FIG. 7. Methods of neurovascular compression (on the left side). 1. cervical rib; 2. hyperabduction; 3 scalenus anterior; 4. costoclavicular; 5. fracture of clavicle; and 6. tumor of first rib.
i = cervical rib or band; ii = 1st rib; iii = subclavian artery; iv = scalenus anterior; v = brachial plexus; vi = pectoralis minor; vii = short head biceps; viii = humerus; and ix = clavicle.

more prevalent in females. In 11 out of 12 patients reported by Ferguson, Burford and Roper,[6] the compression was on the left, although right-handedness is more usual in the general population. Of the 12 with compression, 10 had bone anomalies, either cervical ribs or abnormal 1st or 2nd dorsal ribs. Occlusive disease of the subclavian artery unassociated with thoracic outlet compression was again noted to be left-sided in two-thirds of cases in another series.[16]

(2) The hyperabduction syndrome.
(3) The scalenus anterior syndrome.
(4) The costoclavicular syndrome. Rosenberg[18] indicates that man, with his erect posture, is anatomically predisposed for up to 35° backward clavicular movement at the sternoclavicular joint.
(5) Displaced fractures of clavicle, or excess callus formation of a healing fracture.
(6) Tumors of the first rib.

To these one may add:

(7) Scalenus medius or pectoralis minor muscle compression.

Clagett[3] describes the probable cure of symptoms in all of the above variegated group by first rib resection, but before any procedure, arteriography may be of great value, as the 2 case reports prove.

INDICATIONS FOR ARTERIOGRAPHY

The indications are: (a) pain disturbing work or sleep; (b) history of numbness, color change, or slowly healing wounds; (c) intermittent claudication; (d) deep tenderness in the supraclavicular fossa—a sign of brachial plexus irritation; (e) Neviaser[14] instances the value of a "bruit" in the supraclavicular fossa, or in the infraclavicular fossa in the costoclavicular syndrome; and (f) diminished brachial or radial pulse, or systolic blood pressure difference of 10 or more mm.Hg between the arms in a patient with symptoms.

In the 2 cases reported, the time from the onset of symptoms to established arterial

disease was only 1 year. Thus early arteriography is indicated in patients with a rapid course of compression. In a severely ischemic upper limb from any cause, there is a significant amputation rate.[9] A negative arteriogram is preferable to one showing positive changes, and it is better to show an early change such as post-stenotic dilatation, seen in Figure 8, than the irreversible changes as in Figure 4.

Billig, Hallman and Cooley[1] feel that unless the surgical result is questionable, postembolectomy arteriography is not as mandatory in the upper limb as in the lower, the radial and ulnar arteries being more superficial and so more accessible clinically. Gol, Patrick and McNeel[7] favor follow-up arteriography.

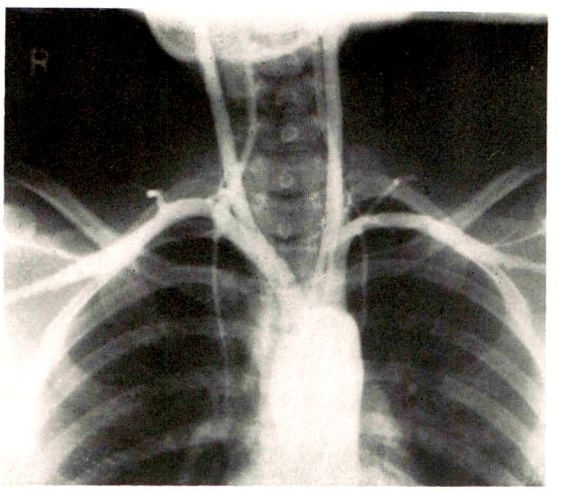


Fig. 8. Arch aortogram. Bilateral narrowing of second part of subclavian artery with post-stenotic dilatation. A 15 year old girl with C8 and T1 motor and sensory loss on the right. Note that the bony portion of the ribs on C7 stops well above the artery.

TECHNIQUE

A percutaneous transfemoral catheter is the most satisfactory approach for arteriography. To pass a catheter, albeit small, into an upper limb artery in this group of patients is always contraindicated.

In all cases, one should first determine the origin of the great vessels from the aortic arch. A young female with limb pain may have a Takayashu syndrome and an associated asymptomatic cervical rib. Even in the young, the great vessels may be tortuous, and oblique views necessary to demonstrate them fully, as in Figure 9, *A* and *B*. Older patients' symptoms may be the result of atherosclerotic disease of the arch vessels, one large series[4] indicating involvement of the left subclavian artery 1.6 times more often than the innominate and left common carotid arteries combined.

The catheter is guided into the subclavian artery on the symptomatic side, with its end just distal to the origin of the vertebral artery. An amount of 20 ml. hypaque 45 per cent is adequate to show all the arteries from shoulder to wrist. Manual injection is preferred, and large numbers of roentgenograms and very rapid filming is unnecessary. Depending upon symptoms and on the results of the arch study, both subclavian arteries may need catheterization. Although the symptoms may be bilateral, compression is not symmetric, but arteriography does correlate well with the side of greater involvement.[6]

DISCUSSION

The majority of patients with bone or soft tissue anomalies at the thoracic outlet never develop compression symptoms. Of those who do, many are young, otherwise well, and require early arteriography to diagnose the site and type of compression. In an ischemic upper limb, this causes even more pain than in a lower, and a general anesthetic is usually needed. The smallest diameter catheter practicable for the age and size of the patient is used via the femoral route. A precise arteriogram is mandatory. Lesions may be multiple, as in Case 1, and assessing the distal arteries in the upper limb is as important as seeing "the trifurcation" below the knee in the lower limb arteriogram. A further series with the arm hyperabducted should be undertaken, depending on the history, if the appearance is normal with the arm in the resting (adducted) position. An early study is favored, as in the cases described; the

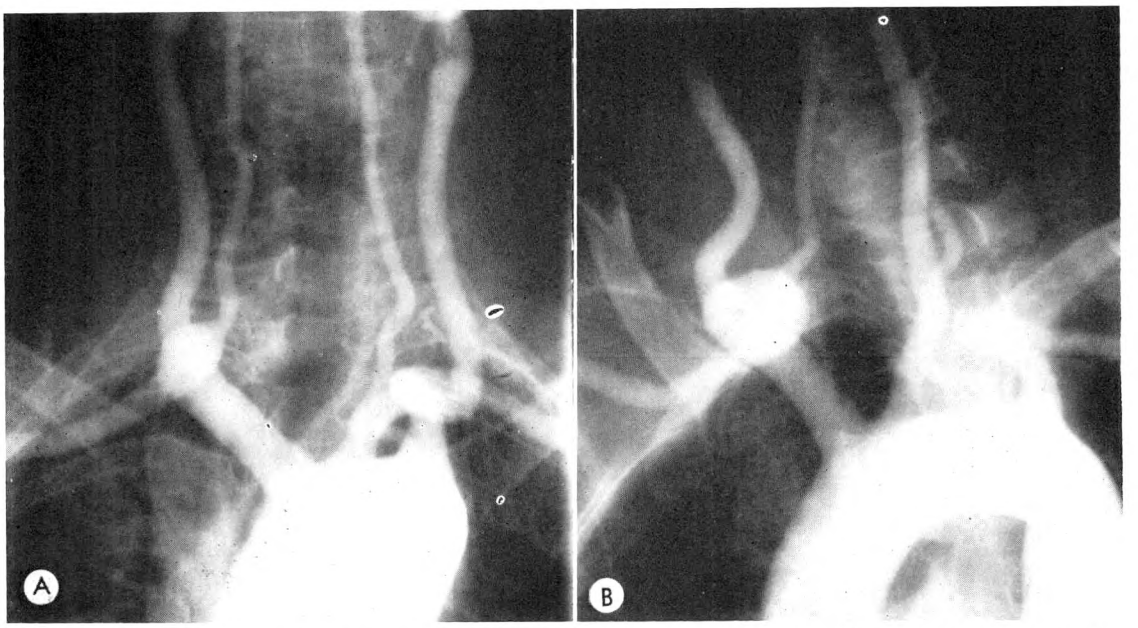


FIG. 9. (*A* and *B*) Oblique views were necessary because of vessel tortuosity to exclude possible arteriopathy of vessel origins in a 23 year old soldier.

patients had symptoms for not more than 1 year, yet had severe arterial changes.

Although arteriography is safe, its safety may always be increased. Jacobsson[10] reports the use of 6 per cent dextran 70 as an intravenous infusion before an arterial study to decrease the risk of thromboembolism by lowering platelet adhesiveness. To complement this, strict hemostasis should be ensured after the study to prevent thombosis and subsequent embolism developing in a patient who may already have one arterial lesion.

Lang[12] showed in a large comparative series, that in a group of patients with thoracic outlet syndromes, those having arteriography before surgery suffered a failure rate of 15 per cent, whereas in another group having surgery based on clinical diagnosis alone, the failure rate was 44 per cent. Rainer, Vigor and Newby[17] point to a further advantage of arteriography in that diagnosis of arterial compression at the outlet is much more definite than that of nerve compression. In a small proportion of patients with normal arteriography, subclavian venography may be necessary. Compression in "the costoclavicular syndrome" may be largely venous.[7]

CONCLUSION AND SUMMARY

Varieties of neurovascular compression at the thoracic outlet are described, and 2 cases of associated arterial emboli in young women are reported. In addition to recurrent emboli to the upper limbs, both had evidence of arteriopathy or embolism proximal to the compression.

Origins of emboli to the upper limb are listed, and the indications for, and technique of arteriography in thoracic outlet compression are suggested.

In view of inevitable sequelae of unrelieved arterial compression, an early study is essential. It is very safe.

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To Mr. A. Wheatley, J. A. Gillespie, and Dr. E. H. Allen for permission to publish the case details. Mr. R. Leng produced the photographs and Dr. Diana Fairclough drew the illustration.

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EPIDERMOID AND DERMOID SEQUESTRATION CYSTS*

By BERT LINCOLN PEAR, M.D.†
DENVER, COLORADO

EPIDERMOID and dermoid sequestration or implantation cysts frequently present a characteristic and even pathognomonic roentgenographic appearance. Epidermoid cysts are lined by well differentiated stratified squamous epithelium and enlarge by cellular proliferation and by desquamation of keratinized debris into the center of the cyst. Dermoid cysts are similar but include dermal structures such as hair follicles, sweat, apocrine and sebaceous glands, and may also contain hair and sebum. If either type of cyst communicates with the skin, a dermal sinus is formed.[8]

Sequestration and implantation cysts are neither related to the inflammatory cholesteatomas of the middle ear nor to the so-called dermoid cysts of the mediastinum, ovary, pineal or hypophyseal region which are true neoplasms and represent cystic teratomas.

TABLE I

EPIDERMOID AND DERMOID SEQUESTRATION CYSTS

I.	*Congenital*
	1. Closure of Neural Groove
	Pericranial
	Intradiploic
	Intracranial
	Intraspinal
	2. Epithelial Fusion Lines
	Head and Neck
	Anorectal
II.	*Acquired*
	1. Post-traumatic
	2. Iatrogenic
	Postsurgical
	Spinal Puncture
	3. Occlusive
	Hair Follicles
	Sebaceous Glands

CONGENITAL SEQUESTRATION CYSTS

Epidermoid and dermoid sequestration cysts can be either congenital or acquired. The congenital cysts are related to implantation of ectoderm—with or without dermal elements—at time of closure of the neural

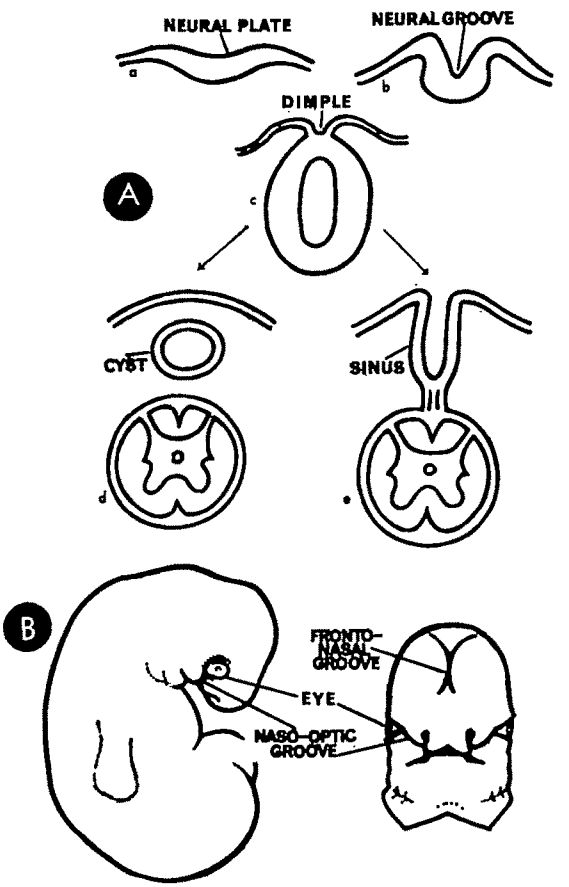


FIG. 1. Hypothetical illustration of development of congenital epidermoid and dermoid sequestration cysts and sinuses during embryonic closure of the neural groove (*A*), and also during coalescence of other epithelial fusion lines especially in the head and neck (*B*).

* Presented at the Seventieth Annual Meeting of the American Roentgen Ray Society, Washington, D. C., September 30–October 3, 1969.

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groove or of coalescence of other epithelial fusion lines (Fig. 1, *A* and *B*). Cysts related to closure of the neural groove, which occurs between the third to fifth week of embryonic life, present in median or paramedian locations in approximation to the central nervous system and spinal canal. Those in the pericranium are undetectable roentgenographically, but the intradiploic cysts result in sharply marginated, lytic defects with scalloped sclerotized borders (Fig. 2). As the cysts enlarge, the diploic space is ballooned and the inner or outer tables eroded and even disrupted. Infection can obliterate the sharp cortical margination and meningitis is always a potential hazard. Intradiploic cysts can reach huge size and extend intracranially to displace the brain (Fig. 3, *A* and *B*).

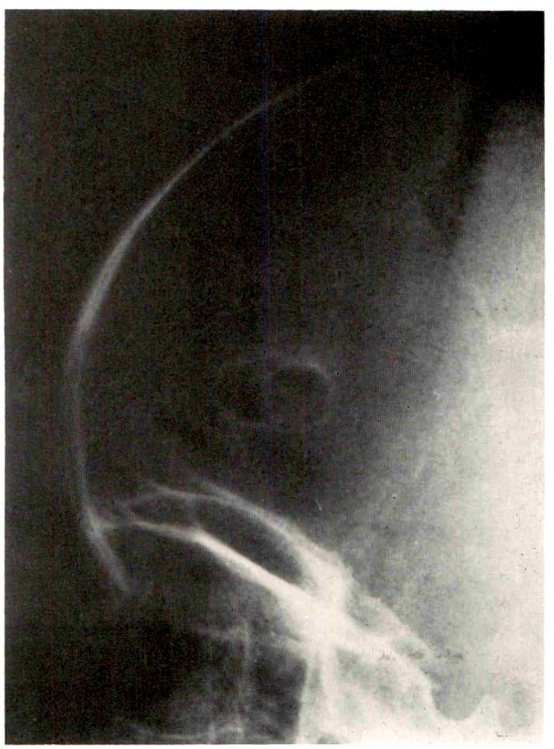


Fig. 2. Within the diploic space, as in this 17 month old infant, sequestration cysts typically have scalloped sclerotic margins. Most cysts in this location are apparently congenital, but experimental evidence indicates that some may be posttraumatic or postsurgical.[12]

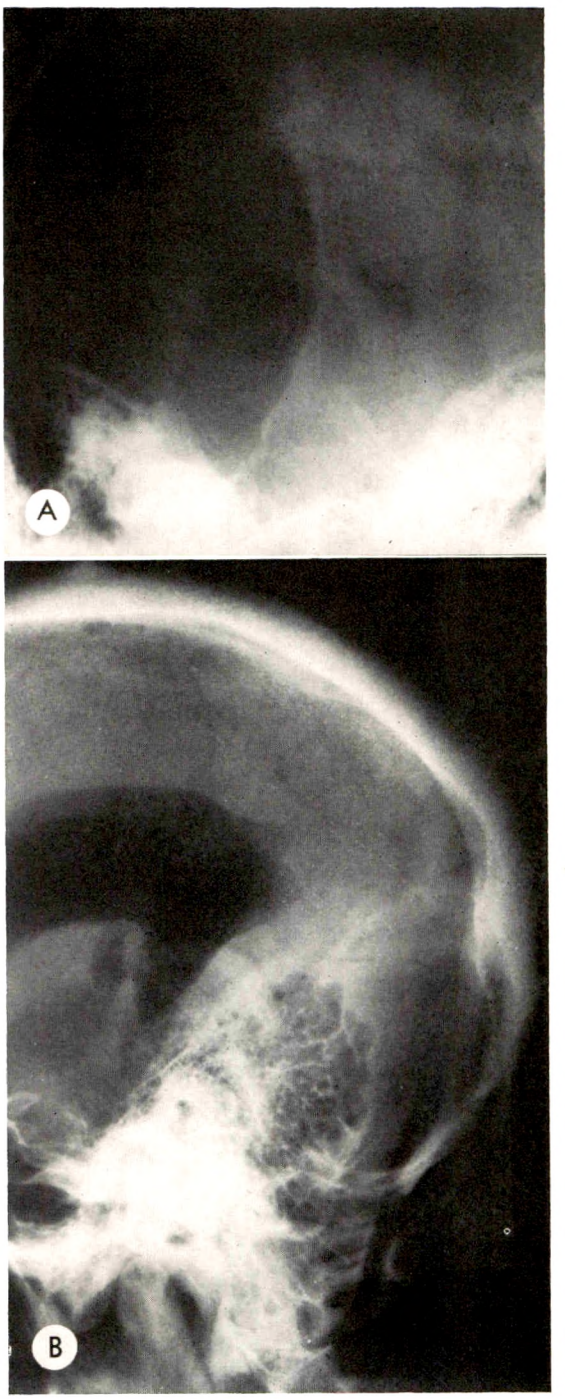


Fig. 3. (*A* and *B*) Intradiploic cysts may be quite large and extend subdurally within the cranial cavity. In this 16 year old boy an extradural dermoid cyst acted as a posterior fossa mass kinking the acqueduct and causing partial ventricular obstruction.

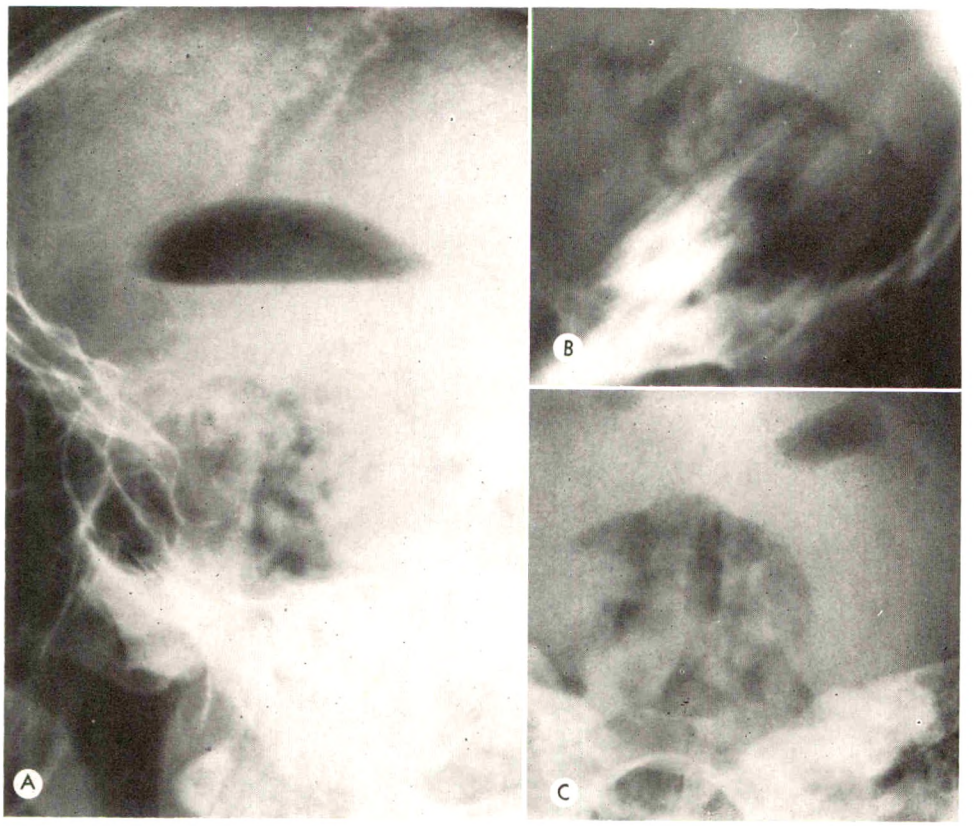


FIG. 4. Within the subarachnoid space or ventricles epidermoid cysts can present a pathognomonic appearance at pneumography. (*A*) A 52 year old man with lobulated parasellar and subtemporal epidermoid cyst whose wall had ruptured allowing gas to extend between the lamellated layers of keratin. (*B* and *C*) Epidermoid cyst within the 4th ventricle in a 34 year old woman. The wall of this cyst was incomplete and gas dissected between irregular whorls of keratinized debris.

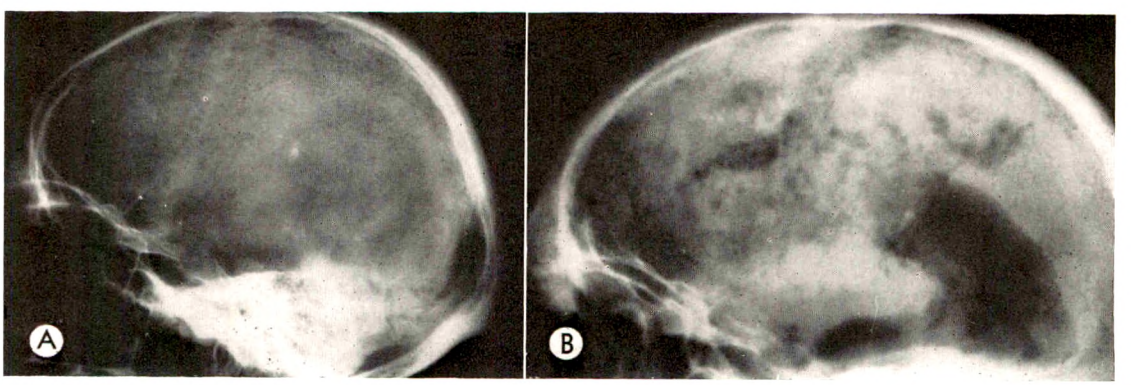


FIG. 5. (*A* and *B*) Occasionally the wall of an epidermoid or dermoid cyst may calcify. This epidermoid cyst in a 56 year old man was located in the longitudinal fissure and caused pressure erosion of the corpus callosum.



Fig. 6. Congenital epidermoid cyst in a 29 year old man presenting as an intramedullary tumor with tethering of the spinal cord at the T12-L1 level. There was a dimple and a hairy spot overlying the cyst. Many congenital intraspinal sequestration cysts have associated dysrhaphic defects and dermal sinuses. The latter are a frequent cause of meningitis.

Within the calvarium epidermoids predominate over dermoids and occur preferentially in the cerebellopontine angle, chiasmatic region, within the longitudinal fissure, corpus callosum, around the quadrigemminal plate, within the ventricles, and in the midline of the cerebellum.[14] Dermoids are more commonly located around the pituitary and pons.

When epidermoids are located within the subarachnoid space or ventricles, a virtually pathognomonic appearance is seen at pneumography if the cyst wall is deficient or if it ruptures allowing the gas to extend into the crevices between the lamellated layers of desquamated keratin (Fig. 4, *A-C*). Rarely the cyst wall may calcify (Fig. 5, *A* and *B*).

INTRASPINAL SEQUESTRATION CYSTS

Congenital intraspinal epidermoid and dermoid cysts are uncommon and account for less than 1 per cent of all spinal cord masses. The congenital cysts may be intramedullary, intradural or extradural and many communicate with the skin through an associated dermal sinus (Fig. 6). Other accompanying dysrhaphic developmental defects are frequently present, such as spina bifida, hemivertebra, malformed vertebra, diastematomyelia and rudimentary duplication of the spinal cord. Tethering of



Fig. 7. The superior lateral margin of the orbit is a common site for dermoid cysts related to implantation during embryonic fusion of the naso-optic furrow. This 23 year old woman had unilateral exophthalmus.

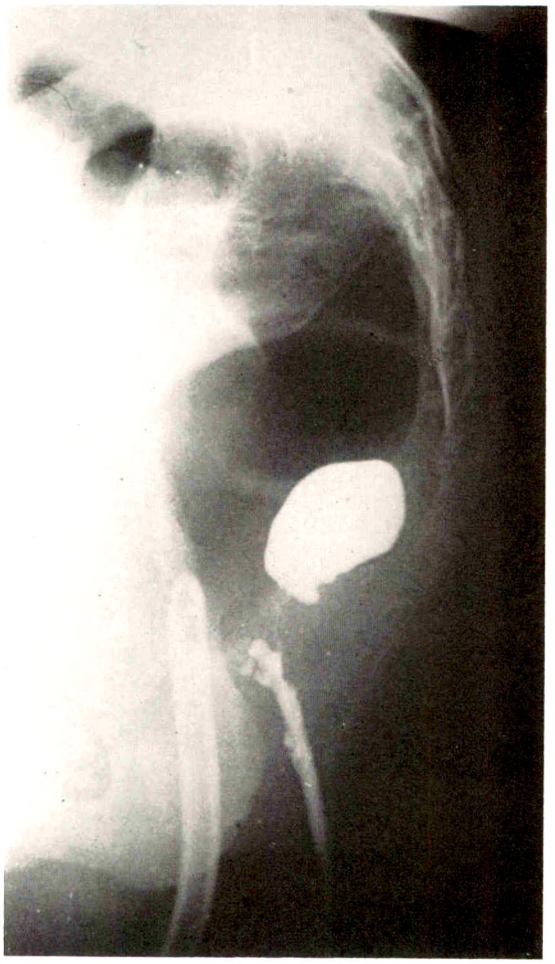


FIG. 8. Presacral epidermoid cysts apparently arise from vestigial epithelium which is sequestrated during the formation of the anal canal. This cyst is demonstrated by sinography. The large tube is within the rectum and the thinner tube in the sinus tract. (From Hanley, P. H. in Turell, R.: Diseases of the Colon and Anorectum. Volume II. W. B. Saunders Company, Philadelphia, 1959.)

the cord can complicate cysts and dermal sinuses in the region of the conus medullaris.[13]

Intraspinal cysts can cause expansion of the spinal canal resulting in posterior scalloping of the vertebral bodies, erosion and flattening of the pedicles and increase in the interpedicular distance.

CLOSURE LINE CYSTS

Congenital sequestration cysts, particularly dermoids, are also found at sites of embryonic epithelial closure lines in the head and neck and anorectal region.[4,5] Dermoids are therefore found at the junction of the maxilla and orbit at the site of the naso-optic furrow where they present as erosive, marginated lacunae or fossae destroying the bone of the orbit, generally in its superior lateral margin (Fig. 7). In this location they are an infrequent cause of uni-



FIG. 9. Most post-traumatic epidermoid sequestration cysts are within the phalanges and are marginated lytic osseous defects. This 11 year old girl had crushed her thumb in a folding chair and sustained soft tissue lacerations.

lateral exophthalmus. A similar origin is responsible for those occurring at the inner angle of the orbit, external angular process of the frontal bone, nasofrontal sulcus, and orbito-ethmoidal and otic locations. Presacral epidermoid cysts also occur and arise from sequestration of vestigial epithelium at time of canalization of the anus[4] (Fig. 8).

ACQUIRED SEQUESTRATION CYSTS

Sequestration cysts may also be acquired. The etiology is most commonly post-traumatic and many are occupationally related due to implantation of fragments of epidermis in the fingers or palms, particularly of tailors, shoemakers and carpenters. Not infrequently, however, the trauma is of the crushing type, as in the closing of a door[5] (Fig. 9). Characteristically the cysts are located within the distal phalanges and show marginated, erosive osseous defects quite like the intradiploic tumors.

Insect bites have also been incriminated in causation of implantation cysts.

Implantation may occur at surgery and epidermoid cysts are common in episiotomy scars. Recently Pear and Wolff[10] reported a case of epidermoid cyst of the cecum and found another in the literature[10] (Fig. 10). Both had followed appendectomy at which time no cecal mass had been noted suggesting that epidermis was implanted at the time of the initial operation.

IATROGENIC INTRASPINAL CYSTS

In 1956, Choremis and his colleagues[3] reported 6 cases of intraspinal epidermoid tumors in children successfully treated for tuberculous meningitis. They postulated that repeated lumbar punctures, which had been performed without stylets, had implanted epithelial cells which in time formed one or more tumors. Since then similar reports have emphasized the iatrogenic nature of epidermoid cysts appearing $1\frac{1}{2}$ to 23 years following lumbar puncture for treatment of meningitis, for spinal anesthesia and for diskography.[1,2,7,9]

Van Gilder and Schwartz[12] have experimentally confirmed the creation of such cysts by implanting fragments of minced skin into the central nervous system of young rats. In 1 of their 18 rats, both an intradiploic epidermoid and dermoid tumor were formed suggesting that human cysts in that location may be acquired as the result of surgical or traumatic epidermal implantation as well as of congenital origin.

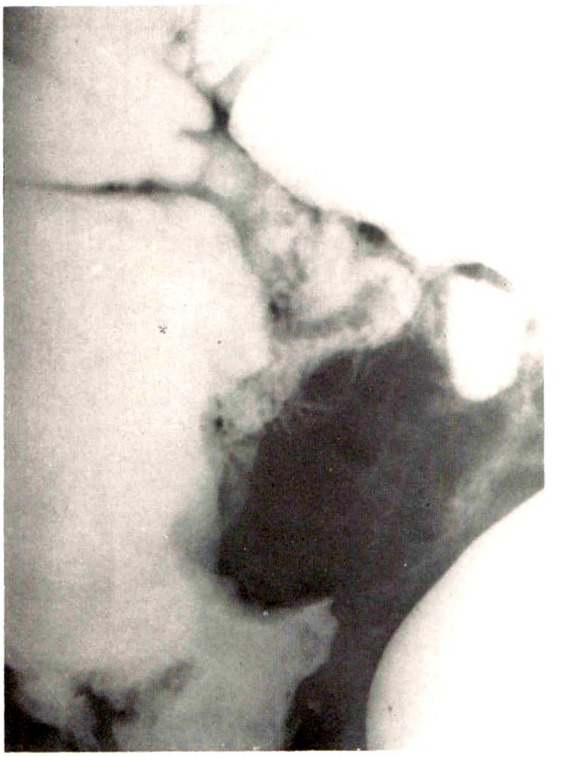


Fig. 10. Epidermoid cyst of the cecum in a 71 year old man following appendectomy. This is felt to represent a postsurgical implantation cyst since no cecal tumor was present at time of the initial operation. (Reproduced with permission from *J.A.M.A.*, 1969, *207*, 1516.)

We have reported 3 cases of iatrogenic intraspinal epidermoid sequestration cysts, including 1 case seen with Boyd in which an intraspinal epidermoid cyst appeared 3 years following a normal myelogram and $2\frac{1}{2}$ years after diskography, the latter performed without a stylet[9] (Fig. 11, *A–C*).

Unlike the congenital cysts, no plain roentgenographic findings have been reported with the acquired intraspinal cysts. The roentgen manifestations have been

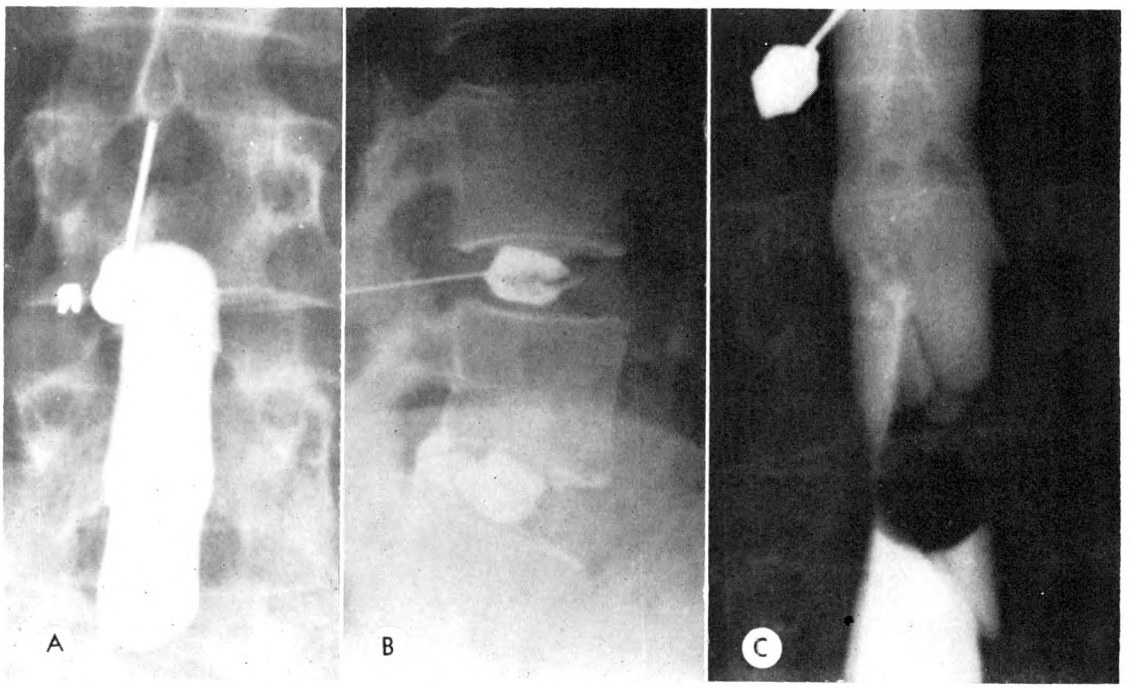


FIG. 11. Iatrogenic epidermoid sequestration cysts occur secondary to implantation of epidermis at the time of lumbar puncture. (*A*) Normal myelogram in a 49 year old woman. (*B*) Six months later diskography was performed without a stylet. (*C*) Repeat myelogram 2½ years after diskography demonstrates an oval intradural mass at previously normal L4–5 interspace. (Reproduced with permission from *Radiology*, 1969, *99*, 251.)

limited to the presence at myelography of one or more rounded or oval tumors within the subarachnoid space. The cysts are generally located posteriorly at a lumbar interspace and show a varying degree of adherence to the nerve roots and arachnoid. Occasionally amorphous debris, probably keratin, has been identified within the caudal sac.[2,6] An acquired cyst has also been found in an epidural location, encircling the cord and presenting as a nonspecific epidural mass lesion.[9]

OCCLUSIVE CYSTS

Cysts, also called epidermoid or sebaceous cysts, can occur within the skin and subcutaneous tissues following plugging of the orifice of a hair follicle or sebaceous gland. An interesting variant of this type of inclusion cyst is the so-called benign ossifying epithelioma of skin (calcified epithelioma of Malherbe) whose origin from degenerated epidermal inclusion cyst is probable.[8] These may occasionally be recognized roentgenographically by faint ossific stippling especially when this is within the scalp (Fig. 12).

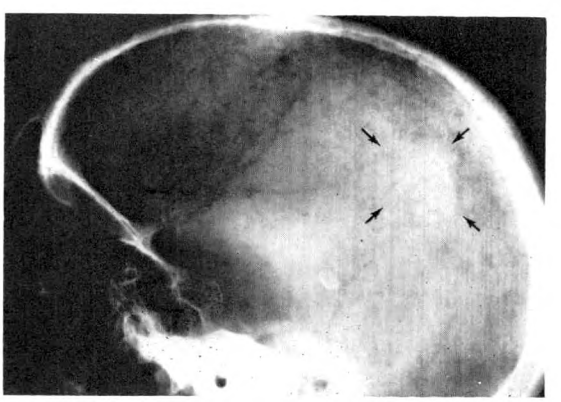


FIG. 12. Benign ossifying epithelioma of the scalp in a 79 year old woman. This was palpable as a hard subcutaneous mass. Faint ossific stippling is visualized on the roentgenogram of the skull.

SUMMARY

Epidermoid and dermoid sequestration cysts are a form of heterotopia due to displaced or implanted ectoderm. They are unrelated to benign cystic teratomas. Congenital cysts occur following inclusion of epidermis at time of embryonic closure of the neural groove or of epithelial fusion lines in the head and neck and about the anus. If only epidermis is included, the cyst is an epidermoid. A dermoid cyst is formed if dermal elements are also present. If either communicates with the skin, a dermal sinus is created.

Acquired cysts may be post-traumatic or iatrogenic. The former generally occur in the phalanges while iatrogenic cysts follow surgery or spinal puncture.

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SEVERE REFLEX ALGODYSTROPHY (SUDECK'S ATROPHY) AS A COMPLICATION OF MYELOGRAPHY: REPORT OF TWO CASES*

By LUIS B. MORETTIN, M.D.,† *and* McCLURE WILSON, M.D.‡
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PROMPTED by our experience with the 2 patients described below, we reviewed the literature and failed to find any previous report of the development of Sudeck's atrophy syndrome following the performance of myelography.

REPORT OF CASES

CASE I. B.M., U.H.#94115-P., 21 year old white female was in excellent health up until 1965 when, at a judo session, she was thrown flat on her back and began experiencing severe low back pain. The pain was constant in nature and location, and radiated down the right leg. Occasionally, mild and intermittent pain extended down the left leg also. The symptoms increased with activity but were not relieved with rest. At another hospital the diagnosis of herniated intervertebral disk was made and a laminectomy performed. No lesion was found. Because of persistent severe symptoms a second operation was performed 1 month later at which time some disk material was removed from the disk between L_4 and L_5 and between L_5 and S_1. The procedure afforded no significant relief.

In September, 1968, the patient was admitted to our institution at which time it was felt that the symptoms were due to arachnoiditis. Myelography was performed which was normal. After removal of the pantopaque, 80 mg. of methyl prednisolone acetate (depo-medrol) was injected in the subarachnoid space. A few seconds after the completion of the procedure the patient experienced sudden excruciating back pain radiating into the left leg. Edema, redness, and blotchy cyanosis of the foot and ankle appeared 1 week later. There was considerable limitation to passive motion of the ankle. Because of severe pain in the left foot she was unable to walk. Roentgenographic changes of acute spotty demineralization were noted on roentgenograms made approximately 8 weeks later (Fig. 1, *A* and *B*; and 2, *A* and *B*). Because of the persistence of symptoms with conservative treatment a lumbar sympathectomy was performed 5 months later. Three weeks after surgery a slight improvement in the symptoms was apparent. Two months later the soft tissue dystrophic changes had improved considerably. The roentgenographic appearance of the bones had remained essentially unchanged although bone pain was much diminished.

CASE II. E.B., U.H.#101012-P., underwent an extensive work-up at another institution for the complaint of persistent and severe headaches in September of 1967. At that time myelography was performed, presumably as a part of such a work-up. When seen by us, the patient clearly stated that immediately after the introduction of the needle in the lumbar subarachnoid space she experienced a sensation of intense warmth and slight pain in the left leg. The symptoms were transient and the procedure was apparently carried out without difficulty. The myelogram was reported as being normal. After completion of the myelogram the pain became progressively worse, extending from the left side of her back into the posterior aspect of the left leg and toes. There was mild hyperesthesia. Trophic changes consisting of redness, blotchy cyanosis and edema appeared in the left leg. Two weeks later the pain in the left foot intensified and became almost unbearable, leading to considerable limitation of motion and inability to walk. In August of 1968, a left lumbar sympathectomy was performed for the purpose of palliation. The symptoms improved only slightly and recurred with full intensity a few weeks after. She was readmitted again in January, 1969. By that time the soft tissue trophic changes involving the left leg had disappeared. The roent-

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genographic changes were not very striking, consisting only of diffuse osteoporosis of the left foot which was apparent when compared with the opposite side. There was no evidence of spotty demineralization. Because of intractable pain a chordotomy was performed. Following chordotomy the symptoms improved and the bone changes remained stationary. Functional improvement was noticeable.

DISCUSSION

Several complications and untoward reactions to myelography have been discussed in the standard textbooks[4,12] and have been the subject of numerous reports. The following are described:

(1) Subjective symptoms and signs common to lumbar puncture (severe headaches, nuchal rigidity, nausea, vomiting, transient hypotension, etc.).[3,12]

(2) Occasionally, there is aggravation of the symptomatology that motivated the examination such as low back pain or sciatica. Postmyelographic back pain of a different type and location is occasionally encountered.[10]

(3) Infection may accidentally be introduced by a breech of aseptic technique.[12]



Fig. 1. Case 1. (*A*) The right foot is normal. (*B*) The tarsal and metatarsal bones of the left foot reveal marked diffuse and spotty demineralization.



Fig. 2. Case 1. (*A*) Lateral roentgenogram illustrates the extent of the changes in the tarsal bones of the left foot. (*B*) The right foot is normal.

(4) Aseptic arachnoiditis occurs and may be the consequence of varying degree of reaction to the contrast substance.[3,11]

(5) Inflammatory reactions affecting the intracranial meninges may lead to obliteration of the cisterns with resultant hydrocephalus. Multiple cranial nerve paralysis has been reported to occur under these circumstances.[5,8]

(6) Rarely, extremely severe and possibly fatal sensitivity reactions may develop.[1,8,13]

(7) Venous intravasation with consequent pulmonary microembolism may occur.[6]

(8) Postmyelographic coccygodynia may appear and is thought to be secondary to trauma or chemical irritation of the sacral nerve roots.[9]

(9) Cardiac arrest can happen, presumably as a consequence of the hemodynamic derangement produced by tilting of the table.[2]

(10) Thirty-seven cases of intraspinal epidermoids have been reported following implantation of epidermal tissue when lumbar puncture was performed.[7]

(11) Puncture of the annulus fibrosus may lead to herniation of disk material.[12]

The severe symptoms and the incapacitating disability occurring in our 2 cases unquestionably constitute a bona fide complication. There is little known about the mechanism of production as post-traumatic algodystrophy (which, in orthopedic practice, is a recognized complication of fractures, particularly of the ankle and wrist). Occasionally the severity of the symptoms are out of proportion to the injury and may follow trivial trauma. In our cases the mechanism of production of the symptoms and the marked trophic changes of the bone and soft tissues is obscure. It is possible that direct trauma to the nerve roots or the chemical insult produced by the contrast substance (pantopaque) or the methyl prednisolone acetate (depo-medrol) triggered the vasomotor reflexes which are sometimes incriminated in the production of reflex algodystrophy. In our first case the appearance of symptoms following the removal of the pantopaque and the injection of the depo-medrol exonerates the contrast substance as the causative agent. In the second case the clear onset of symptoms immediately following the lumbar puncture and preceding the injection of pantopaque suggests that traumatic injury of a nerve root may have been the event that triggered the development of the reflex algodystrophy.

SUMMARY

Two patients are reported in whom the clinical and roentgenographic findings of reflex algodystrophy developed following myelography.

A previous report of this complication is not known to us.

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THE HISTIOCYTE IN RADIOLOGY*

WITH CASE REPORTS OF RETROPERITONEAL XANTHOGRANULOMA AND MALIGNANT FIBROUS XANTHOMA

By BERT LINCOLN PEAR†
DENVER, COLORADO

TUMORS and tumefactive proliferations of histiocytes comprise a confusing array of neoplastic, metabolic, reactive and inflammatory lesions which defy simple classification. Different descriptive terms and a varied nomenclature have been applied to similar lesions depending upon the bias of the observer.

The histiocyte is a ubiquitous cell derived from the primitive reticulum cell with which it is intimately associated in the reticuloendothelial or reticulohistiocytic system (Table 1). Unlike its precursor, the histiocyte is a differentiated cell with specific functions including phagocytosis. It is also known as a macrophage, resting-wandering cell or tissue monocyte.

In loose connective tissue the histiocyte may be nearly as abundant as the fibroblast and, with stimulation, is capable of differentiation into a fibroblast. Within tissue the histiocyte is a sessile, fusiform cell, but in inflammatory processes it becomes ameboid and assumes a variable or rounded shape.

Morphologically and functionally similar cells line the sinuses of lymph nodes, spleen and bone marrow and are probably modified histiocytes.[11] When the monocyte of the blood migrates from the circulation it becomes indistinguishable from the histiocyte. When closely packed, it may become the epithelioid cell of tuberculous or other granulomas and by fusion or amitotic division can form Langhans or foreign body giant cells.

Because of its phagocytic ability, the histiocytic macrophage is important in the maintenance of the local defence of the body against bacterial and protozoal invasion, but it also ingests extravasated erythrocytes, cellular debris, inert foreign matter and both endogenous and exogenous lipids. When engorged with lipid, the histiocyte is called a foam cell or xanthoma cell.

RETICULOHISTIOCYTOSES

Systemic proliferations of histiocytes have been described as reticuloses, histiocytoses, reticuloendothelioses and probably more accurately as reticulohistiocytoses.[11]

The process may be inflammatory as in some bacterial, protozoal or fungal infections or metabolic as in hyperlipemic states in which the histiocyte passively stores lipid. Examples of the latter are the xanthoma diabeticorum and the multiple xanthomatous giant cell tumors of familial hypercholesterolemia.

When the process is systemic and the cells are morphologically neoplastic, the disease is termed malignant histiocytosis. The visceral form resembles leukemia except that enlargement of the liver, lymph nodes and spleen is more pronounced.

When systemic, but not neoplastic, the terms differentiated progressive histiocytosis (Rappaport), idiopathic histiocytosis or histiocytosis-X (Lichtenstein) are used.

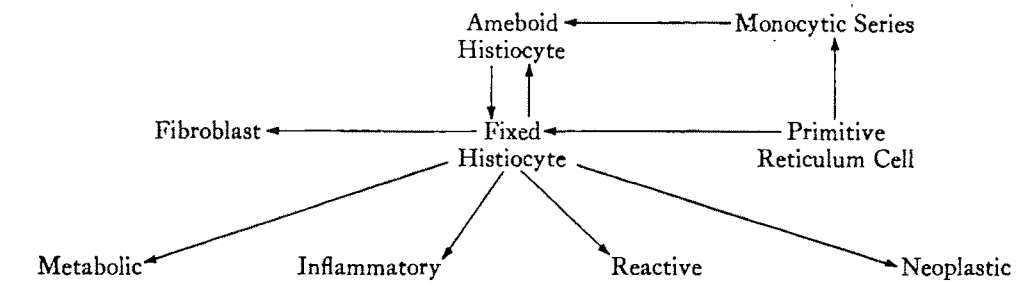
The concepts of earlier European and American investigators were integrated by Lichtenstein in 1953,[7] when he suggested that Letterer-Siwe disease, Hand-Schüller-Christian disease and eosinophilic granuloma represent the acute (and subacute), the chronic and the localized variation, respectively, of a single nosologic entity in which the proliferating cells are differenti-

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TABLE I

THE UBIQUITOUS HISTIOCYTE



Metabolic	Inflammatory	Reactive	Neoplastic
Xanthoma diabeticorum	Histiocytosis-X (?viral)		Systemic malignant histiocytosis
Giant cell tumors of familial hypercholesterolemia	Specific granulomas	Histiocytoma →	Histiocytoma
	Endogenous lipid pneumonia	Xanthoma →	Xanthoma
	Exogenous lipid pneumonia	Xanthogranuloma →	Xanthogranuloma
Xanthelasma	Xanthogranulomatous pyelonephritis	Fibrous xanthoma →	Fibrous xanthoma (benign and malignant)
		Xanthofibroma →	Xanthofibroma
Gaucher's disease	Malacoplakia	Dermatofibroma protuberans	Dermatofibroma protuberans
Niemann-Pick disease	Xanthogranuloma	Sclerosing hemangioma	
		Pigmented villonodular synovitis and variants including giant cell tumor of tendon →	Pigmented villonodular synovitis and variants including giant cell tumor of tendon

ating histiocytes displaying a varying degree of phagocytic activity.

Histiocytic proliferations may also be localized and these focal collections may be granulomatous, reactive or neoplastic. Simple examples of granulomas containing histiocytes are tuberculomas, histoplasmomas, Boeck's sarcoid, regional ileocolitis, among others.

UNUSUAL GRANULOMAS CONTAINING HISTIOCYTES

More interesting roentgenographically are those granulomatous lesions of lung and kidney known variously as chronic interstitial pneumonitis with lipid deposition, cholesterol pneumonitis, endogenous lipid pneumonia and foam cell pneumonia in the lung; and as xanthogranulomatous pyelonephritis in the kidney.

Chronic interstitial pneumonitis with lipid deposition or endogenous lipid pneumonia—colloquially called cholesterol pneumonitis—often presents as a mass lesion which may simulate neoplasm. Roentgenographically a variable portion of lung shows consolidation or atelectasis. When only a portion of a lobe is affected the area of infiltration frequently fans out from the hilus to assume a pyramidal shape with the base at a pleural surface[16] (Fig. 1,*A* and *B*). When the bronchi are patent, bronchogenic carcinoma, but not other forms of neoplasm, can be excluded.

The gross cut section shows an intense yellow color in early cases but this fades and is replaced by gray fibrous tissue flecked with yellow dots in older cases.

TABLE II

UNUSUAL GRANULOMAS CONTAINING HISTIOCYTES

Endogenous lipid pneuomonia
Xanthogranulomatous pyelonephritis
Malacoplakia
Xanthogranuloma

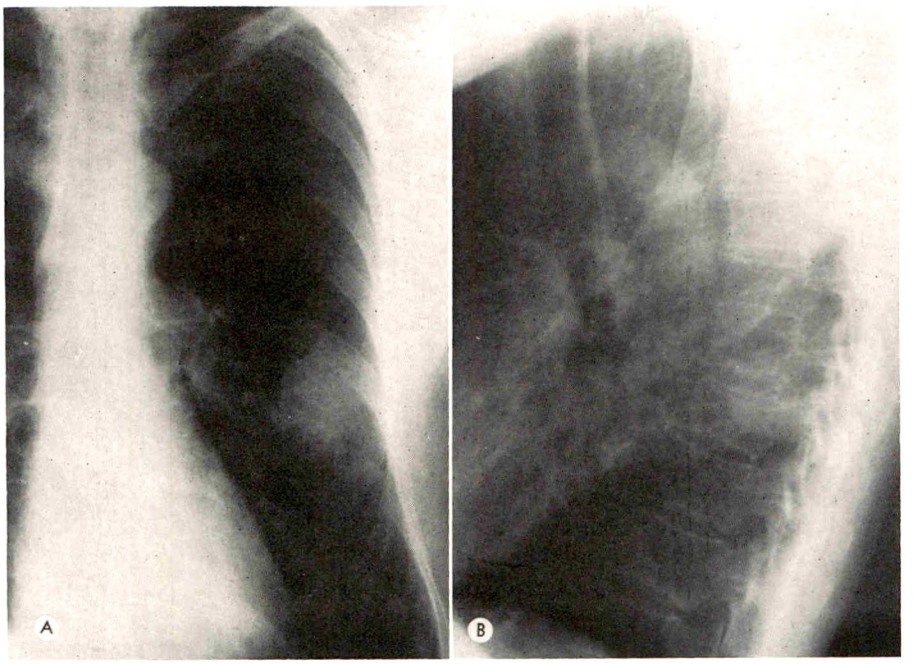


Fig. 1. (*A* and *B*) Endogenous lipid pneumonia in 53 year old man with subsegmental area of consolidation with base bordering on a pleural surface. There was no endobronchial obstruction and the lesion appeared to follow an unresolved pneumonia.

Microscopically the histologic changes center about foam-filled macrophages containing cholesterol and cholesterol esters.[16]

The original 10 cases presented by Waddell and colleagues in 1949,[16] were selected because of lack of coexisting endobronchial obstruction, but since then it has been shown that this process may exist alone, behind bronchial obstruction, or in association with other inflammatory lesions, including bronchiectasis, and there is no reliable way of distinguishing it from neoplasm with which it may indeed coexist. The etiology is nonspecific, but appears to be related to deposition of neutral fat, phospholipid and cholesterol in areas of disintegrating tissue or in inflammatory processes on the basis of pH changes which locally alter the colloidal state of lipids and proteins. The cholesterol and fat are then phagocytized by macrophages.[15] Many cases called organizing pneumonia, postinflammatory tumor as well as pulmonary xanthoma and xanthofibroma appear to be different stages of this same process.[14]

Mineral oil pneumonia, cod-liver oil pneumonia, paraffinoma and other forms of exogenous lipid pneumonia are etiologically and clinically unrelated to endogenous lipid pneumonia but also show lipid phagocytosis by histiocytic macrophages. Histologic differentiation is possible.

Xanthogranulomatous Pyelonephritis

A proliferative inflammatory reaction also occurs in the kidney and is characterized by sheets of clear foamy histiocytes heavily laden with lipid which gives the gross specimen a yellow color. Lymphocytes and plasma cells are also present. The lesion has been variously termed staphylomycosis, foam cell granuloma, pyelonephritis xanthomatosa and, more recently, xanthogranulomatous pyelonephritis.[2,3,12] The kidney is normal in size or larger than usual. When there is replacement of renal parenchyma by the granuloma, the angiographic appearance may be that of an avascular mass and the process has been further described as tumefactive.



FIG. 2. Xanthogranulomatous pyelonephritis in a 49 year old woman with obstructing staghorn calculus in nonfunctioning kidney of normal size. Histologically the interstitial tissue was infiltrated with sheets of histiocytes with foamy cytoplasm and large nuclei.

Almost invariably there is nonfunction of the kidney and the majority—probably about 70 per cent—have obstructing calculi (Fig. 2). Many recent cases have been accompanied by *Proteus* infection but this is not invariable. The common denominator generally appears to be prolonged suppuration, frequently associated with obstruction, as in the analogous lesion of lung.[12]

MALACOPLAKIA

Malacoplakia is an even more obscure lesion of the urinary tract. It has been confused with xanthogranulomatous pyelonephritis by some authors but appears to be an unrelated entity seen most commonly in the urinary bladder, but also in the ureter and renal pelvis. Extraurinary sites have been described and reports include its occurence in the prostate, testes, epididymis, colon, retroperitoneum and even within the stomach.[4]

The lesion is a histiocytic granuloma consisting of plaques or nodules which grossly and roentgenographically may resemble cystitis or ureteritis cystica or the diffuse form of xanthogranulomatous pyelonephritis.

Microscopically the granuloma consists of histiocytes and other inflammatory cells. The former contain phagocytized bacteria, erythrocytes and granular cytoplasmic debris, but the specific finding is that of inclusion of peculiar calcospherites known as Michaelis-Gutman bodies. There is some evidence to suggest that coliform bacilli may be the causative agent.

HISTIOCYTOMA, FIBROUS XANTHOMA AND XANTHOGRANULOMA

Tumors of skin, subcutaneous tissues, gum, lung, mediastinum, retroperitoneum and mesentery also occur and have received a confusing variety of names. In general the nomenclature varies with the location of the tumor, with the altered manifestations of the histiocyte, and with the pathologist's concept of its pathogenesis. Opinions vary as to whether these are neoplastic or reactions to trauma.[9,14]

PIGMENTED VILLONODULAR SYNOVITIS

Pigmented villonodular synovitis, bursitis and tenosynovitis also represent a form of histiocytic proliferation. Experimentally a similar lesion has been produced in dogs following induced hemarthrosis with



FIG. 3. Pigmented villonodular synovitis in a 79 year old woman, presenting as a synovial mass eroding the patella and tibia and perforating the cortex of the tibia.

continued use of the joint. It is possible that the process may initially represent reaction to injury with subsequent metaplasia and even neoplasia.[5,8]

Within synovium, bursa or tendon sheath the proliferative reaction consists principally of a polyhedral cell, apparently a synovial histiocyte, associated with varying amounts of fibrogenesis and collagen. In the cellular areas foam cells and cells containing iron pigments occur, the latter apparently from phagocytized erythrocytes. Multinucleated giant cells are virtually always present. Because of this wide array of histologic appearance, these lesions have also been called fibrous xanthoma of synovium, giant cell tumor of tendon sheath, and even xanthoma.

Villonodular synovitis and its variants may erode contiguous articular cartilage and extend into one or more adjacent bones (Fig. 3). The tumor of tendon sheath can cause pressure erosion of bone, especially of the phalanges, and there is a frequent association with degenerative or post-traumatic arthritis.[5]

REPORT OF CASES

Case 1. This case was contributed through the kindness of Dr. James McCort, San Jose, California, who with Dr. Donald L. Alcott presented this as Case 6 at the Penrose Cancer Seminar, Colorado Springs, Colorado, November 7, 1959. At that time the roentgenographic features were dscussed by Dr. Phillip J. Hodes and the histologic findings by Dr. Arthur Purdy Stout.[1]

The patient was a 73 year old man in March, 1958, when he had several episodes of hematemesis. He had lost 20 pounds of weight in the previous year. Blood pressure was 60/40 mm. Hg and the patient appeared anxious and weak. A firm, fixed mass was palpable in the left upper quadrant of the abdomen.

Roentgen examination revealed upward displacement of the stomach by a large retroperitoneal mass (Fig. 4) extending to the left and partially obliterating the left iliopsoas muscle. There was evidence of reflex ileus of the descending duodenal loop as well as of the horizontal limb of the duodenum due to the mass; the jejunal and ileal loops were also affected by the mass which appeared to lie in the mesentery adherent to the small intestine.



Fig. 4. Case 1. Xanthogranuloma in a 73 year old man, presenting as a 15×10 cm. firm fixed mass displacing the stomach upward and invading the fourth part of the duodenum.

At laparotomy a 10×15 cm. mass was found within the mesentery infiltrating the wall of the fourth part of the duodenum. The tumor was removed with the duodenum and 12 inches of jejunum. The wall of the small intestine appeared infiltrated as far as the submucosa. A 3 cm. ulcerating lesion of the fundus of the stomach was also found and proved to be an ulcerating adenocarcinoma. Excision of this was also carried out.

Histologically the retroperitoneal tumor proved to be a vascular granulomatous lesion heavily infiltrated with polymorphonuclear neutrophils and histiocytes, most of which were foamy and apparently contained lipids. The lesion was sharply circumscribed and there was remarkable uniformity of the morphologic features throughout the sections. Dr. Arthur P. Stout considered this a retroperitoneal xanthogranuloma.

Following surgery the patient's general condition deteriorated rapidly and he expired. An autopsy revealed no evidence of residual or recurrent tumor.

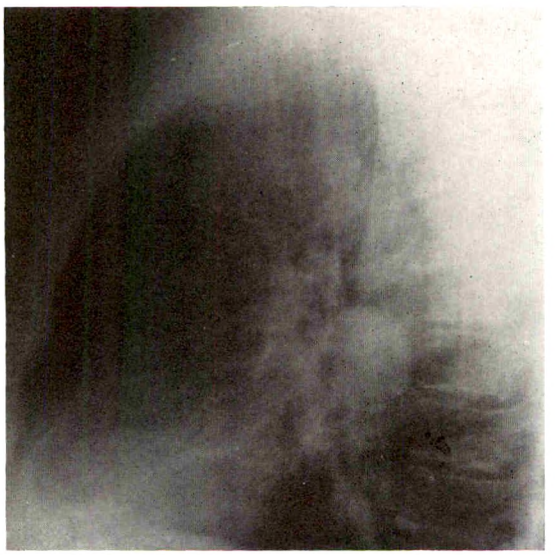


FIG. 5. Case II. The patient was a 50 year old man when a fibrous xanthoma of the abdominal wall was resected. One year later a mediastinal mass appeared in the region of the carina associated with recurrence in the incision site. Following radiation therapy, there was atelectasis of the right lower lobe and appearance of small pulmonary metastases.

CASE II. The patient was a 50 year old white man in December, 1967, when he developed a mass in the left lower anterior abdominal wall. This was biopsied and reported as being a fibrosing histiocytoma for which a subsequent radical resection was done.

One year later a chest roentgenogram revealed a 4 cm. mass below and behind the carina within the mediastinum (Fig. 5). The patient was hospitalized and bronchoscoped and a mass extending into the right main stem and lower lobe bronchus was biopsied.

Recurrence of the mass in the inguinal area was also noted and this was biopsied as well. The sections from the bronchial and inguinal biospy sites were quite similar histologically and considered to be a malignant fibrous xanthoma. The radiotherapist offered the opinion that this might be a well differentiated fibrosarcoma with a histiocytic response.

The patient was started on a course of radiation therapy without benefit. Atelectasis of the right lower lobe developed and was followed by the appearance of small pulmonary metastatic nodules. The recurrent lesion in the left inguinal region continued to grow. The patient was started on chemotherapy but expired in April, 1969.

Autopsy revealed the cause of death to be massive tumor within the trachea extending into the right mainstem bronchus. Multiple metastatic nodules were present throughout the lungs. The mass in the left groin had extended through the abdominal wall and lay adjacent to the descending colon. Final histologic diagnosis was malignant fibrous xanthoma.

DISCUSSION

Solitary tumors of histiocytes and fibrous tissue occur in both children and adults (Table III). These have generally been known as xanthomas, xanthogranulomas and histiocytomas. When the histiocyte, which is a facultative fibroblast as well as a phagocyte, is associated with considerable fibrous tissue, the tumor has also been termed fibrous xanthoma, sclerosing hemangioma, dermatofibrosarcoma protuberans, and recurrent dermatofibroma.[6]

There is no unanimity as to the nature of these tumefactions.[5,6,8–10,13,14,17] Some xanthogranulomas of the retroperitoneum and mesentery are inflammatory, while others appear to be histiocytic tumors capable of metastasizing.[1,17] O'Brien and Stout[10] reviewed 979 examples of fibrous xanthomas and dermatofibrosarcoma protuberans and 537 cases of giant cell tumors and villonodular synovitis of soft tissues. Of the 1,516 cases, 53 were selected as potential malignant tumors on a histologic or clinical

TABLE III

TUMORS OF HISTIOCYTES

Histiocytoma
Xanthoma
Xanthogranuloma
Fibrous xanthoma
Dermatofibroma protuberans
Fibrous xanthoma of synovium
Giant cell tumor of tendon sheath
Pigmented villonodular synovitis
Pigmented villonodular bursitis
Pigmented villonodular tenosynovitis

basis. Of these, 15 proved to be definitely malignant.[10]

It is apparent, therefore, that a small but definite number—about 1 per cent—of apparently benign fibrous xanthomas behave in a malignant fashion and that there are no reliable criteria by which these may be initially distinguished.

SUMMARY

The histiocyte is derived from the reticulum cell and is a member of the reticuloendothelial or reticulohistiocytic system. It is capable of enzymatic production, phagocytosis of bacteria, lipids, erythrocytes, cellular and foreign debris. It is also a facultative fibroblast.

It participates in systemic diseases such as metabolic histiocytosis, malignant histiocytosis, and histiocytosis-X.

It is also the principal cell in many granulomatous diseases including endogenous lipid pneumonia of lung and xanthogranulomatous pyelonephritis of kidney. Malacoplakia also appears to be a type of histiocytic granuloma.

Localized neoplastic or reactive tumor of skin, subcutaneous tissue, lung, mediastinum, retroperitoneum, synovium, bursa and tendon sheath have been variously termed histiocytoma, fibrous xanthoma, xanthogranuloma, xanthofibroma, giant cell tumor, pigmented villonodular synovitis, bursitis and tenosynovitis. A very small percentage of these apparently benign lesions are capable of metastasizing.

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THE CENTRAL X-RAY BEAM IN STEREOTAXIS*

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WITH the advent of stereotaxic surgery of the brain and spinal cord, neurosurgeons and radiologists have had to pay closer attention to various optical phenomena of x-radiation. These phenomena include parallax, geometric distortion, magnification, image density and phenomena which become critical when one attempts to transfer measurements from x-ray film images to a probe-guide located in the stereotaxic frame.[2] An understanding of these phenomena implies a consideration of what has been called the central x-ray beam.

In the majority of the literature concerned with the stereotaxic procedure reference is made to "the central x-ray beam" implying that this is represented by one and only one straight line in space.[1] One then may conclude erroneously that there is only one possible "central x-ray beam" arising from the x-ray tube and that it is the one that is somehow oriented to the axis of the x-ray tube or its collimator. In our own recent article on the subject of geometric phenomena in stereotaxis the phrase was used.[2] It became obvious to us what is axiomatic to physicists: there is an infinite number of "central" x-ray beams emanating from the activated x-ray tube anode. This being true we should be able to prove to ourselves that the stereotaxic frame supporting the patient's head and probe-guide can be aligned with an x-ray tube such that 2 defined points of the frame can be made to intercept a "central x-ray beam" regardless of the position, within limits, of the frame in relation to any axis of the x-ray tube itself. Note that the word "central" now becomes defined in relation to the stereotaxic frame (not the x-ray tube) in that it is that particular x-ray beam which happens to pass through both defined points on the frame.

METHOD

Most sophisticated stereotaxic frames have 2 points which require alignment with a central x-ray beam. We shall call one (the center of) a circle and the other (the center of) a cross, whose images are to be superimposed on the x-ray film. This particular study used a portion of the Todd-Wells stereotaxic frame set up in front of an x-ray tube (Fig. 1).

The observer placed the frame at any position in front of the x-ray tube and adjusted the frame (or tube) until the circle and cross were superimposed with the rotating anode of the x-ray tube. This was accomplished easily in a couple of minutes by the observer simply sighting with his eye along a line estimated to pass through

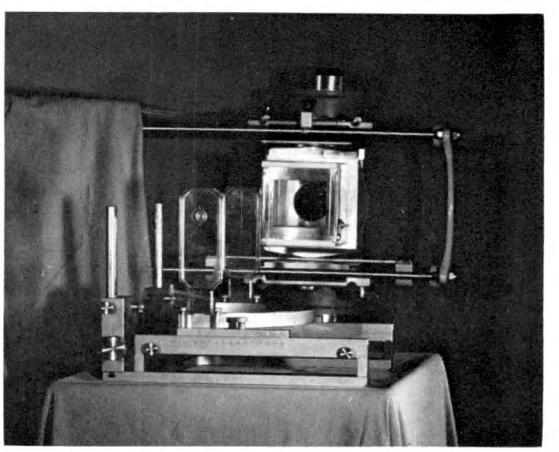


FIG. 1. *Foreground.* Portion of Todd-Wells stereotaxic frame containing 2 defined points (center of radiopaque circle and center of radiopaque cross in plastic supports) for alignment with an x-ray beam pathway. *Background.* X-ray tube in usual metal encasement with cone. Aluminum filter removed but anode not seen clearly in this view.

* From the Neurosurgical Section of the Veterans Administration Hospital and the George Washington University School of Medicine, Washington, D. C.

the circle and cross and adjusting the attitude of the frame until the (inactive) anode of the x-ray tube could be seen as superimposed visually. (For stricter accuracy one can make the sighting using a surveying telescope.) The only restriction was that the frame could not be located beyond the zone of radiation barred by the cone and metal encasement of the x-ray tube. If the observer could see the anode, his eye obviously was in the zone of potential radiation.

Then an x-ray film was placed just beyond the stereotaxic frame and perpendicular to the central x-ray beam axis and a roentgenographic image of the frame obtained. This was done at 10 different positions in space by moving the stereotaxic frame and keeping the x-ray tube stationary. Figure 2 demonstrates the observer's view of the circle and cross superimposed upon the illuminated anode. Note that in this instance the stereotaxic frame is located in the right periphery of the zone of radiation and is nowhere near the imaginary line (BG in Fig. 3) passing through the center of the x-ray cone. In this instance the central x-ray beam is line BH in Figure 3.

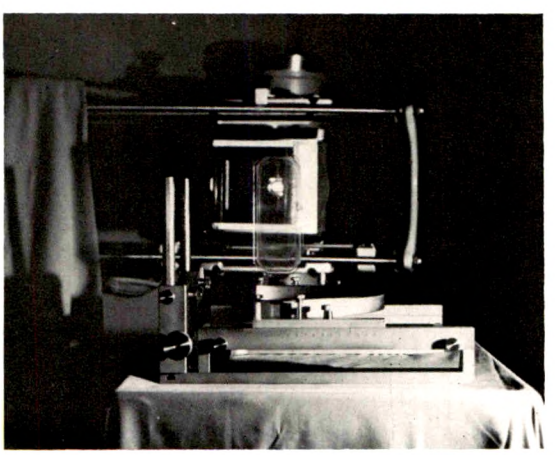


Fig. 2. Observer's view of x-ray tube anode (brightly illuminated by cathode filament) after circle and cross in Figure 1 are superimposed visually on the anode. This view represents the pathway of an x-ray beam selected as "the central x-ray beam" in this case, even though the x-ray cone and tube are "aimed" far to the side of the stereotaxic frame.

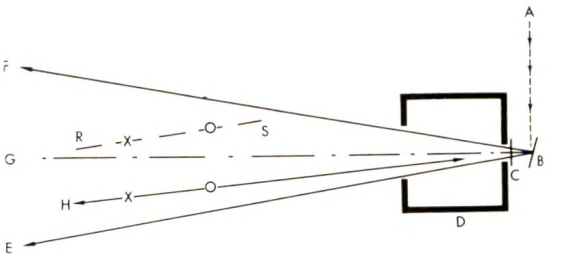


Fig. 3. Diagram of set-up in Figures 1 and 2. (A) Electron beam from cathode of x-ray tube. (B) Rotating anode and source of x-radiation. (C) Aluminum filter. (D) Cone. (BE and BF) Periphery of field of radiation. (BG) Central axis of field of radiation (not necessarily the central x-ray beam). (BH) X-ray beam selected as the central x-ray beam by virtue of the fact that it passes from point source B through points X and O on the stereotaxic frame. (RS) Stereotaxic frame X-O not aligned with any single x-ray beam.

In another series of trials in 10 different positions the circle and cross were lined up with the removable aluminum filter (C in Fig. 3) instead of the x-ray tube anode (B in Fig. 3) which it covers. X-ray photographs again were obtained.

RESULTS

In each instance in which the anode was visualized directly, the x-ray film revealed nearly exact superimposition of the circle and cross (Fig. 4), regardless of the position of the stereotaxic frame within the limits described. When the aluminum filter in front of the anode was used as the sighting target, accuracy was not quite as exact and the picture illustrated in Figure 5 was usually obtained, the cross still always being well inside the smaller circle.

DISCUSSION

This study is a by-product of our research in a new method of stereotaxis utilizing a biplane television system.[3,4] Such a system requires coaxial alignment of a central x-ray beam with the central optical axis of the television camera for

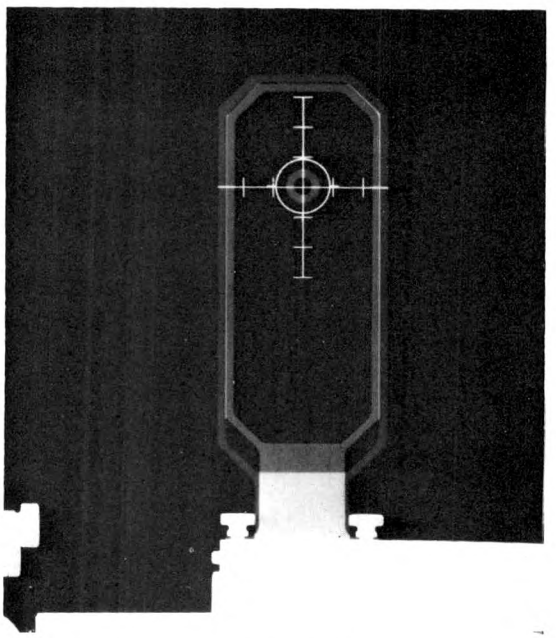


FIG. 4. Example of x-ray photograph of part of stereotaxic frame after alignment in Figure 2 was carried out.

each plane. In arriving at a simple method of effecting this alignment we found that an x-ray beam easily could be determined as the "central x-ray beam" if it passed through the cross and circle. Furthermore this now points out a rapid method of making this determination just prior to positioning of the patient for stereotaxic brain surgery; the surgeon simply adjusts the stereotaxic frame and x-ray tube until he can sight down an axis (Fig. 2) which allows him simultaneously to see the circle, the cross, and the x-ray tube anode or the aluminum filter covering it. In other words, the x-ray tube is not exactly aimed at the stereotaxic frame but rather the stereotaxic frame is aimed at the anode of the x-ray tube. Now he will be certain of alignment verified by x-ray filming (Fig. 4, or 5). Repeated trial and error x-ray filming is *not* necessary.

In Figure 3 lines BF, BG, BH, and BE all qualify as a central x-ray beam if the circle and cross lie in one of them. Note that line RS does not qualify unless the x-ray tube is moved so that line RS can intercept the anode at B. Line BG may be the central x-ray beam but there is no constraint requiring that it be. A perusal of any text on the physics of radiology will confirm that B in Figure 3 is a point source of radiation which emanates in all directions but which may be blocked in certain directions by protective metal encasement.[5] Within the limits of this restriction any of an infinite number of x-ray beams may be selected as the central x-ray beam, the choice depending upon which linear pathway between the circle, cross, and anode is selected by the physician.

SUMMARY

A common misconception is that there exists only one possible "central x-ray beam" arising from the x-ray tube and that it is the one that somehow is oriented to the axis of the x-ray tube or its collimator. However, the following axiom in radiology is emphasized: emanating from

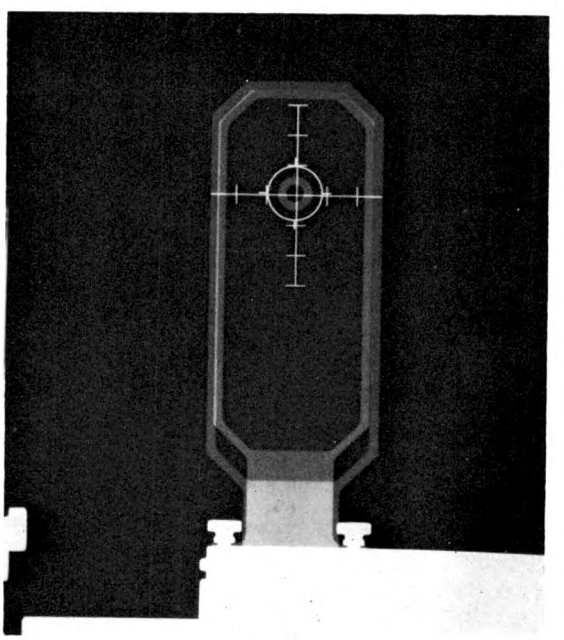


FIG. 5. Example of x-ray photograph of a part of stereotaxic frame after alignment in Figure 2 was carried out with x-ray anode covered by aluminum filter.

a point source of radiation (the x-ray tube anode) is an infinite number of x-ray beams, any one of which may be selected as "the central x-ray beam." In stereotaxis the particular beam selected will be that one which traverses a pathway from the x-ray tube anode through 2 defined points on the stereotaxic frame.

This pathway can be selected prior to the stereotaxic procedure by sighting through these 2 points and adjusting the x-ray tube and/or stereotaxic frame until these points visually superimpose on the x-ray tube anode or its aluminum filter. Regardless of the gross position of the stereotaxic frame with respect to the x-ray tube, within limits, the 2 defined points will have their roentgenographic images superimposed on the x-ray film.

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NEW CROSS COMPRESSION FORCEPS FOR CAROTID ANGIOGRAPHY*

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CROSS compression forceps has been devised in order to avoid radiation exposure to the operator during carotid angiography. In this communication, a new type will be illustrated.

TECHNIQUE

This compression forceps measures approximately 25 cm. in length, and has adjustable compression plates in both limbs. A coiled spring is attached to the limbs which have serrated surfaces on their dorsal aspect. The strength of the coiled spring can be adjusted by changing the position of its attachment on the serrated limbs of the forceps (Fig. 1).

The patient's neck is examined, and the level of carotid bifurcation is palpated and marked with ink. The forceps is then applied to the patient's neck and the carotid artery is compressed against the cervical spine by means of the compression plate with adequate angulation. The forceps is compressed further manually until the carotid pulsation distal to the forceps is not palpable. The forceps is instantly locked and remains in place during angiography (Fig. 2, *A* and *B*). Immediately following angiography, the forceps is unlocked and removed from the neck. During the application of the forceps, the operator stays behind the shield in order to avoid radiation exposure.

The time of compression of the carotid artery with this technique varies from 5 to 10 seconds.

A little practice is required to obtain consistently good results with this method.

DISCUSSION

If one carotid artery is compressed, the pressure gradient of the compressed artery and its cerebral branches becomes lower than the gradient on the noncompressed side. In the presence of a normally func-

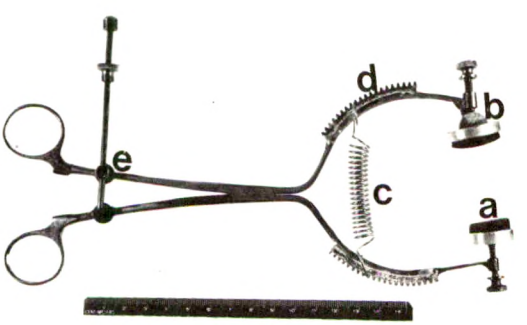


FIG. 1. Compression forceps for carotid angiography: (a) a supporting plate; (b) a compression plate; (c) a coiled spring; (d) a serrated spring rest; and (e) a forceps lock.

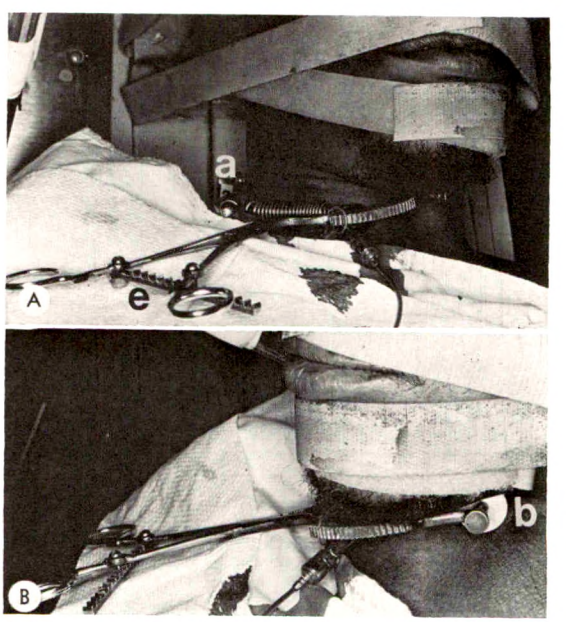


FIG. 2. (*A* and *B*) The compression forceps is in place during carotid angiography.

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tioning circle of Willis, the cerebral artery circulation flows to the compressed side.

The cross compression technique in carotid angiography is based on this principle and is frequently employed for the preoperative work-up of Berry aneurysms. If ligation of the carotid artery is contemplated, bilateral cross compression carotid angiography is required to determine whether there is adequate cross circulation in the cerebral hemispheres.

Adequate cross filling of the cerebral vessels frequently occurs with contralateral compression of the carotid artery. This provided an excellent comparison study in the anteroposterior view without subjecting the patient to an additional carotid puncture on the opposite side.

We routinely perform cross compression angiography in our institution whenever any parasellar mass lesion is suspected. Subtle abnormality of the middle cerebral arteries can be best evaluated by comparison of the vessels in the anteroposterior projection.

The best results of cross compression technique can be obtained by means of digital compression, because during the manual compression one can actually feel cessation of the carotid pulsation when the carotid artery is firmly compressed against the cervical spine. The only disadvantage of the digital cross compression is the unnecessary additional radiation exposure to the angiographer. This exposure problem is compounded in busy neuroradiologic institutions where the cross compression technique is frequently employed.

SUMMARY

A new cross compression forceps for carotid angiography is presented.

The indications for cross compression angiography are briefly discussed.

With the use of this forceps, one can eliminate radiation exposure to the operator during cross compression angiography.

This forceps has been used also for distention of the lymphatic channel in lymphography.[1]

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TELEVISION FLUORODENSITOMETRY*

A SIMPLE AND RAPID METHOD FOR THE EVALUATION OF IMAGE INTENSIFIER-TELEVISION SYSTEMS

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MALMÖ, SWEDEN

WITH the great acceptance of television as the method of choice for viewing the output of image intensified fluoroscopy, equally great effort has been made to determine the most accurate method for objective analysis of the performance of these systems. Several mathematical models have been suggested and of these, modulation transfer function analysis remains widely used.[1–10,12–14,17] As the image intensifier-television chain is employed increasingly for physiologic data acquisition, the need for objective evaluation of the system and accurate adjustment of the system becomes even more critical.[1,4,11,15,16] The use of television fluorodensitometry offers a simple and rapid approach to the solution of these problems using only the equipment required for densitometric data acquisition.

MATERIAL AND METHOD

For these experiments, the densitometric information was obtained using the method described in earlier works.[15,16] The composite video signal and the vertical synchronization timing pulse of the television system were coupled to an oscilloscope* so that the Z-axis voltage of one horizontal raster in the television scan frame was displayed. The resulting curve plotted on the screen of the oscilloscope is that of radiographic density *versus* spatial displacement. Throughout these experiments the following fluoroscopic equipment was employed. An Elema-Schönander Triplex Angiomatic Generator, No. 1003 was coupled to a Siemens Pantoskop 2 diagnostic fluoroscopy unit. A Siemens Biangulix-Rapid high speed x-ray tube with a 100 mm. diameter anode was in place below the examination table. Only the small (0.6 mm.) focal spot was used for our work. The image intensifier was a Siemens Sericon 10/6 inch unit, and this was coupled to the Siemens television chain consisting of a Vidicon camera (625 lines) coupled to a 14 inch monitor. The radiographic factors were limited in such a way that the operational capacity of the image intensifier and the television camera were not a limiting factor. For the experiments demonstrated in the examples shown below, the factors were 74 kvp. and 0.8 ma. Evaluation of the image intensifier-television system was performed in the following manner: A single raster was chosen from the center of the television scan and a curve of the density distribution was recorded with Polaroid film from the screen of the oscilloscope. A raster was then chosen 1 inch from the top of the television image and recorded. This was repeated with a raster 1 inch from the bottom as well. When the best possible density distribution was obtained from adjustment of the various components, analysis of the ability of the system to transmit information was performed. A thin lead foil phantom† (Fig. 1) with spaces of varying width was used for the remainder of the experiments. Phantoms of similar nature have been used in multiple evaluations of this type and

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This work was in part supported by USPHS(NIH-NHI) Grant No. 1-F03-HE-42,651-01.
† Special Research Fellow, National Heart Institute, NIH, at the Department of Radiology, Malmö General Hospital, Malmö., Sweden. On leave of absence from The Thomas Jefferson University Hospital, Department of Radiology.
‡ Professional Service Engineer, Elema-Schönander AB, Malmö, Sweden.

found suitable.[4,8,10,11] Density curves were obtained with the television chain and the image intensifier in predetermined modes of excellent and poor adjustment to demonstrate the ability, or lack thereof, of the system to transmit information.

RESULTS

Figure 2*A* demonstrates the irregularity of transmission that was present on the initial evaluation of the test system. It was necessary to adjust factors in both the television chain and the image intensifier to obtain the curve shown in Figure 2*B*. While not perfectly symmetric, there is a marked improvement in the shape of the curve, indicating more homogeneous density transmission, and an improvement in the signal to noise ratio is apparent. In Figure 3, *A* and *B* the density curves obtained from the upper and lower portions of the television image are demonstrated. While more symmetric than the center raster, there is a difference in amplitude between the lines that is not equal when compared to the center line. We were unable to make any adjustments which would correct this difficulty and the possible significance of this finding will be discussed briefly in a following section.

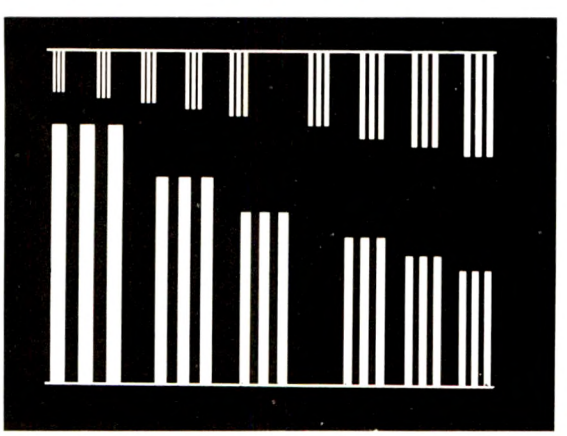


Fig. 1.

The phantom, photographically enlarged, from which the sinusoidal density curves are obtained. It is constructed from lead foil, 50 micra thick, in which lines varying from 0.4 to 2.5 lines per millimeter are etched. Other models of similar construction are available which have a continuous spacial spectrum.

When the imaging system had been as well adjusted as was possible, maladjustments were introduced into the system to demonstrate their effects on the information transfer capabilities of the system. These are seen in Figure 4, *A–E*. Figure 5, *A* and *B* demonstrates the changes accompanying magnification when the image intensifier is changed from the 10 inch to the 6 inch mode.

The degrading effects of these maladjustments are easily recognized and appropriate modulation transfer function calculations would graphically demonstrate this fact. We have chosen to ignore this mode of presentation for reasons which we will describe below.

DISCUSSION

In their communication, Moseley, Holm, and Low[11] demonstrated clearly that a composite modulation transfer function curve could be obtained for an entire image intensifier-television chain, if one excluded the monitor stage. Their use of the composite video signal presented curves of excellent quality which depict the density distribution over the entire surface of the image intensifier, and hence, an average value for the ability of the system to transmit information. Holm[8] had postulated the curve that would be obtained from a single raster, but he did not demonstrate this finding in his preliminary paper. Since the single raster technique is the foundation for physiologic data acquisition using television fluorodensitometry, it seemed reasonable to apply it to the important problem of image intensifier-television system performance analysis and adjustment.[15,16]

While curves such as the ones demonstrated above could, with the proper phantom, be used for the calculation of modulation transfer function, we feel that this is an unnecessary waste of time for any individual machine. There is no doubt that if one wishes to compare the information transferring ability of one system with another, then modulation transfer function curves will give an objective cri-

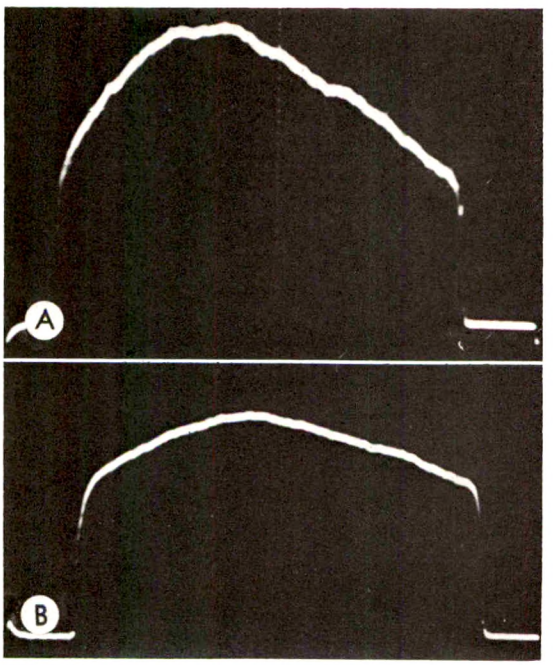


FIG. 2.

(*A*) Transmission irregularities are demonstrated by the asymmetry of the density curve obtained from the image intensifier-television system. The fluoroscopic shutters were fully opened.

(*B*) An improvement in the symmetry of the density distribution and in the signal to noise ratio is noted following adjustment of the video gain, the television camera beam and the focus of the image intensifier.

terion of good quality. If line spread function is used as well, then most imaging systems can be adequately described with respect to this problem.[13,14] However, when the question is how well any one system is transmitting information, then the actual calculation of the modulation transfer function curve is probably a needless expenditure of professional time. When one views the density curves presented on the face of the oscilloscope with a critical eye, one quickly learns to evaluate the changes in amplitude of the density signal with respect to spatial interval. While this may not have the ease using the entire video scan, it has been quite easy using the curve derived from a single raster. It is, therefore, equally easy to recognize maladjustments in the information chain and corrections can be made while the density curve is simultaneously displayed on the oscilloscope. This gives the service engineer a quick and ready reference for the objective evaluation of his adjustment procedure. One might suggest that calculation of the modulation transfer for function will give a standard reference against which continuing performance evaluations of the same system could be made. This is true but unnecessary, since one need only photograph the density curve on the oscilloscope screen when proper adjustment is obtained. By recording the radiographic factors on the photograph, it is a simple matter to duplicate these factors for repeat evaluations and the curves of the

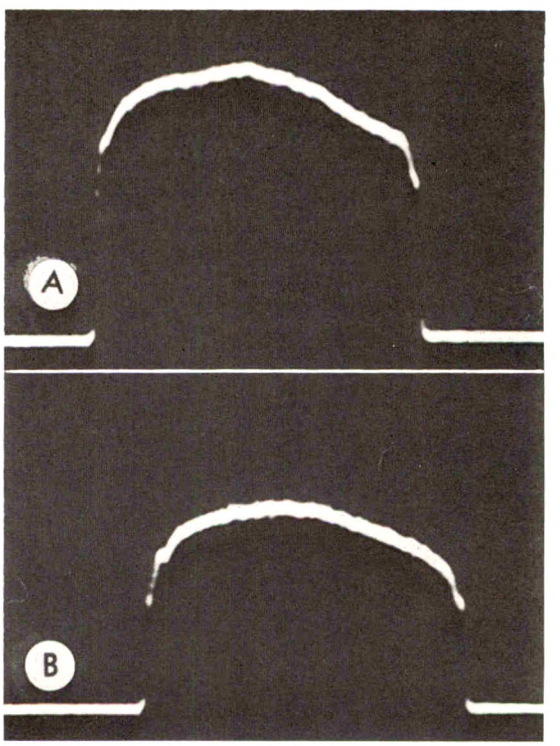


FIG. 3.

(*A*) The density curve obtained from a single raster 1 inch from the top of the television scan.

(*B*) The curve from a raster 1 inch from the bottom of the television scan. These curves are both more symmetric than the curve from a center raster (Fig. 2*A*) and their width is decreased due to the circular format of the television scan. The difference in amplitude between this curve and that in *A* could not be corrected by any adjustments of the system.

density can be readily compared. A review of several important works concerning the calculation of modulation transfer function and line spread function is included in the bibliography.[2,3,6,8–11,17]

Since it was impossible for us to obtain a completely symmetric density curve over the center of the image intensifier surface, the possible causes of this problem must be examined. We have not yet completed the work in this area and it remains an area of uncertainty. The most obvious and easily correctable cause would be a malalignment of the x-ray tube and the image intensifier. In the demonstration above, this was not the case. Since the orientation of the x-ray tube was perpendicular to the axis of the television scan, "heel effect" cannot be the cause. When one considers the discrepancy in transmission between the top and bottom scan lines however, heel effect may play an important role. The orientation of the x-ray tube placed the anode at the bottom of the television scan and the difference noted in the transmission fits nicely into this theory. We have inadequate data at this time to confirm or refute this possibility. Finally, it is possible that inhomogeneity of the density may be due to irregularities in the coating of the input phosphor or, much less likely, the output phosphor of the image intensifier. It is in these areas that we are continuing our investigations, and the results will be the subject of a later presentation.

The inability to explain many of the causes of irregular density transmission

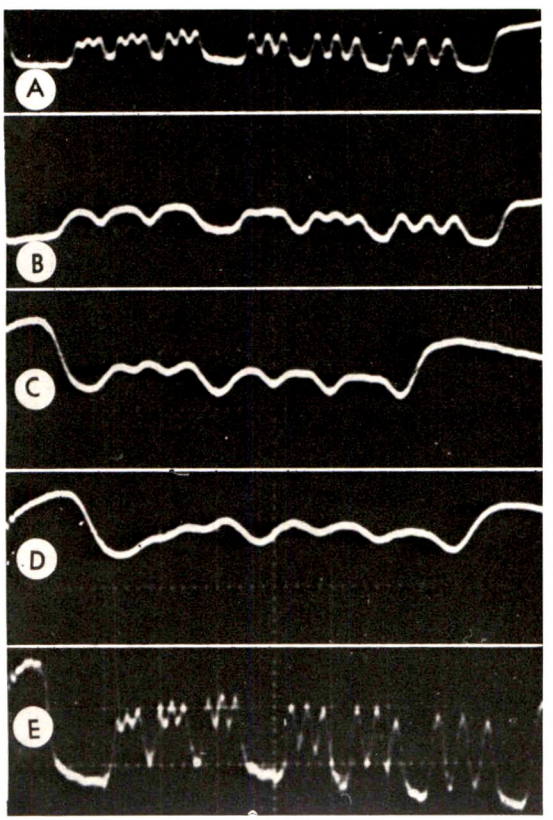


Fig. 4.

(*A*) The density curve obtained from the phantom with proper adjustment of the image intensifier and the television system.

(*B*) The curve obtained with the image intensifier electron focus No. 1 fully deflected to the O-right position and excessive beam to the Vidicon.

(*C*) Electron focus No. 2 fully deflected to the O-right position and excessive beam to the Vidicon.

(*D*) Vidicon out of focus, with the image intensifier well adjusted.

(*E*) Excessive gain to the Vidicon. There has been an improvement in contrast of the most widely spaced lines without a corresponding improvement in the highest spatial frequency group. Hence, the modulation transfer function curve would show a more rapid drop than would be found with the density curve in Figure 3*A*.

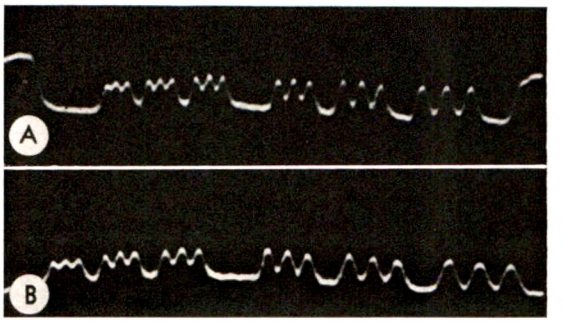


Fig. 5.

(*A*) The density curve obtained with the image intensifier-television chain properly adjusted and in the 10 inch mode.

(*B*) In the 6 inch mode, the degree of magnification is apparent. There is no appreciable loss in ability of the system to transmit information. The phantom was fixed in the center of the image intensifier input phosphor field, which suggests that this portion should be used in densitometric measurements employing this technique.

does not prevent the use of this technique for evaluation and adjustment of image intensifier-television systems. This work does bring into sharp focus the fact that physiologic data acquisition requires the best system adjustment possible. The inhomogeneities of transmission point out the need to use only the center-most portion of the image intensifier when one is interested in accurate densitometry. Perhaps equally important is the need to maintain a fixed field size and geometry throughout any given densitometric experiment. Within this framework, television fluorodensitometry can play an important role in the work of both radiologist and professional service engineer.

SUMMARY

Television fluorodensitometry provides a simple and accurate objective measurement of the ability of an image intensifier-television system to transmit information. The need for the calculation of modulation transfer function curves is discussed with respect to this technique.

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ULTRASONIC DETECTION OF CAVITATION AT CATHETER TIPS*

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CONTRAST techniques have recently been applied to ultrasound cardiography for the purpose of identification of the cardiac chambers. The contrast effect has been obtained with a variety of substances ranging from physiologic saline solution to the patient's blood (reinjected into the heart). The contrast effect is transient, lasting long enough to permit identification of one side of the heart, but not long enough to pass through the pulmonary circulation or the systemic capillaries. It is seen as a multitude of echoes which fill the injected cardiac chamber (Fig. 1). There is no patient reaction during the contrast effect. Joyner[8] is credited with first having used this technique in the study of the mitral valve. Subsequently, Gramiak and Shah[5] used this technique for the identification of the aortic root. Further use of the contrast injection technique was made in another study to establish the chamber anatomy of the heart and to study intracardiac shunting, the configuration of internal cardiac structures, and the functional anatomy of certain disease states.[6]

The purpose of this investigation has been to determine which of 3 mechanisms is responsible for this ultrasound contrast effect: (1) acoustic impedance difference between blood and the injected fluid; (2) turbulence resulting from the injection; or (3) production of miniature bubbles (cavitation) at the catheter tip as a result of the injection.

The experimental techniques center about the ultrasonic study of *in vitro* injections in a plastic chamber, using fluids of different acoustic impedance and various flow rates producing turbulence as well as cavitation. Static pressure applied to the chamber was used to separate the contribution of cavitation from that of turbulence and acoustic impedance difference. Only the echoes produced by bubbles should be reduced by increased pressure. The results clearly implicate cavitation as the primary source of echoes.

ULTRASONIC TECHNIQUE

Reflection of ultrasound occurs at interfaces between media of differing acoustic impedance. In clinical use, short bursts (1 to 2 microseconds) of ultrasonic energy are emitted by a transducer held in contact with the skin. The interval between generation of the pulse of ultrasound and the arrival of the echo is a measure of the depth of the reflecting surface. A high pulse rep-

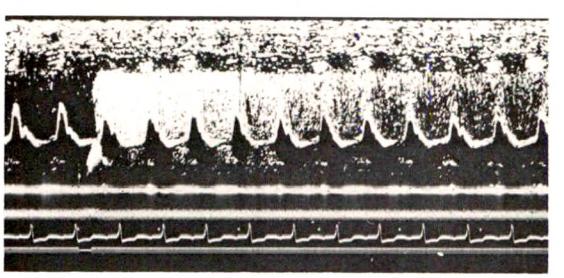


Fig. 1. Contrast effect obtained by the rapid injection of 5 cc. of saline solution into the left ventricular outflow tract. The contrast echoes are contained by the ventricular septum above and the mitral valve below. Clearing of the contrast effect is slow because of aortic insufficiency.

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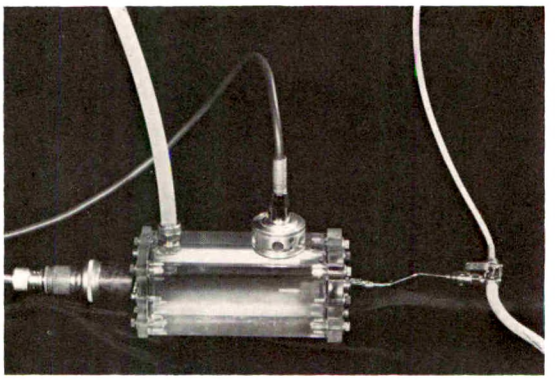


Fig. 2. Plastic test chamber, ultrasonic transducer (left) and microphone (above). Inflow is on the right, through the short catheter. The large outflow tube also connects to a reservoir through which pressure in the chamber can be controlled.

etition rate (200 to 2,000/sec.) permits accurate and detailed recording of the position of rapidly moving structures such as the valves of the heart.

The echo pattern is usually displayed on the face of an oscilloscope by either of 2 methods. "A" mode shows the returning echoes as spikes which move back and forth on the X axis as the depth of the reflecting surface changes and is used for the identification of echo patterns. The height of a peak is proportional to the amplitude of the returning signal. The motion of the reflecting surfaces is generally recorded in "B" mode. In this mode, the echoes produce intensity modulation of the oscilloscope beam such that the "A" mode spikes are converted to points. Strong signals produce intense points of light whereas weaker signals are diminished in brightness. The position of points on the X axis indicates the location of the echo sources. Their movement can be recorded by various means to produce time-motion records.

EXPERIMENTAL EQUIPMENT

The transparent plastic test chamber used for propagation of ultrasound in liquids under various static pressures and injection flow rates is shown in Figure 2. Fluids were injected into the chamber through a short catheter with a bore of 0.93 mm. The chamber measured 10 cm. in length and 2.5 cm. in height and width, giving a volume of 62.5 cc. Figure 3 illustrates the complete experimental system.

A Cordis injector model number 97A-6 provided injection volumes of 2 to 80 cc. with injection pressures up to 800 psi and a signal that indicated the time and length of the injection. An outflow tube from the test chamber was connected to a reservoir tank with a volume of about 1,000 cc. A compressor, connected to the reservoir tank through tubing, provided positive static pressures in the tank and test chamber.

The sounds associated with the injection were detected by a Maico contact microphone and filtered by a Krohn-Hite band pass filter model 310-C with the band pass set at 10–20 KHz and passed through a Sanborn heartsound preamplifier model 350-1700B.

The ultrasonic transducer was placed against the end of the chamber opposite the catheter tip and the heart sound microphone was placed on the side of the chamber over the catheter tip (Fig. 2).

A Physionic VT-500-CA-M somascope with a 2 MHz transducer was used to make all the ultrasonic recordings. Observation of the echo pattern was carried out in "A" mode continuously through the operation period on a Tektronix type 564 oscilloscope. Simultaneously, the same ul-

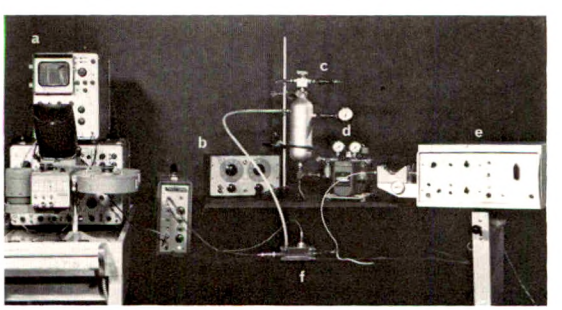


Fig. 3. Experimental system: (a) Ultrasonic generating and recording apparatus; (b) preamplifier and filter for recording cavitation sounds; (c) reservoir tank for fluid outflow and pressure regulation; (d) compressor; (e) injector; and (f) plastic test chamber.

trasonic information was displayed in "B" mode on the upper beam of a Tektronix type 565 dual beam oscilloscope. The lower beam was used in multi-trace operation by means of a type 3A74 Tektronix four-trace preamplifier. This permitted simultaneous recording of a time marker, an injection signal, and of cavitation sounds produced by the injection. Permanent records of the combined ultrasonic and injection data were made on 35 mm. film by means of a Fairchild oscilloscope record camera, type 321-A.

The sensitivity setting of the ultrasound receiver preamplifier was maintained constantly at 6.0 (maximum setting 10.0). The estimation of the amplitude of the ultrasound echoes was made visually from the "A" mode display (Fig. 4, *A* and *B*) and expressed in average signal height (range 0–8 cm. on the oscilloscope screen scale).

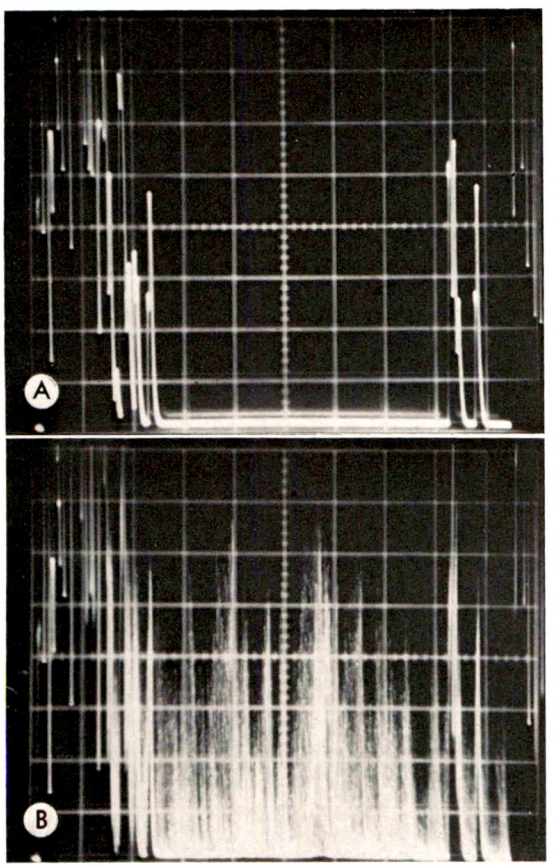


Fig. 4. "A" mode display of experimental echo patterns. (*A*) The upper panel depicts the water filled chamber under static conditions. (*B*) The lower panel was recorded during a rapid injection of water. The numerous echoes arising within the chamber were measured against the oscilloscope screen scale (1 division = 1 cm.).

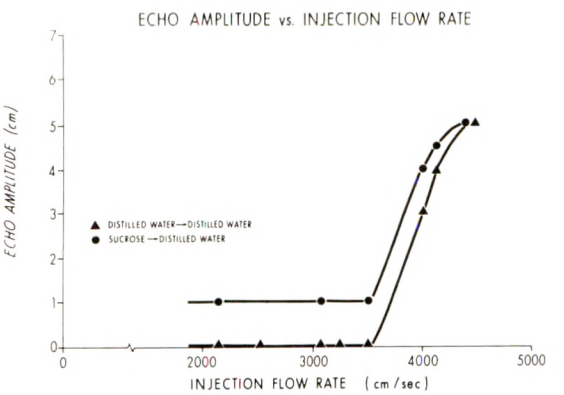


Fig. 5. Injections of distilled water and 1 molar sucrose solution into distilled water. At low flow rates the difference between 1 molar sucrose solution and distilled water injection is due to the difference in their acoustic impedances. The increase in echo amplitude at higher flow rates is due to cavitation.

RESULTS

WATER, SALINE SOLUTION, AND SUCROSE SOLUTION EXPERIMENTS

When distilled water was injected into distilled water, no echoes could be detected below a flow rate of 3,500 cm./sec. (24 cc./sec.).* Higher flow rates were accompanied by the production of echoes within the chamber (Fig. 5), an audible hiss, and a visible jet (Fig. 6). The chamber cleared of echoes immediately following cessation of injection. A pressure of 5 psi applied to the chamber during injection eliminated the echoes, hiss, and visible jet during similar injections (Fig. 7).

Tap water injected into tap water produced echoes, the hiss, and visible jet at slightly lower flow rates (3,200 cm./sec. or

* The injection flow rate is described as a fluid velocity at the catheter tip or a fluid volume flow rate. They are related as follows:

$$\text{fluid velocity} = \frac{\text{volume flow rate}}{A},$$

where A = catheter tip area = 6.8×10^{-3} cm.^{2}.

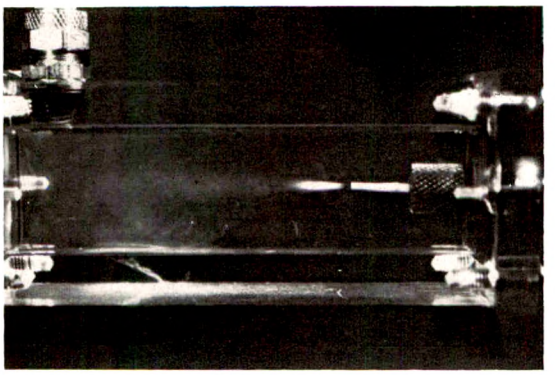


FIG. 6. Close-up of test chamber during injection of water into water. The intense visible jet and its distal diffusion were accompanied by an audible hiss and reflection of ultrasound.

22 cc./sec.) and above. Small bubbles appeared in the chamber and lingered for about 1 to 5 seconds, following injection.

When physiologic saline solution was injected into physiologic saline solution, the results were similar to those obtained with distilled water.

A temperature difference of 20°C. between the injected fluid and the chamber contents produced no additional echoes.

The acoustic impedance difference between 1 molar sucrose solution and distilled water is 0.21×10^5 gm./cm.^{2}-sec.† The injection of 1 molar sucrose solution into distilled water produced echoes with an average amplitude of 1 cm. at injection flow rates up to 3,500 cm./sec. (24 cc./sec.) (Fig. 5). Above this flow rate, the echo amplitude increased to 5 cm. and the hiss and visible jet were present. The addition of 25 psi chamber pressure reduced the echo amplitude to 1 cm. (Fig. 7) and this could not be reduced further by the application of additional pressure. At this pressure the hiss and visible jet disappeared.

The injection of 1 molar sucrose solution into 1 molar sucrose solution produced no echoes at flow rates below 3,500 cm./sec. (24 cc./sec.). Above this flow rate an average echo amplitude of 4 cm. was observed. The echoes produced at this time were also pressure sensitive.

† The amplitude of sound wave reflections due to mixing of fluids of differing acoustic impedance would be a function involving more than just the impedance difference of the fluids. However, qualitative predictions of reflection amplitudes can be made on the basis of the impedance difference.

BEEF BLOOD EXPERIMENTS

The chamber was filled with citrated beef blood. Saline solution, bubble free indocyanine green solution, blood, and 50 per cent hypaque were used as the injected media and all produced essentially the same results. At a flow rate of 3,000 cm./sec. (20 cc./sec.) echoes with an average amplitude of 5 cm. were present in the chamber (Fig. 8). When the flow rate was increased to 4,000 cm./sec. (27 cc./sec.) the average echo amplitude increased to 7 cm. and a hiss could be heard. At all flow rates,§ small visible bubbles were produced in the chamber and they tended to last indefinitely. For a flow rate of 3,200 cm./sec. (22 cc./sec.) a chamber pressure of 30 psi

§ The minimum flow rate used was about 2,000 cm./sec. (14 cc./sec.).

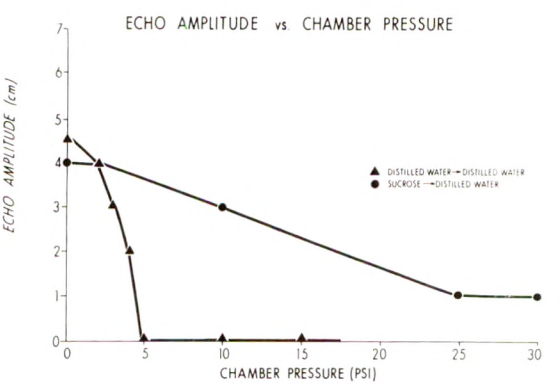


FIG. 7. The reduction of echo amplitude by chamber pressure in the distilled water injection implicates bubbles (cavitation) as the echo origin. The persistence of low amplitude echoes under high pressure conditions during 1 molar sucrose solution injection separates the contribution of acoustic impedance difference from that of cavitation. Turbulent flow present in the distilled water injections made at chamber pressures over 5 psi resulted in no detectable echoes. Flow rate for 1 molar sucrose solution 4,000 cm./sec.; for water 4,200 cm./sec.

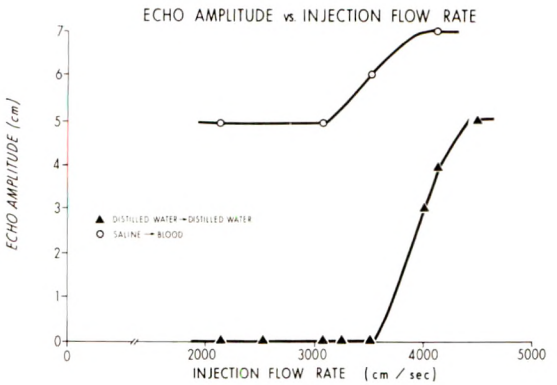


FIG. 8. Comparison of echo amplitude obtained during injection into blood and distilled water. The high echo amplitude obtained from blood at low flow rates results from stable cavitation. The abrupt increase at more rapid flow rates is from transient cavitation.

reduced the echo amplitude to 1 cm. (Fig. 9).

DISCUSSION

For the purpose of this presentation, acoustic impedance can be considered to be the product of the density of a medium and the sound velocity in that medium. The source of echoes obtained during injections into the chamber cannot be attributed solely to differences in acoustic impedance of the fluids utilized, since injection of water into water, 1 molar sucrose solution into 1 molar sucrose solution and blood into blood all produced echo sources in the chamber at sufficiently high flow rates.

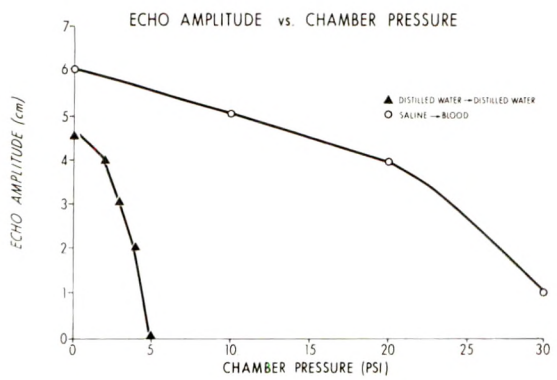


FIG. 9. The injection flow rate into blood was maintained at 3,200 cm./sec. while the chamber pressure was varied. The greater pressure required to reduce the echo amplitude in the blood-filled chamber is probably due to the presence of larger and more numerous bubbles resulting from stable cavitation.

An acoustic impedance difference of 0.21×10^5 gm./cm.^{2}-sec. resulted in a 1 cm. average amplitude signal during injections of 1 molar sucrose solution into water. A smaller average amplitude signal should be obtained from injections of saline solution into blood since the acoustic impedance difference is smaller (0.13×10^5 gm./cm.^{2}-sec.). Therefore, acoustic impedance difference is a small contribution to the total echo amplitude observed in clinical contrast echocardiography.

Sound reflection may occur at the boundaries of moving fluid layers[15] or in areas of turbulence.[12] Meisner and Rushmer,[10] using photographic techniques, were able to demonstrate turbulence beyond a constriction at flow rates as low as 8 cc./sec. We estimate that the tip velocity in their investigation was in the vicinity of 150 cm./sec. These flow rates were exceeded in our experiments with water and no echoes could be detected that might originate from turbulence or moving fluid layers. In these experiments, care was taken to maintain constant flow rates when static pressure in the chamber was altered. In cases where echoes were observed with water, the echo amplitude could be driven to zero by static pressure even though the turbulent flow continued unchanged.

The fact that the echoes produced in our study by injection can be reduced by increasing the environmental pressure in the chamber is the strongest evidence that bubbles represent the primary echo source. Increasing static pressure reduces sound reflection from bubbles in 3 ways:

1. Increasing pressure reduces bubble formation.
2. Increasing pressure reduces the size of any bubbles that are formed by com-

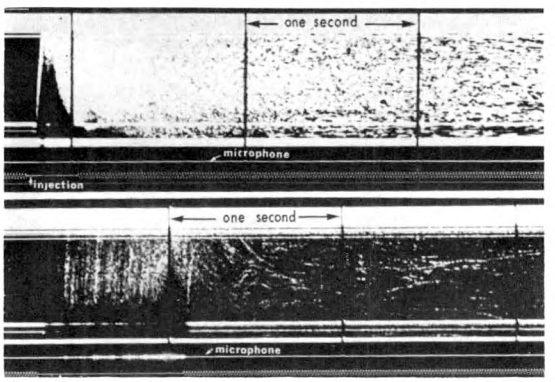


FIG. 10. Time-motion record of echoes obtained by injection into blood (above) and water (below). The top of each panel represents the transducer position on the end of the chamber while the bottom shows the inflow end (see Fig. 2). The time and duration of the injections are shown by the loss of the 60 cycle signal (injection). Sounds arising within the chamber are demonstrated (microphone). Note the persistence of echoes produced at the stable cavitation flow rate of 3,200 cm./sec. in the blood filled chamber and the lack of cavitation sounds. The water-filled chamber shows typical transient cavitation at a flow rate of 4,200 cm./sec. accompanied by a cavitation hiss. Both studies were made at atmospheric pressure.

pressing the gas inside the bubbles. This decreases the sound scattering cross section of the bubbles.

3. Increasing pressure increases the speed with which bubbles redissolve or collapse.

The ultrasonic method appears to be ideally suited for the detection of bubbles since the gas-liquid interface represents an efficient reflector.[13] Heisig and Mitchell[7] have described an industrial ultrasonic bubble detector. Using ultrasound, gas bubbles have been detected in the venous system of animals in experimentally produced bends[4,11,14] as well as in the heart following intravenous injection.[9]

In gas-rich media, such as blood, cavitating flow rates are much lower than those for gas-poor media. The observed bubbles persist for periods in excess of 10 seconds (Fig. 10) and probably contain gases which have been removed from the blood by the low pressures associated with the flow (Bernoulli effect). After formation, the bubbles leave the low pressure region and return their gases to the liquid by diffusion. This relatively slow process, called stable cavitation, accounts for the observed persistence of bubbles in blood. With higher flow rates and resulting lower pressures, transient cavitation* occurs. The bubbles which are formed under these circumstances collapse rapidly when removed from the low pressure zone. Shock waves accompany the collapse giving rise to the hiss of transient cavitation.

Cavitation depends upon the presence of nuclei in the medium to initiate bubble formation.[2] It would appear that blood is an ideal medium for stable cavitation because of its high gas content and probable high availability of cavitation initiating nuclei.

In clinical angiocardiography, injection rates in excess of 20 cc./sec. are common and are therefore probably all accompanied by cavitation. The volume of gas produced is difficult to assess. In general, the contrast effect obtained with 2 cc. of indocyanine green solution (containing foam due to mixing procedure) is considerably greater than that obtained by cavitating blood in the heart with the injection of 10 cc. of saline solution or blood. The amount of foam visible in a typical syringe of indocyanine green is probably less than 1 per cent (0.02 cc.) of its total volume. We therefore conclude that the total volume of bubbles generated during intracardiac cavitation results in 0.02 cc. of gas or less.

Bove *et al.*[1] suggest that there is a potential hazard of air embolism associated with intracardiac cavitation. Our experience suggests that the volume of gas gen-

* Stable and transient cavitation are frequently called gas and vapor cavitation respectively in the literature. The use of the term vapor cavitation implies a knowledge of the mechanism of the rapid formation and collapse of bubbles in a liquid. Since, in fact, very little is known of these processes and, further, since the mechanisms are of little importance in the present study, we prefer to use the phenomenologic terminology introduced by Flynn.[3]

erated during intracardiac cavitation is extremely small and probably no more hazardous than intracardiac injections of foam-containing indocyanine green which have been used for many cardiac output studies without evidence of gas embolization.

CONCLUSION

An ultrasonic contrast effect has been produced in living subjects by the intracardiac injection of liquids at rapid rates. The present study shows that these contrast echoes represent numerous small gas bubbles produced by the injection. The flow rates required to produce these bubbles in blood are below those associated with transient cavitation. Turbulence, temperature difference, and fluid acoustic impedance differences have been investigated and found to produce echo sources which are probably not significant in the clinical setting. The echoes produced experimentally could be reduced by an increase in the environmental pressure, strongly suggesting that bubbles represent the primary echo source. Clinical angiocardiographic studies use flow rates which are great enough to cause bubble formation. In our experience this degree of intracardiac bubble formation is not accompanied by any evidence of gas embolization.

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UNEVENTFUL PERFORATION OF A CORONARY ARTERY DURING SELECTIVE ARTERIOGRAPHY*

A CASE REPORT

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WE HAVE recently observed a remarkably uneventful perforation of a branch of the left coronary artery occurring during selective coronary arteriography. Because we are unaware of the reported occurrence of such a complication, the details of this case are presented.

REPORT OF A CASE

T.C., a 44 year old male, had been followed in our clinic for 10 years prior to the present admission in November, 1969. The diagnosis of rheumatic heart disease with aortic valve involvement had been made several years before. He had occasional anginal pains precipitated by exercise and intercourse. There was electrocardiographic and roentgenographic evidence of left ventricular enlargement. On auscultation there were systolic and diastolic murmurs over the aortic area. A typical Austin-Flint murmur was heard over the cardiac apex. A loud ventricular gallop was also present. Right and left heart catheterization performed on November 10, 1969, revealed the presence of moderately severe aortic insufficiency without significant stenosis. The cardiac output and cardiac index as determined by the dye dilution technique were within normal limits. The aortic pressure was 174/48 mm. Hg with a mean of 90. The left ventricular pressure was 174/0 with an end diastolic of 26. It was recommended that a coronary arteriogram be obtained prior to aortic valve replacement in order to rule out the presence of significant lesions of the coronary arteries which may have contributed to or caused the patient's angina.

On November 12, 1969, a selective coronary arteriography was performed using the technique described by Judkins.[2] The examination was performed by 2 residents in radiology, under the close supervision by one of us (L.B.M.). A No. 5 (medium size) coronary catheter was introduced without difficulty into the left coronary artery. A test dose of 2 cc. of 76 per cent meglumine diatrizoate was injected by means of a mechanical injector with a pressure of 150 psi. Immediately following the injection and as the contrast agent disappeared from the vessels, a crescent shaped collection of contrast substance with ill defined borders remained lateral to the left aortic cusp from which it was separated by a distance of approximately 1.5 cm. The tip of the catheter was then withdrawn from the coronary artery into the left coronary cusp. A spot roentgenogram was obtained to demonstrate the collection of contrast substance and its relation to the aortic cusp (Fig. 1*A*). The patient did not complain of any symptoms and only when questioned referred to slight "tightness" in the precordium. The electrocardiogram remained unchanged without evidence of myocardial injury or arrhythmia. The aortic pressure was constantly monitored and remained unchanged. At this time it was felt that extravasation of contrast substance had occurred. Because of the good flow through the vessels and the size and shape of the collection of radiopaque agent, it was felt that the injection was not subintimal. Considering the absence of symptoms or apparent untoward effects, it was decided to continue with the examination. The catheter was then reintroduced in the left coronary artery taking considerable care to avoid excessive penetration of the catheter tip. A total of 8 injections of 6 cc. each were then made again utilizing a mechanical injector with a pressure of 150 psi. Single spot roentgenograms and a 35 mm. cine study were obtained in multiple projections in order to visualize the entire left coronary system. With each successive injection the amount of extravasated contrast substance increased slightly (Fig. 1, *B–D*). There were no further symptoms. The electrocardiogram and pressure

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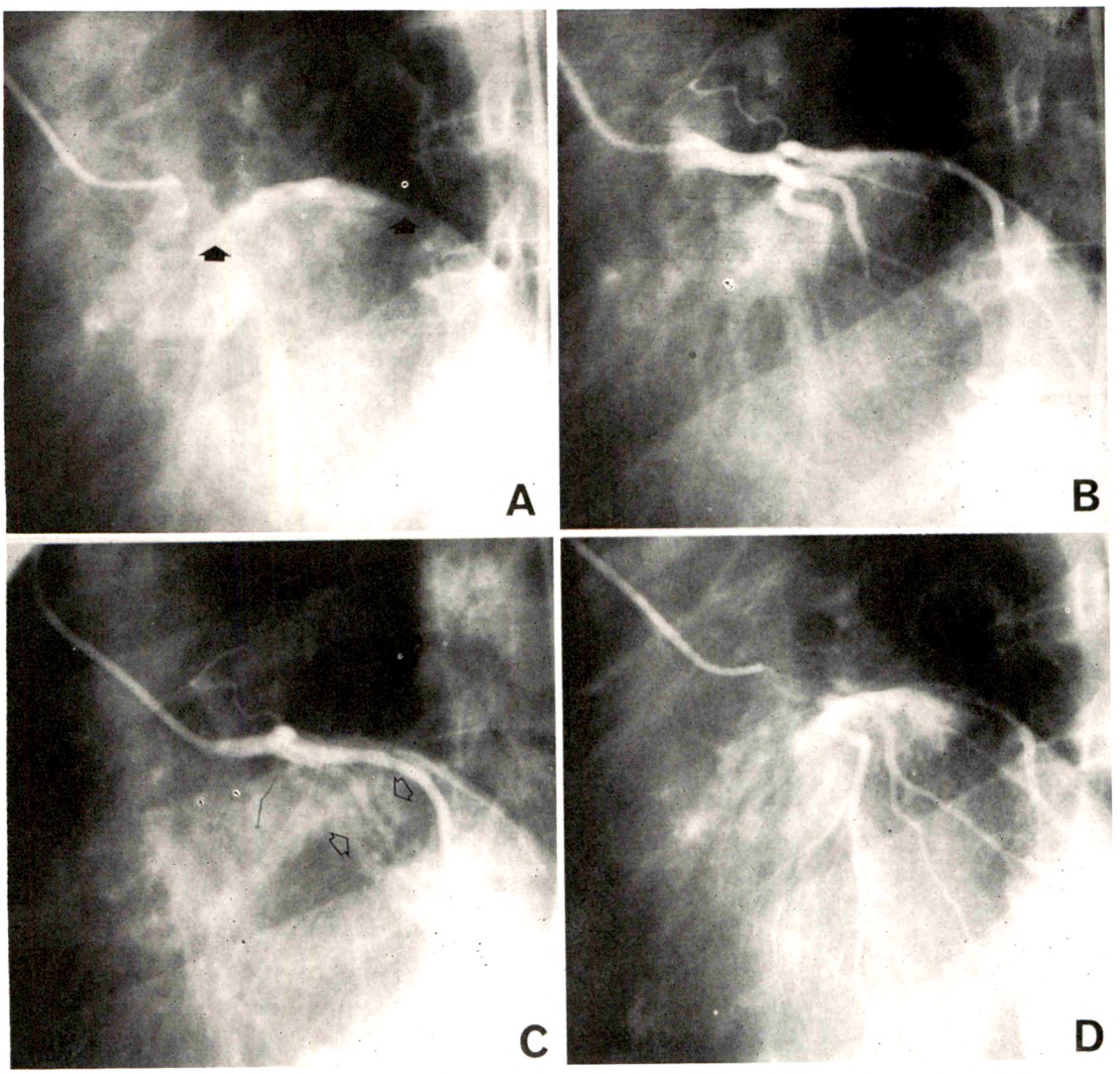


Fig. 1. Roentgenograms of 4 different injections. Left anterior oblique projection. Left coronary artery. (*A*) Following the initial test dose the catheter tip has been removed from the left coronary artery and placed in the left coronary cusp. A crescent shaped collection of contrast substance persists in the epicardiac surface of the heart (solid arrows). It is not in close proximity to the left aortic cusp. (*B*) A second injection shows its relation to the branches of the left coronary artery, which appear otherwise normal. (*C*) The walls of the diagonal branch are seen as a negative shadow outlined by the contrast substance outside (open arrows) and inside of the vessel. (*D*) The amount of extravascular contrast substance increased following the next injection.

tracings remained stable. The entire left coronary system was well visualized and revealed no significant obstructing lesions (Fig. 2, *A–C*). The aortic valves were heavily calcified. Finally a small injection was again made into the left coronary cusp in order to evaluate its relationship to extravasated contrast substance.

Several attempts were made to catheterize the right coronary artery but they were unsuccessful.

The extravasated contrast material was reabsorbed rapidly and 30 minutes after the completion of the examination it had disappeared almost completely. (Fig. 2*D*). The patient still complained of only a slight soreness in the chest. A 12 lead standard electrocardio-

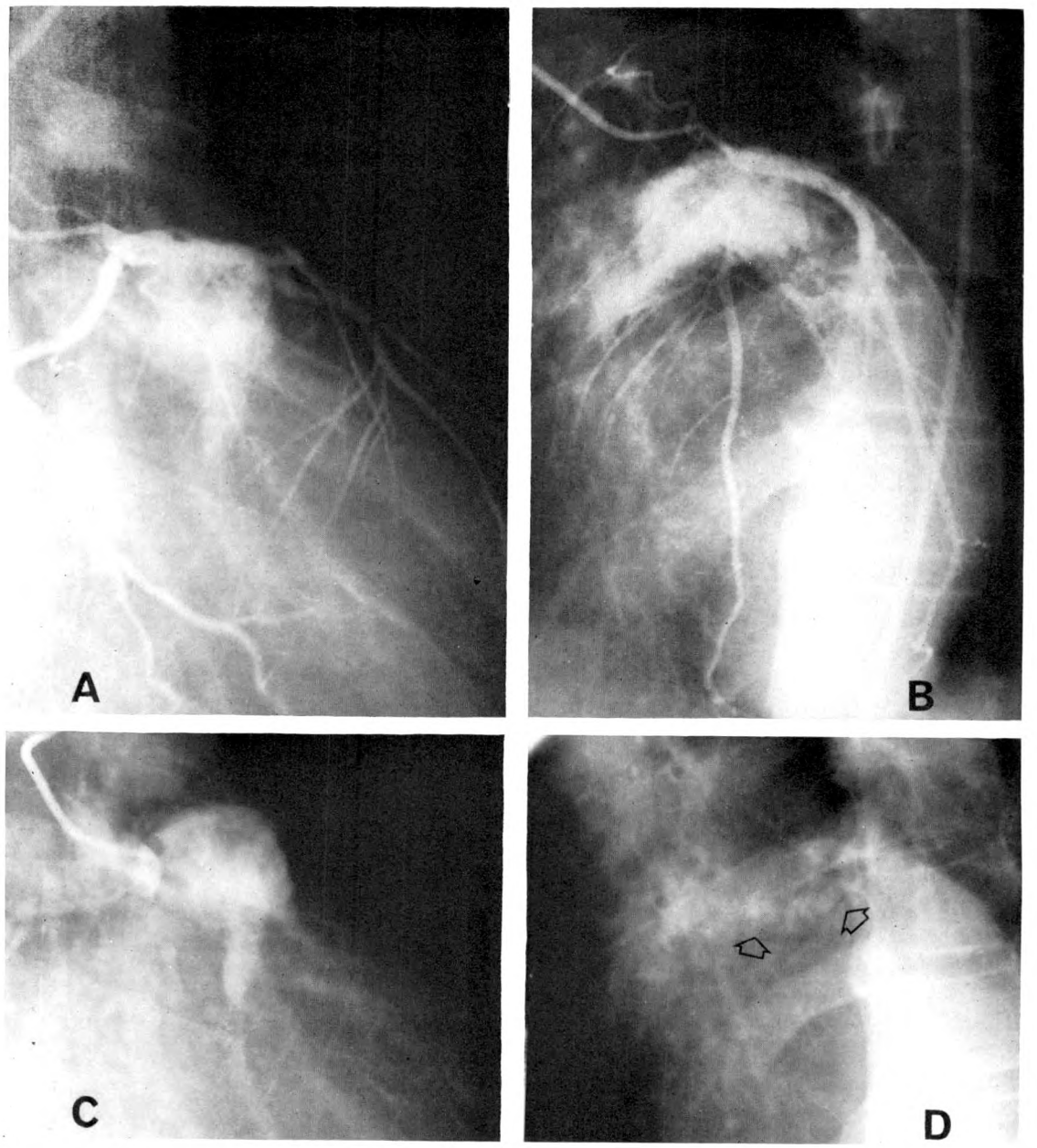


FIG. 2. (*A*) Right anterior oblique projection. (*B*) Left anterior oblique projection. The size of the opaque collection continues to increase and extends anteriorly. (*C*) The left aortic cusp is outlined and intact. (*D*) Spot roentgenogram obtained 30 minutes after the completion of the examination. The contrast substance has been almost completely reabsorbed. A small collection still remains (open arrows).

gram was done which revealed no changes from tracings obtained before the procedure. Physical examination and laboratory data including enzyme studies also remained unchanged.

The patient underwent open heart surgery on November 21, 1969, at which time the aortic valve was replaced by a Starr-Edwards prosthetic valve. At operation a flat collection of clotted blood of approximately 8 cm. in diameter was noted in the epicardiac surface of

the heart after elevating the left atrial appendage. It was centered around the area of the bifurcation of the left coronary artery. The tissues were somewhat friable in the region of the partially organized hematoma. Because of local conditions no attempt was made to identify the actual site of the extravasation. There was no evidence of blood or adhesions in the pericardial cavity. The aortic valve cusps and the immediate supravalvular portion of the aorta were carefully inspected and failed to reveal any evidence of perforation or intimal damage.

DISCUSSION

Coronary arteriography by the percutaneous femoral approach is gaining increasing popularity. The ease with which intubation of the coronary arteries is achieved as well as the stability of the catheters within the orifices of the coronary arteries is responsible for this increasing popularity. The over-all incidence of complications by this method does not appear to be different than with the method originally described by Sones and Shirey.[3] A report of 2 cases of subintimal injection in the left coronary artery also occurring with the percutaneous femoral technique has recently appeared.[1] Both of these cases had significant immediate complications consisting of chest pain, ventricular fibrillation and signs of myocardial damage. Our case differs from those previously reported in that there was extravasation rather than subintimal injection of the contrast material. What is most remarkable is that no untoward effects from such extravasation were apparent, either immediately or following the examination. The absence of symptoms and serious effects is due to the fact that the extravasation, unlike a subintimal injection, did not result in a significant reduction of the caliber of the involved vessel. Bleeding was apparently self limited and of small magnitude. It should also be noted that the examination was carried to completion, providing important information regarding the absence of hemodynamically significant lesions of the coronary system which may have altered a surgical procedure. This is the first time that we encountered a local complication in approximately 200 cases of selective coronary arteriography. For the past 2 years we have been utilizing mechanical injections with pressures of 150 psi. With our injection system, this allows the delivery of approximately 6 cc. of contrast substance in 1 second. Although we cannot exactly determine the cause of the perforation, we suspect that the considerable elasticity and plastic memory of the catheter may have exerted sufficient force to cause the perforation. No structural abnormalities of the walls of the coronary arteries were noted. The presence of aortic insufficiency may have contributed to the perforation. The wide pulse pressure and the increased stroke volume with the resulting bounding pulsations of the aorta and the coronary artery could conceivably have exerted unusual stresses upon the catheter and the wall of the coronary vessels so as to cause excessive penetration and perforation. Inadvertent perforation by the wire used for the insertion of the catheter can be ruled out in this case since it was removed under fluoroscopic control while the catheter tip was located high in the ascending aorta.

We find selective coronary arteriography by the percutaneous femoral technique using the catheters introduced by Judkins a very valuable and much simpler method than others we have tried, but we would like to call attention to the occasional excessive penetration of the catheter tip within the lumen of the main coronary artery. This penetration tends sometimes to carry the tip of the catheter rather forcefully into the anterior descending or circumflex branches. This tendency, aided by the force with which the loop of the left coronary catheter tries to resume its natural closed configuration, may be sufficient to traumatize the coronary vessels. This case illustrates one of the possible complications

from these characteristics of the left coronary catheter.

SUMMARY

A case is reported in which perforation of a branch of the left coronary artery occurred during selective coronary arteriography. The mishap was remarkably uneventful and not attended by any complications. Surgical confirmation of the perforation of the coronary artery was obtained.

The probable causes and their relation to the patient's condition and the physical properties of the catheters are discussed.

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Fifty-third Annual Meeting: Maria Isabel Hotel, México, D.F., México, March 15–18, 1971.

EDITORIALS

THE NEURORADIOLOGIST AND THE EAR

RECENT advances in Neuroradiology have included excellent radiographic visualization of fine arterial and venous structures of the posterior fossa. Hypocycloidal tomography with cuts at 1 mm. intervals enabled Valvassori[1] to pioneer the rebirth of knowledge of the anatomy of the ear by roentgenographic methods. Using the Polytome, he showed the importance of anteroposterior and lateral views to demonstrate the internal auditory canal and meatus, whereas a slight obliquity of the petrous bone was optimal for demonstration of the cochlear and oval window. Hanafee *et al.*[2] recently emphasized tomography, using the basal projection, to adequately demonstrate the internal auditory canal.

Regardless of which projection or tomographic device is employed, a kvp. in the range of 60–75 and a relatively high mas. are necessary to achieve good detail.

The Neuroradiologist, although well versed in the pneumography and arteriography of the posterior fossa, has begun to incorporate this additional otologic study in his methodology. Thus he is combining two separate disciplines and hopefully will make advances that a radiologist without a neurologic background would find difficult to achieve.

Otologic problems, such as cholesteatomas associated with perforation and otosclerosis, are important to recognize radiographically, especially in establishing a differential diagnosis between conductive and neurosensory hearing loss. Otologists state that these diseases are easily diagnosed clinically, but the extent of these lesions, as regards to attic or antral involvement by cholesteatomas, or cochlear and ossicular damage by otosclerosis, is well documented by tomography. Also, the best definition of the jugular foramen is obtained by anteroposterior and lateral tomograms of the petrous bones at the level of the posterior semicircular canals.

Jugular venography and carotid angiography will assist in evaluating glomus jugulare tumors.

An important field in which the Neuroradiologist has a decisive role is the early detection of an acoustic neuroma. This requires a special technique. Valvassori[3] as well as Hitselberger and Hause[4] have adequately described this technique which necessitates the use of a small amount of pantopaque inserted into the lumbar subarachnoid space. The patient is placed on the roentgenographic table in the lateral decubitus position with the involved side down, tilting him approximately 35° Trendelenburg, in order to fill the involved cerebellopontine angle cistern, to visualize the lesion.

Wilner and co-workers[5] have recently published a series, using 3 ml. of pantopaque injected into the lumbar subarachnoid space; after maneuvering the opaque material under fluoroscopic control into the abnormal cerebellopontine angle, they have obtained cross-table anteroposterior and supine tomograms of the ear structures.

Many additional observations concerning the diagnosis of cerebellopontine angle tumors are noted. Acoustic neuromas which are intracanalicular or slightly extracanalicular may cause substantial neurosensory

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hearing loss, without producing elevation of cerebrospinal fluid protein. Patients may have neurosensory hearing loss without tinnitus or vertigo and still have a small acoustic tumor. These tumors can be found in adolescent or adult patients without any other clinical signs of neurofibromatosis.

The simplicity of the use of 1–3 ml. of the positive contrast material in diagnosing small acoustic neuromas, makes this the method of choice in our experience. This small amount prevents any loss of visualization of detail of the meatus or canal. One can quantitate the size of the extracanalicular portion of the tumor, and thus aid in the surgical approach made by the neurosurgeon or otologist. The study can be obtained on an outpatient basis, with the use of steroids given to the patient following the test being helpful in preventing headaches. The small amount of contrast material collects and remains in the lumbar subarachnoid space, permitting the restudy of suspected cases that are not diagnosed initially. We still do not know the exact growth rate of an acoustic neuroma, although no report of a tumor appearing within one year after an initial negative study is found in the literature.

Fluoroscopic control is essential in maneuvering the contrast material from the lumbar subarachnoid space into the affected cerebellopontine angle. This is to prevent confusion with a high spinal cord tumor, low brainstem or anterior cerebellar mass which may present with 8th nerve involvement.

Acoustic neuromas are usually smooth, rounded tumors and leave a similar impression upon the positive contrast material. Recently, we have noted a case where the normal side filled with contrast material, but the clinically abnormal side had no contrast material enter the affected cerebellopontine angle region. At surgery arachnoidal adhesions were found on the abnormal side. Thus arachnoiditis can occur unilaterally as well as bilaterally, and is a definite pitfall in interpretation. All cases in which a smooth cap was present, had a tumor. Bilateral tumors are very rare, and if neither cerebellopontine angle cistern fills with contrast material, arachnoiditis is more likely than bilateral tumors. We have had no false negative results.

Angiography has helped mostly in excluding a cerebellopontine angle meningioma and in visualizing the anterior inferior cerebellar artery which may be displaced by a mass near or in the acoustic canal. A small cerebellopontine angle tumor may not show a significant arteriographic change.

Pneumoencephalography with accompanying tomography of the cerebellopontine angle cisterns is a worthwhile substitute or adjunct to the early diagnosis. The morbidity is usually greater, however, and outpatient as well as follow-up studies on questionable intracanalicular lesions, are not obtainable.

The choice of type of contrast study obviously depends on the experience of the physician. A more difficult problem is whether a patient with a negative tomogram of the canal, should have any further studies? Conversely, should a patient showing a positive tomogram with destruction or enlargement of the canal, have further investigation?

Rarely do we proceed with a contrast study in the absence of positive plain film roentgenographic changes unless the clinical evidence is extremely suspicious. On the other hand, some cases exhibiting very wide internal canals, even with asymmetric enlargement, have had negative contrast studies. The extent of the mass is also determined by the contrast study.

In summary, the past several years have shown definite progress in the early diagnosis of cerebellopontine angle tumors, as a result of increased knowledge of the radiographic determination of ear pathology. The trained Neuroradiologist, skilled in the use of contrast material, contributes immeasurably to this important progress.

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DOUGLAS SHELDON KELLOGG, M.D.
1899-1969

DR. DOUGLAS S. KELLOGG, retired Colonel, U. S. Army Medical Corps, died on October 20, 1969 at the age of 70.

Dr. Kellogg was born May 3, 1899 in Minneapolis, Minn. He received his preliminary education in Sioux City, Iowa and obtained his B.S. degree in 1923 from the University of Illinois. He received his M.D. degree from the University of Illinois College of Medicine in 1925 and served his internship at the Seattle General Hospital in 1924–1925.

After a competitive examination, he received a regular army commission as 1st Lieutenant, Medical Corps in 1925. He graduated from Medical Field Service School at Carlisle Barracks, Pennsylvania, in 1926. His first military assignment was at Letterman General Hospital, San Francisco, California. He had overseas tours of duty in Hawaii, Germany and New Guinea, and during World War II was Commanding Officer of the 35th General Hospital, stationed in New Guinea.

From 1925 to 1943 he had been promoted through the ranks from 1st Lieutenant to Colonel and received numerous decorations during his military career.

Like many of the old-time radiologists, Dr. Kellogg was attracted to Radiology while in the military service, and received his radiology residency training under the tutelage and preceptorship of Dr. John J. Moore at Letterman General Hospital, San Francisco, California, from 1926–1927. A pioneer military radiologist, Dr. Kellogg was mainly interested in diagnostic radiology, applied all of his efforts in this area, and contributed freely to the radiologic literature. An excellent teacher, he was instrumental in developing the radiology residency programs in the U. S. Army Hospitals and served as teacher and preceptor to many of the radiologists who had their training at the Fitzsimmons General Hospital, Denver, Colorado. Dr. Kellogg was a keen diagnostician who emphasized the differential diagnostic approach in the interpretation of roentgenograms. After retirement from the military service, he continued his radiologic work as a radiologist with Radiology Associates in San Antonio, Texas. He was a consultant to the Texas State Mental Hospital in San Antonio, and to the Randolf Field Base Hospital, San Antonio.

Dr. Kellogg was an active member of many medical and radiologic societies and was a member of the A.M.A., Texas Medical Association, Bexar County Medical Society, American Roentgen Ray Society, Association of Military Surgeons of the U. S., a Diplomate of the American Board of Radiology, a Fellow and a member of the Phi Rho Sigma Medical fraternity. For many years he was an eminent abstract editor in the Department of Abstracts of the Am. J. Roentgenology, Rad. Therapy & Nuclear Med. This work he did with great distinction and enthusiasm, until he died.

Dr. Kellogg was a member of the Christian Church. He married Juanita W. Bower in Des Moines, Iowa, who survives him and is survived by a son Dr. Douglas S. Kellogg, Jr. of Atlanta, Ga. and 3 grandsons, Thomas Merritt, James Douglas and Paul. He was a devoted family man and loved to play golf. One of Dr. Kellogg's biggest thrills was his "hole in one" in 1940, at the Fort Sam Houston Golf Course in San Antonio. Dr Kellogg was playing golf with his devoted wife, Juanita, when he had his fatal heart attack on October 20, 1969. He was buried in the Fort Sam Houston National Cemetery, with full military honors.

Dr. Kellogg worked and played throughout his life, with love and devotion. A kindly, quiet and unassuming giant in the field of radiology, he will be missed by all of his colleagues, and by his many friends who are scattered all over the world.

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NEWS ITEMS

THE AMERICAN RADIUM SOCIETY RESIDENT-FELLOW AWARD

The membership of the American Radium Society takes pleasure in announcing a perennial award to be presented at the Annual Meeting of the Society for the best paper submitted to the Resident Award Committee.

The purpose of the award is to stimulate Residents and Fellows who are preparing for a career in the field of oncology to participate either in clinical or fundamental investigations in cancer research, and to further acquaint those entering this field with the functions of the American Radium Society.

1. The Award consists of travel and maintenance expenses for the trip to the Annual Meeting to be held in Mexico City, March 15–18, 1971 plus a certificate of Award which will be presented to the Author at the meeting.

2. The paper must be submitted by a Resident or Fellow working with or sponsored by a member of the American Radium Society.

3. The paper must deal with some phase of cancer research. It may be a report on clinical studies of patients with cancer, basic research on some aspect of cancer, or a relevant theoretic dissertation.

4. The paper must be read by the Resident or Fellow at the Annual Meeting of the American Radium Society.

5. The paper must be written by and represent primarily the work of the Resident or Fellow although he may list one or more co-authors. There should be two title pages, one containing the name(s) of the author(s) and their institution, the other the title only. The work should be current but need not have been completed entirely during this year.

6. A person who has already completed his residency may be a candidate if the work was done during his residency training and he is no more than one year post residency at the time of the Annual Meeting.

7. Papers published elsewhere are not acceptable as entries for the award.

8. An original and 4 copies of the complete paper must be sent to the Chairman of the Resident Award Committee. A 200 word abstract which can be included in the Program for the Annual Meeting must be included. Each author will receive a postcard acknowledging receipt of the paper.

Manuscripts must be submitted by November 15, 1970 to the Chairman of the Resident Award Committee, Malcolm A. Bagshaw, M.D., Professor and Director, Division of Radiation Therapy, Stanford University School of Medicine, Stanford, California 94305.

SOUTHERN MEDICAL ASSOCIATION RADIOLOGY REFRESHER COURSES, POSTGRADUATE INSTRUCTION

The Sixty-fourth Annual Meeting of the Southern Medical Association will be held in Dallas, Texas, November 16–18, 1970. At that time the Radiological Society of North America, in cooperation with the Section on Radiology of the Southern Medical Association, and as a part of a continuing program in Postgraduate Education, will sponsor 5 refresher courses.

Two of the courses will be presented by James J. McCort, M.D., San Jose, California, "Roentgenology of the Acute Abdomen Including Trauma"—Parts I & II. The other 3 courses will be: Edward B. Singleton, M.D., Houston, Texas, "Urinary Tract Problems of the Pediatric Patient," John A. Kirkpatrick, M.D., Philadelphia, Pennsylvania, "Disorders of Aeration in the Pediatric Patient," and Eleanor D. Montague, M.D., Houston, Texas, "Radiotherapy in the Definitive Management of Breast Cancer."

The instruction sessions will be held at the Convention Center in Dallas on Mon-

day, November 16 from 1:30 P.M. to 5:00 P.M.; Tuesday, November 17 from 8:30 A.M. to 12:00 NOON; and Wednesday, November 18 from 1:30 P.M. to 3:00 P.M.

Attendance is limited to physicians, including graduate students and residents in radiology, and medical students certified by the Dean of their medical school. There will be no registration fee for these courses but all who attend must be registered at the meeting so that they may be properly identified.

Any comments concerning the courses or suggestions for future presentations should be directed to the Chairman of the Refresher Course Committee: Hillier L. Baker, Jr., M.D., Mayo Clinic, Rochester, Minnesota 55901.

POST GRADUATE COURSE IN ANGIOGRAPHY, SELECTION OF X-RAY EQUIPMENT AND TECHNIQUES

This course, sponsored by the Department of Diagnostic Radiology, University of Alabama School of Medicine, will be held Friday and Saturday, October 2 and 3, 1970, in the Engineering Auditorium, 1919 South 8th Avenue, Birmingham, Alabama.

The Course Directors are Alberto Barcia, M.D., and Benigno Soto, M.D.

The Guest Faculty includes: Herbert L. Abrams, M.D., Boston, Massachusetts; William M. Angus, M.D., Ph.D., Philips Medical Systems, New York, New York; Joseph J. Bookstein, M.D., Ann Arbor, Michigan; George D. Davis, M.D., Rochester, Minnesota; Oskar G. Dunisch, Dr. med. h.c., Siemens Corporation, Erlangen, Germany; Melvin M. Figley, M.D., Seattle, Washington; Harry W. Fischer, M.D., Eloise, Michigan; George Fredzell, Ph.D., Elema Schönander, Stockholm, Sweden; Donald E. Graves, General Electric Medical Systems, Milwaukee, Wisconsin; Franz J. Hallermann, M.D., Ph.D., Corpus Christi, Texas; Daniel G. Lawrence, Eastman Kodak Corporation, Rochester, New York; Sven Paulin, M.D., Boston, Massachusetts; Manfred Pheiler, Ph.D., Siemens Corporation, Erlangen, Germany; Manuel Viamonte, Jr., M.D., Miami Beach, Florida; and Robert E. Wayrynan, Ph.D., E. I. DuPont de Nemours & Company, Inc., Wilmington, Delaware.

The Local Faculty comprises: Alberto Barcia, M.D., Norman P. Deason, Sang Y. Han, M.D., William B. Jones, M.D., Robert B. Karp, M.D., John W. Kirklin, M.D., Clifton K. Meador, M.D., T. Joseph Reeves, M.D., and Benigno Soto, M.D.

On Friday, October 2, at 5:00 P.M. there will be a tour through the University of Alabama Angiocardiographic Laboratories located in the Sixth Floor of Hillman Building, 619 South 20th Street.

For further information please contact Dr. Alberto Garcia, Director, Division of Cardiac Radiology, University of Alabama in Birmingham, 619 South Nineteenth Street, Birmingham, Alabama 35233.

POSTGRADUATE COURSE IN NEURORADIOLOGY

A Postgraduate Course in Neuroradiology will be held under the direction of Juan Taveras, M.D., the Mallinckrodt Institute of Radiology, Washington University of Medicine, St. Louis, Missouri, October 5–9, 1970.

This course is designed for radiologists, neurologists, and neurosurgeons. The course will follow a didactic approach and will include discussions on technical, anatomic, physiologic and pathologic considerations as well as recent developments in the selected major topics.

In addition, there will be a special half day session at which will be discussed all recent developments in neuroradiology reported at the triennial International Neuroradiology Symposium taking place this summer in Gothenburg, Sweden.

The Faculty includes: Sten Cronqvist, M.D., Lund, Sweden; David O. Davis, M.D., St. Louis, Missouri; Herbert I. Goldberg, M.D., Philadelphia, Pennsylvania; Sadek K. Hilal, M.D., New York,

New York; Edward Ralph Heinz, M.D., Pittsburgh, Pennsylvania; Irvin I. Kricheff, M.D., New York, New York; William H. McAlister, M.D., St. Louis, Missouri; Howell S. McCreary, M.D., St. Louis, Missouri; D. Gordon Potts, M.D., New York, New York; Mannie M. Schechter, M.D., New York, New York; Juan Taveras, M.D., St. Louis, Missouri; and Ernest H. Wood, M.D., New York, New York.

All lectures will be given in the convention facility of Stouffer's Riverfront Inn.

For further information and registration please apply to Juan Taveras, M.D., Director, Mallinckrodt Institute of Radiology, Washington University School of Medicine, 510 South Kingshighway, St. Louis, Missouri 63110.

RADIOLOGY IN OTOLARYNGOLOGY AND OPHTHALMOLOGY

The Department of Radiology of the University of Illinois, with Galdino E. Valvassori, M.D. as Faculty Coordinator, is presenting the Fourth Annual Conference on Radiology in Otolaryngology and Ophthalmology at the Illinois Eye and Ear Infirmary, University of Illinois at the Medical Center, Friday and Saturday, November 27 and 28, 1970.

The course will include lectures and visual representations by a distinguished faculty.

Enrollment will be limited.

Further information may be obtained by writing to Division of University Extension, University of Illinois at the Medical Center, 715 S. Wood, Chicago, Illinois 60612.

SYMPOSIUM ON CISTERNOGRAPHY AND HYDROCEPHALUS

The Divisions of Nuclear Medicine and Neurologic Surgery at Georgetown University Hospital will sponsor a 2 day symposium on radioisotope cisternography and hydrocephalus at the Statler Hilton Hotel, Washington, D.C., May 6–7, 1971.

This symposium will bring together basic scientists and clinicians interested in cisternography and hydrocephalus to share the most recent concepts in both areas; to define problems in current cisternographic procedures, scan interpretation and clinical problems which can be resolved by cisternographic procedures; and to outline future research directions.

The symposium will be organized according to: (1) *Basic Physiology*—Studies relating to the formation, flow and reabsorption of cerebrospinal fluid and the alterations which take place in hydrocephalus. (2) *Diagnosis*—New techniques, instrumentation and radiopharmaceuticals. Special problems in computer techniques and dosimetry will be covered. Comparison between radioisotope and roentgenographic procedures will be reviewed. (3) *Clinical Correlations with Cisternographic Findings*—Interpretations in both pediatric and adult hydrocephalus. The evaluation of shunts and the changing dynamics of shunting will be covered. Of special importance will be the prognostic significance of cisternography.

Outstanding investigators from the United States and abroad have been invited to keynote the major areas of discussion. Submitted papers dealing with these topics are now being received. A complete collection of papers from the Symposium will be published.

Abstracts should be sent before December 1, 1970, to John C. Harbert, M.D., Division of Nuclear Medicine, Georgetown University Hospital, Washington, D.C. 20007.

BOOKS RECEIVED

ANATOMY AND PHYSIOLOGY FOR RADIOGRAPHERS. By C. K. Warrick, M.B., B.S. (Lond.), M.R.C.P., F.R.C.S.(E), F.F.R., D.M.R., Radiologist-in-Charge, Royal Victoria Infirmary, Newcastle upon Tyne; Lecturer in Radiology and Radiological Anatomy, University of Newcastle upon Tyne: Advisor in Radiology to No. 1 Regional Hospital Board and Honorary Advisor in Radiology to Sierra Leone. Third edition. Cloth. Pp. 286, with many figures. Price $9.00. Edward Arnold (Publishers) Ltd., London. Exclusive U. S. agent, The Williams & Wilkins Company, Baltimore, Md. 21202, 1969.

OSTEOPOROSIS. Edited by Uriel S. Barzel, M.D., Montefiore Hospital and Medical Center, Bronx, N. Y. Cloth. Pp. 290, with many figures. Price, $25.00. Grune & Stratton, 757 Third Avenue, New York, N. Y. 10017, 1970.

MEDICAL RADIONUCLIDES: RADIATION DOSE AND EFFECTS. Proceedings of a symposium held at the Oak Ridge Associated Universities, Dec. 8–11, 1969. Sponsored by U. S. Public Health Service, Environmental Health Service, Bureau of Radiological Health and U. S. Atomic Energy Commission, Division of Biology and Medicine. Edited by Roger J. Cloutier, C. Lowell Edwards, and Walter S. Snyder. AEC Series No. 20. Paper. Pp. 528, with some figures. Price, $3.00. Clearinghouse for Federal Scientific and Technical Information, Springfield, Va. 22151, 1970.

MORTALITY FROM MALIGNANT NEOPLASMS, 1955–1965. World Health Organization, Geneva. In two parts. Bilingual edition (English and French). Paper. Pp. 1,147. Price, $36.00. The American Public Health Association, Inc., 1740 Broadway, New York, N. Y. 10019, 1970.

A CROSS-SECTION ANATOMY. By Albert C. Eyeleshymer, B.S., Ph.D., M.D., Late Professor of Anatomy, St. Louis University; and Daniel M. Schoemaker, B.S., M.D., Late Associate Professor of Anatomy, St. Louis University. Drawings by Tom Jones, Late Instructor in Drawing, St. Louis University. Cloth. Pp. 215, with many drawings. Price, $35.00. Appleton-Century-Crofts, 440 Park Avenue South, New York, N. Y. 10016, 1970.

BONE MINERAL METABOLISM IN CANCER. Recent Results in Cancer Research; Volume 27. By Janusz Szymendera, M.D., Research Assistant in Nuclear Medicine, Department of Isotopes, Institute of Oncology, Warsaw, Poland. Cloth. Pp. 128, with 43 figures. Price, $8.80. Springer-Verlag New York, 175 Fifth Avenue, New York, N. Y. 10010, 1969.

TUMORS OF THE LIVER. Recent Results in Cancer Research: Volume 26. Edited by George T. Pack, M.D., F.A.C.S., Late Attending Surgeon Emeritus, Memorial Hospital for Cancer and Allied Diseases; Clinical Professor of Surgery, New York Medical College; and Abdol H. Islami, Ph.D., M.D., F.A.C.S., Attending Surgeon and Director of Department of Medical Education, St. Barnabas Medical Center, Livingston, N. J.; Assistant Clinical Professor of Surgery, New Jersey College of Medicine and Dentistry, Jersey City, N. J. Cloth. Pp. 270, with 175 figures. Price, $15.40. Springer-Verlag New York, 175 Fifth Avenue, New York, N. Y. 10010, 1969.

RADIOACTIVE NUCLIDES IN MEDICINE AND BIOLOGY. Basic Physics and Instrumentation. By Edith H. Quimby, Sc.D., Professor Emeritus of Radiology, College of Physicians and Surgeons, Columbia University; Sergei Feitelberg, M.D., Late Director, Andre Meyer Department of Physics, The Mount Sinai Hospital; and William Gross, Ph.D., Associate Professor of Radiology (Physics), College of Physicians and Surgeons, Columbia University, New York, N. Y. Third edition. Cloth. Pp. 390, with 94 figures. Price, $12.50. Lea & Febiger, 600 S. Washington Square, Philadelphia, Pa. 19106, 1970.

THE PHARYNGOESOPHAGEAL SPHINCTER. By Costantino Zaino, M.D., Associate Clinical Professor of Radiology, New York University Medical Center; Research Associate, Division of Diagnostic Radiology, Montefiore Hospital and Medical Center; Roentgenologist, Union Hospital; Harold G. Jacobson, M.D., Chief, Division of Diagnostic Radiology, Montefiore Hospital and Medical Center; Professor of Radiology, Albert Einstein College of Medicine; Harold Lepow, M.D., Associate Professor of Pathology, Albert Einstein College of Medicine; Pathologist and Director of Laboratories, Lincoln Hospital; and Cahit H. Ozturk, M.D., Attending Surgeon, Neillsville Memorial Hospital; Surgeon, Neillsville Clinic Group, Wisconsin. Cloth. Pp. 209, with many illustrations. Price, $18.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1970.

EXERCISES IN DIAGNOSTIC RADIOLOGY. I: The Chest. By Lucy Frank Squire, M.D., Lecturer, Harvard Medical School; Visiting Radiologist, Massachusetts General Hospital, Boston, Mass.; William M. Colaiace, M.D., Lecturer in Medical Science, Brown University; Radiologist, Roger Williams General Hospital, Providence, R. I.; and Natalie Strutynsky, M.D., Assistant Professor, Radiology, New York Medical College, New York, N. Y. Paper. Pp. 86, with many figures. W. B. Saunders Company, West Washington Square, Philadelphia, Pa., 1970.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual meeting: Deauville Hotel, Miami Beach, Fla. September 29–October 2, 1970.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Jerome M. Vaeth, Saroni Tumor Institute, 1600 Divisadero St., San Francisco, Calif. 94115. Annual meeting: Mexico City, Mexico, March 15-18, 1971.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary-Treasurer, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., November 29–December 4, 1970.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: St. Louis, Mo., Chase-Park Hotel, March 30–April 3, 1971.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga., 30322. Annual meeting: Atlantic City, N. J., June 20–24, 1971.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Denver, Colo., Dec. 7–11, 1970, Denver-Hilton Hotel; Bal Harbour, Fla., June 7–11, 1971, Americana Hotel; and Dallas, Tex., Dec. 6–10, 1971, Statler-Hilton Hotel.

Written examinations are scheduled in June of each year in 12 large centers, and applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Beginning in 1970 the deadline for receiving applications for any examination is Sept. 30 of the year preceding.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, Leonard Stanton, Hahnemann Medical College, 230 N. Broad St., Philadelphia, Pa. 19102. Annual meeting to be announced.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Carl R. Bogardus, Jr., University of Oklahoma Medical Center, Oklahoma City, Oklahoma 73104 Fall meeting: Mountain Shadows, Scottsdale (Phoenix), Ariz., November 11-13, 1970.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, F. J. Fry, M.Sc., Bioacoustics Lab., University of Illinois, Urbana, Ill.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary-Treasurer, Dr. Eugene V. Leslie, Edward J. Meyer Memorial Hospital, 462 Grider St., Buffalo, N. Y. 14215.

THIRTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Meeting: Madrid, Spain in Fall 1973.

TENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Manuel Viamonte, Jr., University of Miami School of Medicine, Jackson Memorial Hospital, Miami Fla. 33136.
President, Dr. Victor A. Marcial, Puerto Rico Nuclear Center, Caparra Heights Station, San Juan, Puerto Rico 00935.
Meeting: San Jeronimo-Hilton Hotel, San Juan, Puerto Rico, May 16-22, 1971.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.

ALABAMA CHAPTER OF ACR
Secretary, Dr. William V. Weldon, Medical Arts Building, Birmingham, Ala. 35205. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY
Secretary, Dr. Bruce C. Wright, Providence Hospital, Anchorage, Alaska. Meets third Wednesday each month.

ARIZONA RADIOLOGICAL SOCIETY
Secretary, Dr. Wesley S. Fee, 2421 E. 6th St., Tucson, Ariz. 85719. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS CHAPTER OF AMERICAN COLLEGE OF RADIOLOGY
Secretary-Treasurer, Dr. Wilma C. Diner, Univ. of Arkansas Medical Center, Little Rock, Ark. 72201. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Elliott C. Lasser, University Hospital of San Diego County, San Diego, Calif. 92103. Annual Meeting: Durham, N. C., May 13–15, 1971, with the Duke University and University of North Carolina Radiology Departments serving, as co-hosts.

ATLANTA RADIOLOGICAL SOCIETY
Secretary, Dr. Richard S. Colvin, Emory University Clinic, Atlanta, Ga. 30322. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Advisor, Colonel Paul E. Sieber. *Secretary*, LTC Peter B. Riesz, USAH Bad Cannstatt, APO 09154, New York, N. Y. Meets quarterly.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. R. John Gould, 41 Lombardy Rd., Drexel Hill, Pa. 19026.

BLUEGRASS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Joseph A. Sayeg, Ph.D., Radiation Physicist, University of Kentucky, Lexington, Ky. 40506. The Society meets once each month during the school year.

BRONX RADIOLOGICAL SOCIETY, NEW YORK STATE, CHAPTER ACR
Secretary-Treasurer, Dr. David Bruce Hayt, 600 E. 233rd St., Bronx, N. Y. 10466. Meets 4 times a year.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Rubem Pochaczevsky, 300 Skillman Ave., Brooklyn, N.Y. 11211. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Herbert Pirson, 145 High Park Blvd., Buffalo, N.Y. 14226. Meets second Monday evening each month, October to May inclusive, at University Club.

CALIFORNIA RADIOLOGICAL SOCIETY, CALIFORNIA CHAPTER ACR
Secretary-Treasurer, Dr. John L. Gwinn, Childrens Hospital of Los Angeles, P.O. Box 54700, Los Angeles, Calif. 90054.

CATAWBA VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Emmett R. White, P. O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 P.M.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Central New York Radiological Society
Secretary-Treasurer, Dr. David N. Cheris, Community General Hospital of Greater Syracuse, Broad Road, Syracuse, N. Y. 13215. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary, Dr. Robert J. Mounts, 4520 Summit Ridge Rd., Columbus, Ohio 43220. Meets second Thursday in October, November, January, and March 15 and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Chicago Roentgen Society
Secretary-Treasurer, Dr. William T. Moss, 250 E. Superior St., Chicago, Ill. 60611. Meets third Thursday of each month, October to April, except December, at the Bismarck Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. James Farmer, 18869 Canyon Rd. Fairview Park, Ohio 44126. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society
Secretary, Dr. Marvin L. Daves, Univ. of Colorado Medical Center, 4200 E. Ninth Ave., Denver, Colo. 80220. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. V. V. Kirby, 1722 Spring Lake Dr., Arlington, Tex. 76010. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

East Bay Radiological Society
Secretary-Treasurer, Dr. S. Walter Kran, Doctors' Hospital of San Leandro, 13855 East 14th St., San Leandro, Calif. 94578. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. Eberhard F. Besemann, Baroness Erlanger Hospital, Chattanooga, Tenn. 37403. Meets in January and September.

Florida Radiological Society
Secretary, Dr. Wm. F. Lindsey, 1215 Hodges Dr., Tallahassee, Fla. 32303. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Allen L. Sheer, University Community Hospital, 13505 N. 31st St., Tampa, Fla. 33612. Meets in January, March, May, July, September and November.

Georgia Radiological Society
Chapter of the American College of Radiology
Secretary, Dr. Walker Harris, The Medical Center, Columbus, Ga. 31902. Meets in spring and fall at Annual State Society Meeting.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. John Kathe, North Shore Hospital, Miami, Fla. 33150. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. Roland P. Ernst, 3720 Washington Ave., St. Louis, Mo. 63108.

Hawaii Radiological Society
Secretary-Treasurer, Dr. Virgil R. Jobe, Jr., 888 South King St., Honolulu, Hawaii 96813 Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, R. J. Augustine, National Center for Radiologic Health, 1901 Chapman Ave., Rockville, Md. 20852. Annual meeting to be announced.

Houston Radiological Society
Secretary, Dr. Kenneth M. Jensen, 1615 St. Joseph Prof. Bldg., Houston, Texas 77002. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025

Idaho State Radiological Society
Secretary, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the spring and fall.

Illinois Radiological Society, Inc.
Secretary, Dr. C. R. Markivee, 674 North Seminary St., Galesburg, Ill. 61401. Meets in the spring and fall.

Indiana Roentgen Society, Inc.
Secretary, Dr. Dale B. Parshall, Elkhart General Hospital, Elkhart, Ind. 46514.

Iowa Radiological Society
Secretary-Treasurer, Dr. John Huston Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society
Secretary-Treasurer, Dr. Robert C. Lawson, 310 Medical Arts Bldg., 10th and Horne, Topeka, Kan. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter, American College of Radiology
Secretary-Treasurer, Dr. Ralph C. Quillin, 1221 S. Broadway, Lexington, Ky. 40504. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N.Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Harold L. Atkins, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Arthur W. Reiter, Saint Luke Hospital, 2632 East Washington Blvd., Pasadena, Calif. 91107. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

Maine Radiological Society
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. Nathan Stofberg, 4519 Hawksbury Rd., Pikesville, Md. 21208.

Massachusetts Radiological Society
Secretary, Dr. Jerome H. Shapiro, Department of Radiology, Boston City Hospital, 818 Harrison Ave., Boston, Mass. 02118.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Webster Riggs, Jr., The University of Tennessee College of Medicine, Department of Radiology, Walter F. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Ind. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary, Dr. David P. Corbett, Harper Hospital, Detroit, Mich. 48201. Meets monthly, first Thursday, Octo-

ber through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary-Treasurer, Dr. James E. Bell, 8700 W. Wisconsin Ave., Milwaukee, Wis. 53213. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Warren L. Kump, 4243 Glenwood Ave., Minneapolis, Minn. 55422. Meets twice annually, fall and winter.

MISSISSIPPI STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ottis G. Ball, 5356 Balmoral Drive, Jackson, Miss. 39211. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

MISSOURI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Arthur A. Porporis, 100 N. Euclid Ave., St. Louis, Mo. 63108.

MONTANA RADIOLOGICAL SOCIETY
Secretary, Dr. Colvin H. Agnew, Doctor's Building, 1231 N. 29th Street, Billings, Mont. 59101.

NEBRASKA CHAPTER ACR
Secretary-Treasurer, Dr. Gordon F. Johnson, 4239 Farnam, Omaha, Neb. 68131. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEVADA RADIOLOGICAL SOCIETY
Secretary, Dr. William G. Arbonies, Department of Radiology, St. Mary's Hospital, Reno, Nev.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Stefan C. Schatzki, 1180 Beacon St., Brookline Mass. 02146. Meets third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY
Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

NEW MEXICO SOCIETY OF RADIOLOGISTS
Secretary, Dr. Donald A. Wolfel, Albuquerque, New Mexico. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

NEW YORK ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Samuel H. Madell, 1 E. 82nd St., New York, N. Y. 10028. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M.

NEW YORK STATE CHAPTER OF THE AMERICAN COLLEGE OF RADIOLOGY, INC.
Secretary-Treasurer, Dr. John J. Magovern, 520 Franklin Ave., Garden City, N. Y. 11530.

NORTH CAROLINA CHAPTER OF ACR.
Secretary-Treasurer, Dr. James F. Martin, 300 S. Hawthorne Road, Winston-Salem, N. C. 27103. Fall Meeting, Mid Pines Hotel, Southern Pines, N. C., Oct. 31, 1970, 2 P.M.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Marshall Landa, 1702 13th St., So., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

NORTH FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. John W. Morris, III., Department of Radiology, Halifax District Hospital, Daytona Beach, Fla. 32015. Meets quarterly in March, June, September and December.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Herbert F. Reilly, Jr., 12 Sandalwood Rd., Scotia, New York 12302. Meets in Albany area on third Wednesday of October, November, March, April, and May.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Kevin Ryan, Woodland Medical group, Woodland, Calif. 95695. Meets fourth Monday of Sept., Nov., Jan., March and May at Aldo's Restaurant in Sacramento.

NORTHWESTERN OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

OHIO STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph Hanson, 1544 South Byrne Road, Toledo, Ohio 43614.

OKLAHOMA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Haven W. Mankin, The Mercy Hospital, 501 N. W. 12th St., Oklahoma City, Okla. 73103. Meets in January, May and October.

ORANGE COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Roderick A. Silveira, 100 E. Valencia Mesa Dr., Fullerton, Calif. 92632. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at the Orange County Medical Association Bldg., Orange, Calif.

OREGON RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Clinton B. Sayler, 214 Medical Dental Bldg., Portland, Oregon 98205. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd.; Beaverton, Oregon 97005. Meets annually in Portland, Oregon, Seattle, Washington or Victoria or Vancouver, British Columbia, in early May.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. Theodore A. Tristan, Harrisburg Polyclinic Hosp., Harrisburg, Pa. 17105.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. Norman Williams, Department of Radiotherapy, Montefiore Hospital, 3459 Fifth Ave., Pittsburgh, Pa. 15213. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

RADIATION RESEARCH SOCIETY
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016.

RADIOLOGICAL SOCIETY OF CONNECTICUT, INC.
Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meetings are held quarterly

RADIOLOGICAL SOCIETY OF GREATER CINCINNATI
Secretary-Treasurer, Dr. Donald E. Gunderson, 3553 Bayard Dr., Cincinnati, Ohio 45208. Meets first Monday of each month at Cincinnati Academy of Medicine.

RADIOLOGICAL SOCIETY OF GREATER KANSAS CITY
Secretary, Dr. Leo G. Goertz, 155 S. 18th St., Kansas City, Kans. 66102. Meets last Friday of each month.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

RADIOLOGICAL SOCIETY OF LOUISIANA
Secretary, Dr. Lester W. Eavenson, 2700 Napoleon Ave., New Orleans 15, La. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

RADIOLOGICAL SOCIETY OF RHODE ISLAND
Secretary-Treasurer, Dr. David R. Hallman, The Memorial Hospital, Pawtucket, R. I. 02860.

RADIOLOGICAL SOCIETY OF SOUTH DAKOTA
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Gladden V. Elliott, 5565 Grossmont Center Dr., Suite 1, La Mesa, Calif. 92041. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. M. Pinson Neal, Jr., Medical College of Virginia, 1200 E. Broad St., Richmond, Va. 23219. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Kenneth E. Robinson, Rochester General Hospital, 1425 Portland Ave., Rochester, N. Y. 14621. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo. Aug. 20–22, 1970.

San Antonio-Civilian-Military Radiological Society
Secretary, Dr. Lee F. Rogers, Department of Radiotherapy, Bexar County Teaching Hospital, 4502 Medical Drive, San Antonio, Texas. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

San Diego Radiological Society
President-Secretary, Dr. Q. H. Lehmann, 5565 Grossmont Center Dr. Suite 1, La Mesa, Calif. 92041. Meets first Wednesday of each month at the Town & Country Hotel.

San Francisco Radiological Society
Secretary-Treasurer, Dr. Warren M. Russel, Franklin Hospital, Castro & Duboce, San Francisco, Calif. 94114. Meets quarterly at various hospitals (contact Secretary).

Santa Clara County Radiological Society
Secretary, Dr. Emory G. West, 285 S. Drive, Mt. View, Calif. 94040. Meets monthly at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Southern Medical Association
Secretary, Dr. Phillip W. Voltz, Jr., 120 Medical Professional Bldg., San Antonio, Tex. 78212. Annual Meeting: Dallas, Tex., Nov. 16–19, 1970.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles 27, Calif. Annual meeting: Deauville Hotel, Miami Beach, Fla., September 28, 1970.

Society of Nuclear Medicine
Secretary, Dr. James J. Smith, 140 E. 54th St., New York, N. Y. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting: Los Angeles, Calif., June 26–July 2, 1971.

South Bay Radiological Society
Secretary, Dr. Emerson C. Curtis, University Dr., Menlo Park, Calif. 94025. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Dakota Radiological Society
Secretary, Dr. Donald J. Peik, 1417 S. Minnesota Ave., Sioux Falls, S. Dak. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Aaron G. Fingerhut, 1000 W. Carson St., Torrance, Calif. 90502. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Annual meeting: Grand Hotel, Pointe Clear, Ala. Jan. 29-31, 1971.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society
Secretary-Treasurer, Dr. Lawrence R. Nickell, Maury County Hospital, Columbia, Tenn. 38401. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas Radiological Society
Secretary, Dr. Herman C. Sehested, 815 Medical Tower, Room 100, 1550 W. Rosedale St., Fort Worth, Tex. 76104. Annual meeting at the Flagship Hotel on Pier, Galveston, Tex.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary-Treasurer, Dr. Leon M. Neal, St. Benedict's Hospital, 3000 Polk Ave., Ogden, Utah 84403. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society
Secretary, Dr. John R. Williams, 160 Allen St., Rutland, Vt.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. James S. Redmond, Suite 7 Medical Center, Lynchburg, Va. 24501.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Joan Wohlgemuth, 5021 Seminary Rd., Alexandria, Va. 22311.

Washington State Radiological Society
Secretary-Treasurer, Dr. Paul S. Paulson, 1001 Broadway Seattle, Washington 98122. Meets quarterly.

West Virginia Radiological Society
Secretary-Treasurer, Dr. J. Dennis Kugel, 510-517 Med. Arts Bldg. Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Edgar Palmer, 650 Main St., New Rochelle, N. Y. 10801. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary-Treasurer, Dr. Robert E. Douglas, 1209 S. Commercial St., Neenah, Wis. 54956. Meets twice a year, May and September.

Wyoming Radiological Society
Secretary, Dr. J. D. Grant, Memorial Hosp., Sheridan,

Wyo. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Jorge Vargas Segura, Apartado 5367, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Roberto Calderón, Calle Central Oeste No. 218, Managua, Nicaragua, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de El Salvador
Secretary, Dr. Julio Astacio, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.
Coahulia No. 35, México 7, D.F.
Secretary-General, Dr. Bernardo Serviansky. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Apt. 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. Pierre Archambault, Hôpital Charle Le Moyne, 121 Boul. Taschereau, Greenfield Park, P.Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics.
Honorary Secretary Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada. Annual Congress, to be announced.

Edmonton and District Radiological Society
Secretary, Dr. M. J. Brindle, Fifth Floor, Northgate Bldg., 10049 Jasper Ave., Edmonton 15, Alberta, Canada Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, 47 Lincoln's Inn Fields, London, W.C.2, England.

Faculty of Radiologists, Royal College of Surgeons in Ireland
Registrar, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists
Honorary Secretary, Dr. F. Robert MacDonald, *Associate Honorary Secretary*, Dr. Champlain Charest, 1555 Summerhill Ave., Montreal 25, Que., Canada. Annual meeting to be announced.

Montreal Radiological Study Club
Secretary, Dr. Leonard Rosenthall, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française de Radiologie
Secretary General, Dr. Guy Duckett, 1385 est, rue Jean Talon, Montréal, P.Q., Canada. Meets every third Tuesday from October to April. Annual meeting: Montreal, P.Q., Canada, November 5–7, 1970.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September, through May.

College of Radiologists of Australasia
Honorary Secretary, Dr. T. P. Loneragan, 147 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Fral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Urqiza 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional de Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Miguel Mario, Céntola, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiología
Secretary-General, Dr. Juan R. Heilbuth Pacheco, Santa Fe 1171, Buenos Aires. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Cxa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigaderio Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Carlos Vildósola, Casilla, 13426 Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Raul Fernández Angula, Calle 43 No. 44-70 Barranquilla, Colombia. Meets last Thursday of each month.

Sociedad Equatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 4569, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dr. Julio Ormeño del Pino, Instituto de Radiología "Cayetano Heredia" Hospital Arzobispo Loayza, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41–110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología de la Plata
Secretary, Dr. Hiram D. Haurigot.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Alejandro Glausiuss, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia

da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Modesto Rivero González, Apartado No. 9213 Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Second Congress of the European Association of Radiology.
President, Professor Dr. J. R. von Ronnen, State University of Leiden, The Netherlands.
Secretariat, c/o Holland Organizing Centre, 16 Lange Voorhout, The Hague, The Netherlands. Congress Meeting: Amsterdam, The Netherlands, June 14–18, 1971.

Österreichische Rontgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Permanent Secretary, Dr. Jaques Sauvegrain, Hôpital des Enfants-Malades, 149, rue de Sèvres, Paris 15e, France.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru Paris 8e, France.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Rontgengesellschaft
President, Professor Dr. med. Hanno Poppe, Radiologische Univ.-Klinik, Gossler Str. 10, 34 Göttingen, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano Torino, Italy. Meets annually.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. C. B. A. J. Puijlaert, Prof. Dondersstraat 73, Tilburg, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. E. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägen 5 C., Finland.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. D. José Bonnati, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Schweizerische Gesellschaft Für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. L. R. Parthasarathy 44, Constable Road, Madras—23, India.

Indonesian Radiological Society
Secretary, Professor Sjahriar Rasad, Taman Tjut Mutiah I Diakarta, Indonesia

Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Africa

Association of Radiologists of West Africa
Honorary Secretary, Dr. S. B. Lagundoza, University College Hospital, Ibadan, Nigeria.

South African International radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa. Second Congress: President Hotel, Johannesburg, September 3-9, 1970.

ABSTRACTS OF RADIOLOGICAL LITERATURE

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ROENTGEN DIAGNOSIS

Head

Occleshaw, J. V. The posterior inferior cerebellar arteries: some quantitative observations in posterior cranial fossa tumours and the Arnold-Chiari malformation. *Clin. Radiol.*, Jan., 1970, *21*, 1–9. (From: Department of Radiology, Regional Medical and Surgical Neurology Unit, Walton Hospital, Liverpool 9, England.)

The author reviews the development of the technique of vertebral angiography. The vertebral angiograms of 110 cases were studied. Fifty were normal and 6 were posterior cranial fossa expansive lesions. Three cases of Arnold-Chiari malformation were included.

The roentgenologic anatomy of the normal posterior inferior cerebellar artery is described. Measurements were made of the position of the apex of the cranial loop of this artery in relationship to Twining's line, and the position of the caudal loop in relationship to the foramen magnum.

The tumor cases were divided into 5 subgroups with reference to their location in the posterior cranial fossa. The measurements of these subgroups were treated statistically and summarized in table form.

The apex of the cranial loop of the posterior inferior cerebellar artery was not depressed in any of the cases, except those with tumor involvement of the vermis and adjacent cerebellar hemisphere. The displacement of the posterior inferior cerebellar artery as a reliable lateralizing sign was confirmed by this series. Displacement of the caudal loop of the posterior cerebellar artery with herniation of the cerebellar tonsils through the foramen magnum is discussed in detail.

The author also discusses the frequency of occurrence of bilateral filling of the posterior inferior cerebellar arteries.—*James R. Knapp, M.D.*

Obenchain, Theodore G., Clark, Robert, Hanafee, William, and Wilson, Gabriel. Complication rate of selective cerebral angiography in infants and children: a comparison with a similar adult series. *Radiology*, June, 1970, *95*, 669–673. (Address: William Hanafee, M.D., UCLA School of Medicine, Los Angeles, Calif. 90024.)

The authors describe the type and incidence (5 per cent) of complications in 100 consecutive pediatric cerebral angiograms. The age range of the patients was from 18 days to 13 years. Of the 100 cases, 98 returned to a status equivalent to the preangiographic state within 24 hours. The complications included 1 hematoma, 1 vascular complication manifested by loss of dorsalis pedis pulsation without hematoma, 2 cases of postangiographic fever, and 1 death. The death occurred in a 5 month old male who was admitted in semicoma with signs and symptoms suggesting a posterior fossa tumor. His postoperative course was one of fulminating infection and death ensued 36 hours following angiography.

The authors indicate that the complication rate in their selected pediatric group was less than in a corresponding adult group. Four of the 5 complications in the corrected adult rate were neck hematomas secondary to direct carotid punctures. The authors believe that this assumes greater importance because fewer direct carotid punctures were performed in adults than were femoral artery punctures in either adults or children. This would also prove that the danger of hematoma in a femoral artery puncture is less because of the ability to exert direct pressure on the femoral artery. There is no greater risk of complication with an increased volume of contrast agent.

The authors' conclusion is that selective cerebral angiography performed by the femoral artery route is as safe in the pediatric age group as in adults.—*J. P. Eberts, M.D.*

Akerman, M. La photo-scintigraphie cérébrale au pertechnétate de ^{99m}technetium. (Cerebral photoscintigraphy by pertechnetate with technetium 99m.) *Presse méd.*, April, 1970, *78*, 937–944. (Address: Hôpital Foch, F-92-Suresnes, France.)

The cerebral exploration of 600 patients by photoscintigraphy using technetium 99m proved to be more efficacious than with chemicals used hitherto.

Of these patients, 196 had expansive intracranial lesions in which the diagnostic accuracy was as follows:

Tumors of the cerebral hemispheres	105 (91 per cent correct)
Tumors of the hypophysis and 3rd ventricle	24 (75 per cent correct)
Subtentorial tumors	26 (81 per cent correct)
Angiomas and hematomas	15 (87 per cent correct).

This is an over-all total of 87 per cent correctly diagnosed by scanning, with the poorest results in tumors of the hypophysis and 3rd ventricle, where the normally strong radioactivity of the base causes confusion. Often the tumors are not large, and are hard to detect when they enlarge upward. Even so, scannography may prove to be superior to those methods which are most useful when there is midline deflection.

There were also 66 nonexpansive lesions, the distribution of which was as follows: softening of brain tissue, 28; trepannation, 22; superficial and cerebral contusions 9; Paget's disease 2; fibrous dysplasia 1; and various 4.

The accuracy of photoscintigraphy was remarkable in finding all 10 neurinomas of the pontocerebellar angle in the series.

Technetium 99m, aside from its physical conveniences, combines with numerous molecules, as in serum albumen, colloids and red cells, which considerably diversifies its utility.—*Frank A. Riebel, M.D.*

WAGNER, HENRY N., JR., HOSAIN, FAZLE, DELAND, FRANK H., and SOM, PRANTIKA. A new radiopharmaceutical for cisternography: chelated ytterbium 169. *Radiology*, April, 1970, *94*, 121–125. (From: Division of Nuclear Medicine, The Johns Hopkins Medical Institutions, Baltimore, Md.)

The technique of radionuclide cisternography and ventriculography is currently of renewed interest because tracer techniques are ideal for determining patterns of cerebrospinal fluid movement in man, and current data suggest that neurosurgical shunting procedures may help certain patients with alterations in the normal circulation of cerebrospinal fluid. The procedure is termed cisternography or ventriculography depending on the site of introduction of the radioactive tracer. The course of flow of the radioactive material is imaged sequentially by rectilinear scanning or gamma camera scintiphotography over varying periods of hours or days. This technique aids in the diagnosis of many states of altered cerebrospinal dynamics: cerebrospinal rhinorrhea or otorrhea, varying types of hydrocephalus, evaluating patency of neurosurgical shunts, etc.

Radioiodinated human serum albumin in a dose of 100 μc has been the classical agent. The photon yield from this is less than desired, and some potential for toxicity is inherent in this material. Tc^{99m} labelled albumin has been used in millicurie amounts to produce better photon yields, but the problem of maintaining daily quality control limits its use. The report of Bell and associates suggesting Tc^{99m} labelled insulin as an appropriate agent led the authors to investigate the use of ytterbium 169 chelated with diethylenetriaminepentaacetic acid (Yb^{169}DTPA). They had previously introduced this agent for brain scanning and renal studies, and described its preparation. Minor modifications to reduce the ytterbium content, and the molar concentration of DTPA were employed. Calcium chloride was added to reduce chelation of calcium from the cerebrospinal fluid. One millicurie doses of the subsequent preparation were found adequate for high photon yield.

The preparation has been proven chemically stable. Studies show it to be physiologically stable when injected intravenously or into cerebrospinal fluid. The material is excreted from the blood into the urine by glomerular filtration, unchanged, and in a rapid fashion. The biologic half life, in the cerebrospinal space, is approximately 10 hours. The radiation dose is difficult to calculate due to the geometric configuration of the cerebrospinal fluid space, but on the basis of certain assumptions, the absorbed radiation dose to the cerebrospinal fluid space is about 5 times that of Tc^{99m} albumin, doses being equal. The average adult dose used in 30 patients was 15 μc/kg. with an average chelated complex content of 0.03 mg./mc. In dogs, doses between 200–1,000 times the usual human dose failed to elicit toxic response.

The comparatively long physical half life of Yb^{169} of 32 days suggests that Yb^{169}DTPA might be produced by a radiopharmaceutical company, and quality control in detail practiced by the firm rather than relying on quality control immediately prior to use as is necessary with the shorter lived radionuclide preparations. Its rapid excretion, and subsequent short biologic half life result in an acceptable absorbed dose of radiation.

The accompanying photographs delineate excellent images of the cerebrospinal fluid compartment, yielding quality diagnostic information.—*F. C. Petty, M.D.*

SUZUKI, SOJI, and KAWASHIMA, KENKICHI. Sialographic study of diseases of the major salivary glands. *Acta radiol.*, Nov., 1979, *8*, 465–478. (From: Departments of Radiology and Surgery, The Tokyo Medical and Dental University School of Medicine, Tokyo, Japan.)

The place of sialography in the diagnosis and management of patients with suspected salivary gland pathology has been the subject of controversy since Arcelin introduced the procedure in 1913.

The technique of this neglected roentgenographic procedure is discussed. The authors use a polyethylene catheter introduced by the Seldinger technique. They prefer urografin 76 per cent and lipiodol-ultrafluid as the contrast media.

They present the findings in 160 sialographies. The procedure is indispensable in the diagnosis of sialolithiasis, chronic sialoadenitis and various sialoadenopathies. It is also useful in the differential diagnosis of sialomas and parasialomas.—*Richard P. Taylor, M.D.*

ACHESON, E. D., COWDELL, R. H., and JOLLES, B. Nasal cancer in the Northamptonshire boot and shoe industry. *Brit. M. J.*, Feb. 1970, *1*, 385–393. (Address: E. D. Acheson, D.M., Medical Director, Oxford Record Linkage Study, Oxford, England.)

A study of the incidence of nasal cancer in Northamptonshire, England, during the period 1953 to 1967, revealed 46 patients with cancer of the nose and related sinuses. Also included in the study are 5

other cases diagnosed either before 1953 or after 1967.

The incidence of nasal cancer of all types was significantly higher in males within the boot and shoe industry, who were exposed to the dust of materials used in the manufacture of footwear.

The authors suggest that 2 carcinogenic factors might be present in the industry with one related to the production of adenocarcinoma and the other to squamous and other types of carcinoma. Possible hazardous substances implicated in this study are leather, rubber, fiberboard, vegetable material used in tanning, chromate salts, and crysoidine dye.

The authors estimate that the latent period for adenocarcinoma was 54 to 56 years, and for squamous, transitional, and anaplastic tumors, 41 to 47 years.

It is not known if the factors are still present in the industrial environments since the standards of industrial hygiene in the industry have improved substantially since these cases were first exposed.

The possibility of increased risk of nasal adenocarcinoma in the footwear repairing industry is also suggested, but insufficient evidence is present in the material collected at this time.

The evidence furthermore suggests that snuff taking should be considered as a possible contributory factor in both industrial and nonindustrial nasal cancer.—*James R. Knapp, M.D.*

Neck and Chest

Johnson, T. H., Jr., Howland, W. J., and Reagan, T. A. Aerosol laryngography and bronchography. *J. Canad. A. Radiologists*, March, 1970, *21*, 16–18. (Address: T. H. Johnson, Jr., M.D., Department of Radiology, Presbyterian-University Hospital, Pittsburgh, Pa.)

A new technique for aerosol laryngography and bronchography is described.

The contrast material used is a commercial preparation of barium sulphate-carboxymethylcellulose called redi-FLOW. The apparatus for aerosol introduction of a topical anesthetic material such as 1 per cent lidocaine hydrochloride (Xylocaine) followed by the barium preparation is a No. 15 DeVilbiss atomizer with a compressor air source. The variable air pressure control prevents surface damage. The direction of the spray is controlled by manipulation of the atomizer tip which can be turned up and down. The contrast material deposits a coating on the wall of the bronchi and the patient is positioned so that the contrast material flows in the direction of gravity. This is felt necessary to obtain delineation of areas which have no air exchange or to outline shut-off bronchial passages.

Barium contrast materials for tracheobronchial diagnosis are not yet approved for human use by the Food and Drug Administrations; however, it is hoped that this will soon be remedied.

Laryngography is simple with this technique. It has proved diagnostic in all cases. Bronchography by this technique is a logical extension of the laryngography procedure. Bronchography is acceptable and diagnostic but the authors have had some difficulty filling the upper lobe bronchi.

It seems likely that aerosol contrast materials and also suitable topical anesthetic agents will eventually be prepared in pressurized cans which would further simplify the performance of bronchography.—*William T. McLaughlin, M.D.*

Genereux, George P. Lipids in the lungs: radiologic-pathologic correlation. *J. Canad. A. Radiologists*, March, 1970, *21*, 2–15. (From: Department of Diagnostic Radiology, University Hospital, Saskatoon, Saskatchewan, Canada.)

The author presents a classification of lipids in the lungs based on pathogenesis:

I. *Exogenous*
  A. Aspiration lipid (lipoid) pneumonitis
  B. Postlymphangiographic lung
  C. Other
    (1) bronchography
    (2) myelography
    (3) hysterosalpingography
    (4) urethrography

II. *Endogenous*
  A. Cholesterol pneumonitis
    (1) with bronchial obstruction
    (2) without bronchial obstruction
  B. Fat embolism
  C. Pulmonary alveolar proteinosis
  D. Lipid storage diseases
    (1) familial
    (2) granulomatous
  E. Disseminated lipogranulomatosis

The lesions of the exogenous group appear in the lungs as a result of inhalation, aspiration or vascular embolization of vegetable, animal or mineral oils. The endogenous lipids are normal components of the lungs or extrapulmonary soft tissues.

The author discusses in detail the radiologic-pathologic correlation of these diseases with a presentation of the literature and the differential diagnosis.

The reader is directed to the original article for an in-depth study of these interesting conditions.—*S. A. Kaufman, M.D.*

Highman, J. H. Chest infection in childhood. *Clin. Radiol.*, Jan., 1970, *21*, 52–61. (From: Paddington Green Children's Hospital, Paediatric Unit, St. Mary's Hospital, London, W.2, England.)

The author reviews a series of childhood chest infections observed over a 3 year period from 1964 to 1966, which were associated with abnormalities of the chest roentgenogram.

A total of 384 children were considered to have a significant roentgenologic abnormality. The age distribution shows a fairly consistent level during the first years of life, while later in the first decade only infrequent severe chest infections were seen. The first 3 years presumably represent a period in which the child meets a variety of pathogens and develops immunity. Many cases clear remarkably within 1 week, although most cases clear within 3 to 4 weeks. The right lung is more often affected than the left and there was a very high incidence of right middle lobe lesions, while both lower lobes were almost equally affected. This weighing of lower lobe involvement suggests a considerable element of aspiration in the causation of much childhood pneumonia.

Lung collapse was a feature in 27 cases and was believed to be due to aspiration of mucus from the upper respiratory tract or to excessive local mucus formation.

Particulate foreign matter was encountered in only 2 cases in the whole series. No specific cause could be determined for pulmonary collapse in the other 25 cases and the lungs re-expanded following conservative treatment.

Death occurred in 11 cases. In 1 it was due to overwhelming bronchopneumonia, while in others it was due to fibrocystic disease, chronic renal disease, and other abnormalities. The etiology could not be determined in approximately 90 per cent of the cases. Ten per cent of the lesions were ascribed to cystic fibrosis, aspiration and inhalation, tuberculosis and staphylococcal pneumonia.

The author briefly describes the roentgenographic findings in 5 cases of cystic fibrosis and aspiration pneumonia, 21 cases of postprimary tuberculosis, and 3 cases of staphylococcal pneumonia.—*James F. Martin, M.D.*

SIEGEL, R. R. The basis of pulmonary disease resolution after nephrectomy in Goodpasture's syndrome. *Am. J. M. Sc.*, March, 1970, *259*, 201–213. (Address: Division of Nephrology, Department of Medicine, University of Kentucky Medical Center, Lexington, Ky.)

Goodpasture's syndrome is a form of rapidly progressive glomerulonephritis associated with intrapulmonary hemorrhage and subsequent pulmonary hemosiderosis. Cessation of the pulmonary lesions following nephrectomy is documented. The reported case is the fourth in the literature and is the longest reported survivor, living in good health $3\frac{1}{2}$ years postnephrectomy and transplant.

The glomerular lesions of Goodpasture's syndrome are marked by a pronounced tendency to necrosis and basement membrane disruption. The most plausible role of glomerular necrosis in the pathogenesis of Goodpasture's syndrome is in stimulating the production of anti-basement membrane antibodies by liberating basement membrane antigen from disrupted glomeruli. This material would be carried by venous and lymphatic drainage directly to the lung, and in the presence of circulating antibody, an intense reaction would occur focally in the area of macromolecular antigen embolization. Glomerular necrosis occurs to a minor degree in all cases of glomerulonephritis, and minor pulmonary damage has been demonstrated in many of these cases, without overt hemoptysis, and hence not classifiable as Goodpasture's syndrome. Anti-basement membrane antibodies, which have been demonstrated consistently in Goodpasture's syndrome, and less frequently in other nephritides, may thus be involved in the genesis of pulmonary lesions. Postnephrectomy resolution of the lung lesions may be due to the removal of the source of basement membrane antigens.

This hypothesis provides a rationale for nephrectomy when renal failure supervenes in Goodpasture's syndrome—a measure which is nevertheless independently justified by accumulating clinical experience. Continued freedom from pulmonary disease may depend upon the integrity of the renal homograft, since rejection could provide a new source of basement membrane antigen to exacerbate the pulmonary lesion.—*Mark D. Reiss, M.D.*

PAIROLERO, PETER C., HALLERMANN, FRANZ J., and ELLIS, F. HENRY, JR. The left ventriculogram in experimental myocardial infarction. *Radiology*, May, 1970, *95*, 311–316. (Address: Franz J. Hallermann, M.D., Section of Roentgenology, Mayo Clinic and Mayo Foundation, Rochester, Minn. 55901.)

Using adult Mongrel dogs and producing myocardial infarcts of the entire anterolateral wall of the left ventricle, the authors recorded angiocardiographically the changes in the left ventricular contraction pre and post infarction.

The radiographic features were akinesis of the involved myocardial wall, reduced ejection fraction, frequent mitral incompetence, and sequestration of the segments of the left ventricle. The greatest extent of akinesis was at 24 hours post infarction with improvement occurring over the first 2 weeks following infarction; yet values failed to reach preinfarction levels even after 3 months.

The radiographic sequence of events after infarction was gradual decrease of the akinetic segment of approximately 50 per cent of its original size, increase of the ejection fraction of the left ventricle, and frequent disappearance of the mitral incompetence.—*William L. Graham, M.D.*

Harell, George S., Friedland, Gerald W., Daily, William J., and Cohn, Roy B. Neonatal Boerhaave's syndrome. *Radiology*, June, 1970, *95*, 665–668. (From: Departments of Radiology, Pediatrics, and Surgery, Stanford University School of Medicine, Stanford, Calif.)

The authors describe a case of spontaneous esophageal rupture (Boerhaave's syndrome) in a neonate who developed dyspnea on the second day of life. The patient manifested the roentgenographic findings on upright frontal and lateral roentgenograms of the chest characteristic of this syndrome: tension hydropneumothorax, pneumomediastinum, and thin-walled ring shadows on the side of the pneumothorax. Oily dionosil study of the esophagus demonstrated a tubular serpentine tract extending from the lower esophagus into the pleural space.

A discussion of the characteristic clinical and roentgenographic findings in Boerhaave's syndrome follows the case presentation. Differences between neonatal and adult spontaneous esophageal rupture are pointed out. In adults, the hydropneumothorax is not under tension, mediastinal emphysema is present in about 60 per cent, males predominate, and rupture usually occurs in the left pleural cavity. In the neonate, tension pneumothorax or tension hydropneumothorax is a characteristic feature, pneumomediastinum is uncommon, females predominate, and rupture usually occurs into the right pleural cavity.

The etiology of spontaneous neonatal esophageal rupture is not known. In this case the histologic changes in the esophagus were somewhat similar to the changes seen in the bowel in necrotizing enterocolitis, leading to the speculation that the esophageal changes may have been a form of this process in an unusual location.—*John G. Hutka, M.D.*

Abdomen

Friedland, Gerald W., Mason, Robert, and Poole, G. Joseph. Ladd's bands in older children, adolescents, and adults. *Radiology*, May, 1970, *95*, 363–368. (From: Departments of Radiology and Surgery, Stanford University School of Medicine, Stanford, Calif.)

Congenital extrinsic duodenal obstruction, excluding annular pancreas, in infants was first described by Ladd in 1933. This obstruction was subsequently referred to as "Ladd's bands." It has been recognized that patients of any age may present with this syndrome, and the purpose of this paper is to report 4 cases recognized in older children or adults and to describe the characteristic roentgenographic findings. These obstructing bands or adhesions may occur in patients with and without anomalies of intestinal rotation.

Roentgenographically 2 forms are recognized: acute and chronic. The acute form presents as a high grade duodenal obstruction and is characterized clinically by an episode of acute upper intestinal obstruction. A history of previous episodes of abdominal pain or vomiting, or both, is universal in older infants, children, and adults. The chronic form may present itself in one of 2 forms: (a) intermittent vomiting and pain or (b) a malabsorption syndrome, secondary to chronic midgut volvulus, with partial occlusion of lymphatics and veins in the mesenteric root.

Upper gastrointestinal series may show a normal stomach or may show moderate amount of fluid in the stomach. The extent of dilatation of duodenum will vary from slight dilatation to considerable dilatation. Active peristalsis is seen and a so-called to-and-fro movement of the barium in the second and third portions of the duodenum will often be present. The site of obstruction in the duodenum may be anywhere from the region of the crossing of the superior mesenteric artery to the ligament of Treitz. These findings are more evident when the patient is upright, but when the patient assumes the prone or genu pectoral position are less evident. The authors present the hypothesis that the so-called superior mesenteric artery syndrome and duodenal obstruction by Ladd's bands are identical conditions.

Four case reports are included in which the diagnosis was made and surgery performed lysing adhesions and bands of fibrous tissue which were found partially obstructing the duodenum.

It is the opinion of the authors that the syndrome may be missed by radiologists unless a specific point is made of examining duodenal motility in the upright and prone positions and also possibly in the genu pectoral position. It is also their feeling that when symptomatology suggestive of Ladd's syndrome is present and the upper gastrointestinal series appears normal, a repeat examination during an acute attack may be positive and permit diagnosis of this syndrome.—*Donald N. Dysart, M.D.*

Reid-Smith, A. The use of sorbitol in the investigation of the small bowel. *J. Canad. A. Radiologists*, March, 1970, *21*, 35–38. (From: Victoria Hospital, London, Ontario, Canada.)

It would be valuable to reduce the time of a small bowel procedure in terms of convenience to the radiologist, the staff and the patient.

Sorbitol is a pleasant tasting sugar which is prepared by the hydrolysis of glucose. It is a white, odorless, hygroscopic powder. Several workers have found that sorbitol hastens the passage of barium through the small bowel because of the production of increased peristaltic activity. The hypertonic solution which is not absorbed draws fluid into the

bowel by osmosis. The increased fluid content stimulates peristalsis.

A study was performed to determine whether the use of sorbitol is likely to result in incraesed accuracy of diagnosis in small bowel examination. One hundred patients were examined in a routine manner using 20 ounces of barium sulphate in colloidal suspension. Another group of 100 patients were examined using a commercial preparation, Gi-Prep Liquid. A single dose of Gi-Prep Liquid contains 13.5 gm. of d-sorbitol and 3 mg. of homatropine methylbromide. The latter is included for its spasmolytic effect.

The roentgenograms were examined by 3 independent observers without the knowledge of whether or not sorbitol had been used. Particular attention was paid to the detail of the mucosal pattern and the transit time to the small bowel.

Loss of mucosal detail interferred with interpretation in 17 per cent of cases in the control group but rose to 56 per cent when sorbitol was used.

It is concluded that the use of sorbitol cannot be recommended for detail study of the small bowel and is of doubtful value for routine study. It may be of help to some workers when the time factor is of predominant importance or when mucosal detail is not the main point of interest.—*William T. McLaughlin, M.D.*

PARKS, T. G., CONNELL, A. M., GOUGH, A. D., and COLE, J. O. Y. Limitations of radiology in the differentiation of diverticulitis and diverticulosis of the colon. *Brit. M. J.*, April 18, 1970, *2*, 136–138. (Address: T. G. Parks, Senior Registrar, Royal Victoria Hospital, Belfast, Northern Ireland.)

This report represents a radiologic analysis of 461 patients diagnosed and treated for diverticular disease of the colon at the Royal Victoria Hospital in Belfast.

The authors have chosen the term "diverticular disease" to include diverticulosis and diverticulitis. They agree that the barium enema examination is the most useful method for investigation of colonic diverticular disease.

They present evidence that the terms "diverticulosis" and "diverticulitis" are unsatisfactory and show that the traditional criteria are not accurate in determining the presence of inflammatory change in colonic diverticula.—*Richard P. Taylor, M.D.*

DICKINSON, S. JEROME, SHAW, ANTHONY, and SANTULLI, THOMAS V. Rupture of the gastrointestinal tract in children by blunt trauma. *Surg., Gynec. & Obst.*, April, 1970, *130*, 655–657. (From: Division of Pediatric Surgery, the Brooklyn-Cumberland Medical Center; the Pediatric Surgical Service of the Department of Surgery, Harlem Hospital Center; the Surgical Service, Babies Hospital; and the Department of Surgery, Columbia University College of Physicians and Surgeons, New York, N. Y.)

The authors report 5 cases, ages 3 to 5 years, in which blunt trauma to the abdomen produced lesions in the sigmoid, the duodenum, and the jejunum.

They point out that injuries to the abdominal viscera from blunt trauma involve a solid viscus in the vast majority of patients. In all age groups, only 16 per cent of such injuries specifically affect the gastrointestinal tract. When rupture does occur, it is usually in the fixed portions, such as the duodenum, right colon, and at the junction of the sigmoid colon and rectum.

The roentgenographic findings were positive in 2 cases, negative in 2 cases, and not done in 1. Plain film roentgenographic studies of the abdomen may be useful to detect free gas or separation of the loops of the intestine by fluid. The importance of delayed roentgenograms is stressed in that many of the patients do not show any evidence of perforation until about 24 hours after the injury. The retroperitoneal perforations manifest themselves by slowly increasing pain in the abdomen and a slow development of signs of an acute condition of the abdomen.

The authors feel that the differential diagnosis between this condition and a delayed rupture of the spleen is difficult and probably can be made only at laparotomy.

The mortality rate has been reduced greatly in recent years when the injury was not a part of massive additional trauma. Mortality rates of 92 per cent are recorded in the older literature and 16 to 20 per cent in more recent years.

The authors report no deaths in their series. They stress the importance of early recognition and operation.—*James F. Martin, M.D.*

BLENDIS, L. M., MCNEILLY, W. J., SHEPPARD, LOUISE, WILLIAMS, ROGER, and LAWS, J. W. Observer variation in the clinical and radiological assessment of hepatosplenomegaly. *Brit. M. J.*, March 21, 1970, *1*, 727–730. (Address: L. M. Blendis, M.B., Research Fellow, Medical Research Council Group on Metabolism and Haemodynamics of Liver Disease, Department of Medicine, King's College Hospital, London S.E. 5, England.)

The size of the liver and spleen of 32 patients with a variety of clinical conditions was assessed independently by 4 clinicians from physical examination, and by 4 radiologists from a plain abdominal roentgenogram.

There was good agreement found between the 4 radiologists in grading liver and spleen size; in ap-

proximately 90 per cent of the cases at least 3 of 4 radiologists agreed on the findings. The greatest observer variation arose in cases in which the liver or spleen was poorly seen on the roentgenogram. The least variation was seen when the organs were significantly enlarged.

Clinical assessment was found to be subject to greater variation, particularly in regard to the liver. It is concluded that a plain roentgenogram of the abdomen is a useful adjunct to clinical examination. —*Mark D. Reiss, M.D.*

Tavernier, J., Delorme, G., Lafitte, J., Tessier, J., and Bellet, M. Apports de l'angiographiecoeliaque et mésentérique à la radiographie pancréatique. (The contributions of celiac and mesenteric angiography to pancreatic radiography.) *J. de radiol., d'électrol. et de méd. nucléaire*, Dec., 1969, *50*, 867–880. (From: Service de Radiologie, Hôpital Saint-André, C.H.U. de Bordeaux, France.)

The knowledge of pancreatic pathology has been so enriched by scintigraphy and arteriography that both modalities should be used; especially is arteriography valuable in revealing not only volumetric changes in the pancreas, but the nature and causes of disease.

Classically the celiac trunk and superior mesenteric vessels are successively catheterized, and then superselective studies are made. The authors performed 120 such studies, and angiographically disclosed 11 of the 12 cases of cancer, 11 of the 17 cases of pancreatitis, and all of the 6 cysts. Many other lesions (hepatic, gastric, splenic, etc.) were revealed.

In tumorous cases, whether benign or malignant, the authors describe the displacement, compression or attenuation of each artery and vein, and the capillary phases. In pancreatitis, infringement on arteries is classified as wasp-waisted, conical, cylindrical, amputated or absent. Veins and collaterals are described, with evaluations as to capillary phases and portal hypertension.

As there are many developmental variants in this whole vascular bed, they are also recorded; accordingly, the surgeon can plan his attack and decide what risks he is taking in regard to the rest of the pancreas, colon, gallbladder, liver and jejunum.—*Frank A. Riebel, M.D.*

Op den Orth, J. O. A radiologically demonstrated fistula between the commou bile duct and the right renal pelvis. *Radiol. clin. et biol.*, 1969, *38*, 402–405. (Address: Exoolaan 12, Almelo, The Netherlands.)

The author reports an unusual case of a biliary renal fistula in a 63 year old female.

Cholecystography showed gas in the biliary tree and no excretion of contrast material by the right kidney. Right retrograde pyelography demonstrated a fistulous tract between the superior calyx and the bile duct.

The author postulates that an anomaly of the gallbladder is probably the primary etiology.—*Richard P. Taylor, M.D.*

Hardcastle, J. D. Acute non-tuberculous psoas abscess; report of 10 cases and review of the literature. *Brit. J. Surg.*, Feb., 1970, *57*, 103–106. (From: The London Hospital, London, England.)

With the decreased incidence of spinal tuberculosis, the majority of psoas abscesses now seen are of nontuberculous origin. They are most commonly related to intraabdominal pathology, and 4 of the 10 cases reported were in patients with acute appendicitis. Two of the 4 cases with appendicitis had roentgenographic evidence of air in the psoas muscle and/or thigh.

Inflammatory and neoplastic lesions involving the retroperitoneal structures in the lower abdomen and pelvis may present with symptoms suggesting hip pathology or gynecologic disorders, with possible subsequent delay in diagnosis.

Extension of infection from the kidney into the psoas sheath is uncommon, but may occur following perforation of the renal pelvis by a calculus, or in relation to a perinephric abscess. Psoas abscess may also result from extension of spinal osteomyelitis, from suppurating iliac lymph nodes, or as a primary blood-borne infection.—*Mark D. Reiss, M.D.*

Gynecology and Obstetrics

Du Bois, R., Flipo, B., Monnier, J. C., Guiot, P., and Delecour, M. (Lille, France.) L'hystérographie de l'utérus césarisé. (Hysterography after cesarean section.) *J. de radiol., d'électrol. et de méd. nucléaire*, Dec., 1969, *50*, 887–892.

Hysterographies were performed on 56 women who had submitted to cesarean section from 6 weeks to 2 years before the radiologic study. About half had transverse, the rest longitudinal incisions. Thirteen cases had deformities; these were classified as diverticular pouches, rounded niches, cruciform, spiculated or spur-like patterns, dehiscence, narrowing, fistulae or lacunae. Indirect abnormal signs were angulation, bayonet patterns and gaping deformities from poor muscular tone. In a few cases the lesions were multiple.

It is important to record not only the pattern, but also the location of the defects and their size, those over 4 to 5 mm. having importance. The authors found that age and parity are significant, most

primiparas under 30 years recovering without visible scars. Transverse incisions produced more deformities than did longitudinal ones; infections or complications and operative hemorrhage produced morbidity, as did surgery before term.

Hysterography is recommended after cesarean section except in those cases where the tubes are tied or future pregnancies are otherwise precluded; they are especially recommended if a future delivery may not be done surgically.

The study should not be done within 3 months, and preferably 6 months, after cesarean section.—*Frank A. Riebel, M.D.*

AGÜERO, OSCAR, and ZIGHELBOIM, ITIC. Fetography and molegraphy. *Surg., Gynec. & Obst.*, April, 1970, *130*, 649–654. (From: Maternidad Concepción Palacios Hospital, Caracas, Venezuela.)

Amniography is a method which allows placental localization, the diagnosis of fetal intrauterine life, the demonstration of contrast medium in the gastrointestinal tract of the fetus, and topographic localization of the placenta. The usual water soluble contrast material is used in this procedure.

Fetography is a procedure used to diagnose external anomalies of the fetus and is accomplished by injecting iophendylate (myodil) which has an affinity for the vernix caseosa, permitting an excellent outline of the soft tissues of the fetus *in utero*. The fetal outline is usually evident on 48 hour roentgenograms.

The authors suggest the term molegraphy for the image produced when phendylate is used in the diagnosis of hydatid mole.

Amniography performed with an oil soluble contrast medium gives some details which are not achieved with the water soluble material. These include: design of fetal contour; its low diffusion in the amniotic fluid eliminates the opaqueness of this fluid and defines more clearly the fetal outline; its affinity for the vernix caseosa, and the small quantity injected diminishes fetal ingestion; and its low excretion or its persistence in the amniotic sac does not allow the observation of the pyeloureteral cystograms.

The authors report their experience with fetography in 29 patients. In most patients, the amount of contrast medium used was 9 milliliters. The roentgenographic films were exposed in 24 and 48 hours in most cases, but in the case of hydatid mole, good studies could be obtained at the end of 15 minutes. The lace-like image seen in hydatid mole is rather characteristic of this entity.

The method proved very useful in the detection of malformations of the fetus, especially anencephalic fetuses, in intrauterine deaths in which an increased separation was observed between the fetal skull and the scalp, and in other intrauterine anomalies.

The basic indications for fetography are hydramnios, history of previous fetal malformation, diabetes, presumption of fetal death, and erythroblastosis.—*James F. Martin, M.D.*

GENITOURINARY SYSTEM

BENNESS, G. T. An evaluation of anti-diuretic hormone in intravenous urography: "sniff urography." *Clin. Radiol.*, April, 1970, *21*, 157–162. (From: Department of Surgery, University of Sydney, Sydney, Australia.)

The value of preliminary dehydration in intravenous urography has been long appreciated. However, the suspicion that it was not always effective led to the evaluation of lypressin, an Anti-Diuretic Hormone (A.D.H.) in urography.

The investigator studied the effect of A.D.H. on 4 prison volunteers and found that it increased the urinary concentration of the contrast medium and improved the quality of the pyelogram.

The A.D.H. was administered by sniffing 20 I.U. of the material 10 to 20 minutes before the intravenous injection to obtain best results.

It is suggested that the use of this procedure may even permit a reduction of contrast material needed. The use of A.D.H. allows the elimination of preliminary dehydration in patients with impaired renal function.

Lypressin (Sandoz) is a synthetic preparation (lysine-8-vasopressin) identical with the naturally occurring antidiuretic hormone found in some animals.—*Arch H. Hall, M.D.*

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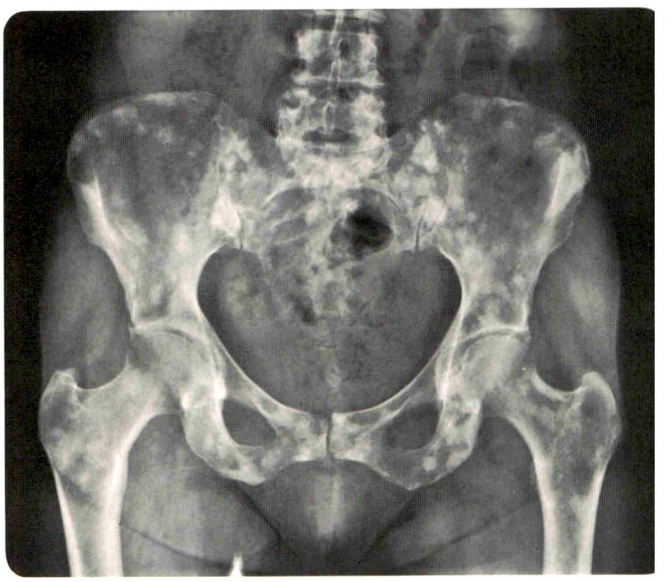
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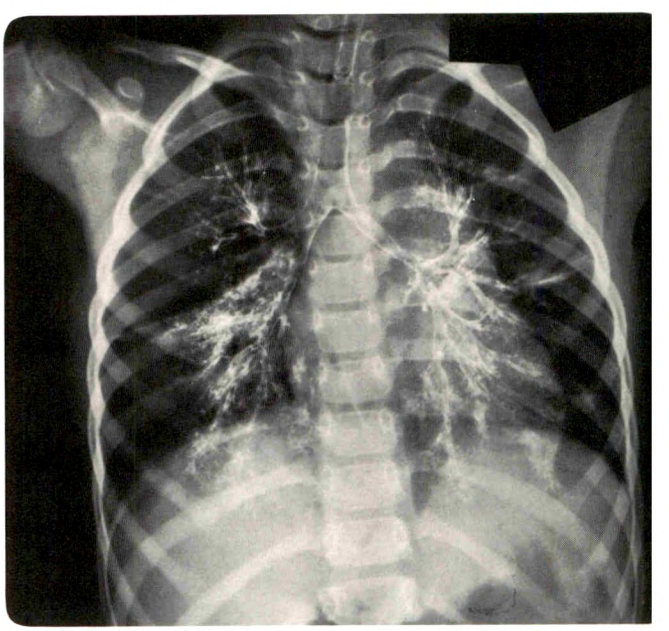
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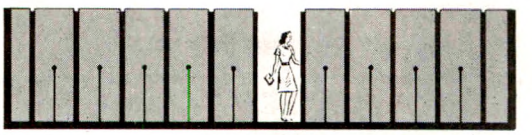


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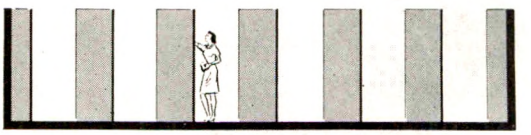
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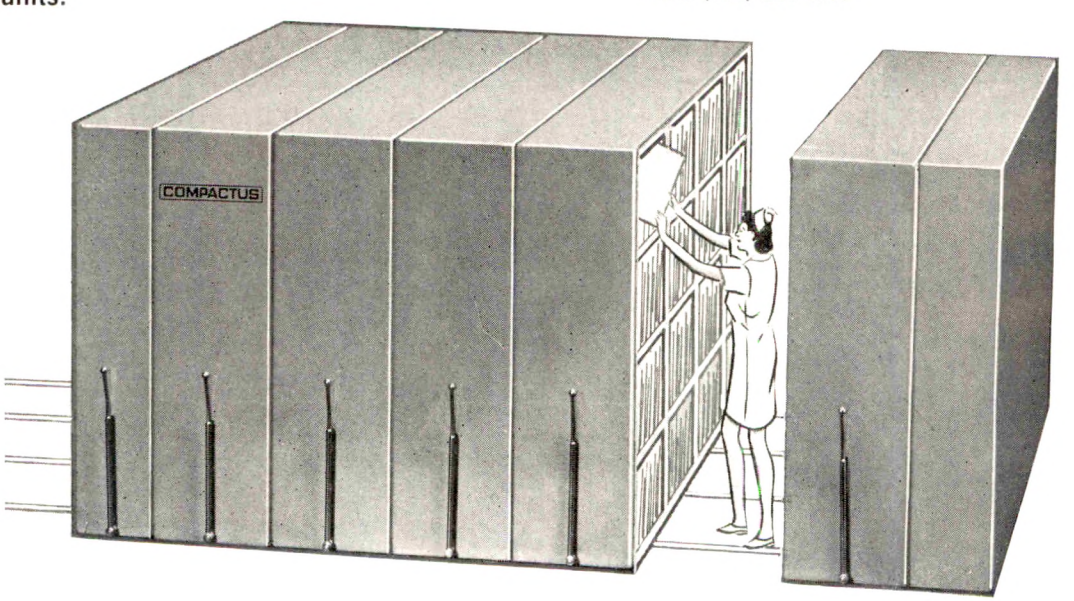
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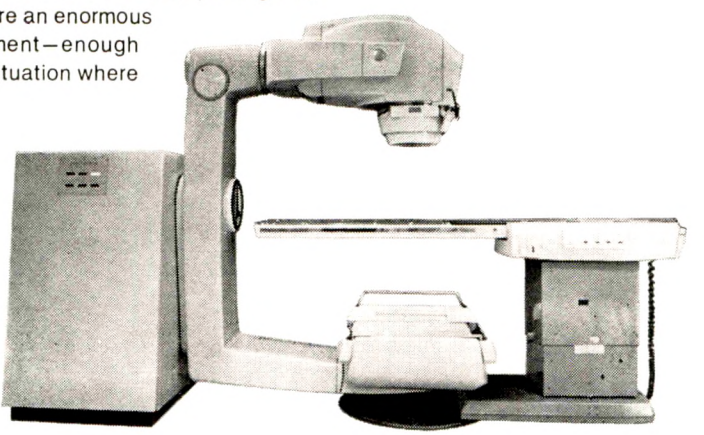
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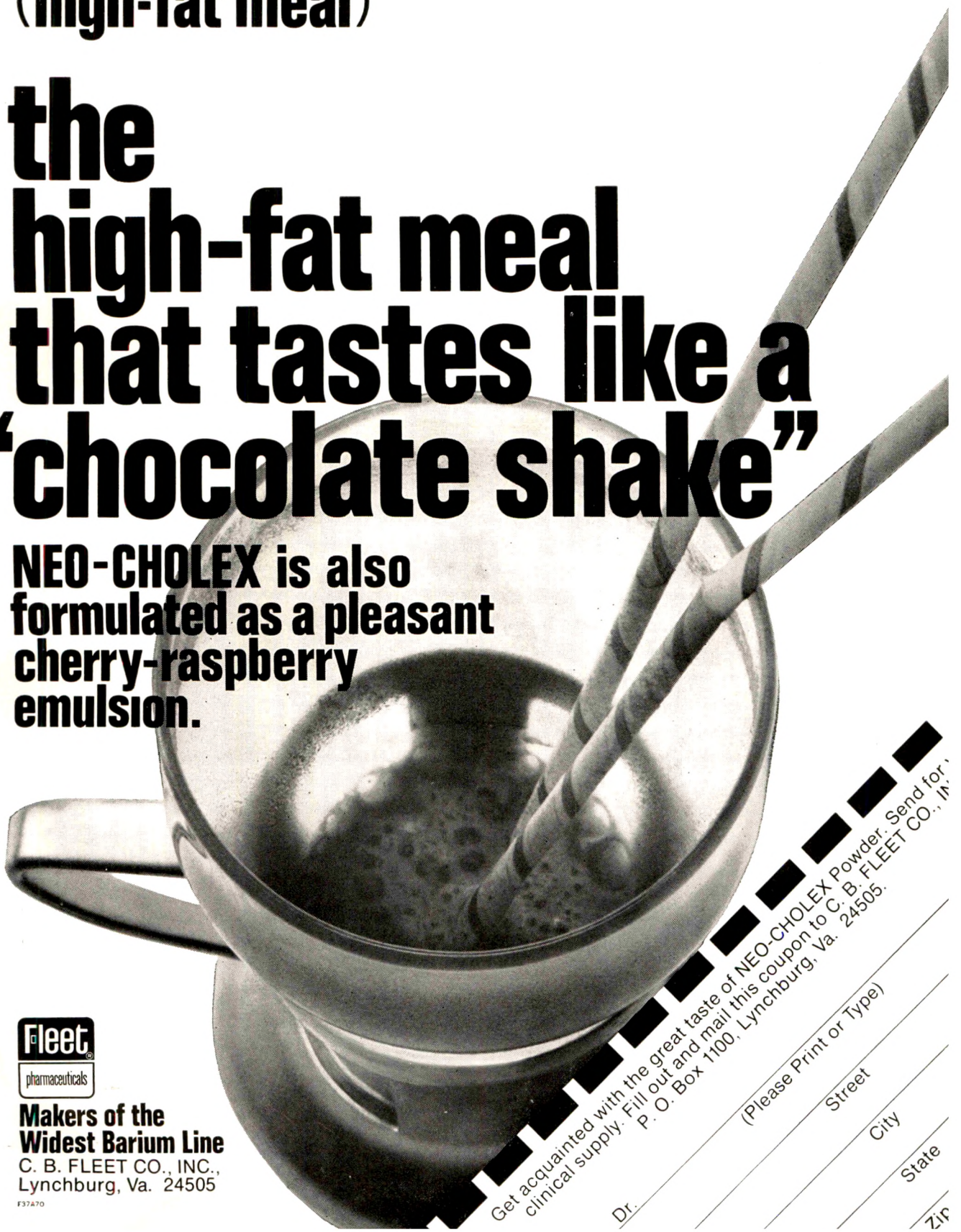
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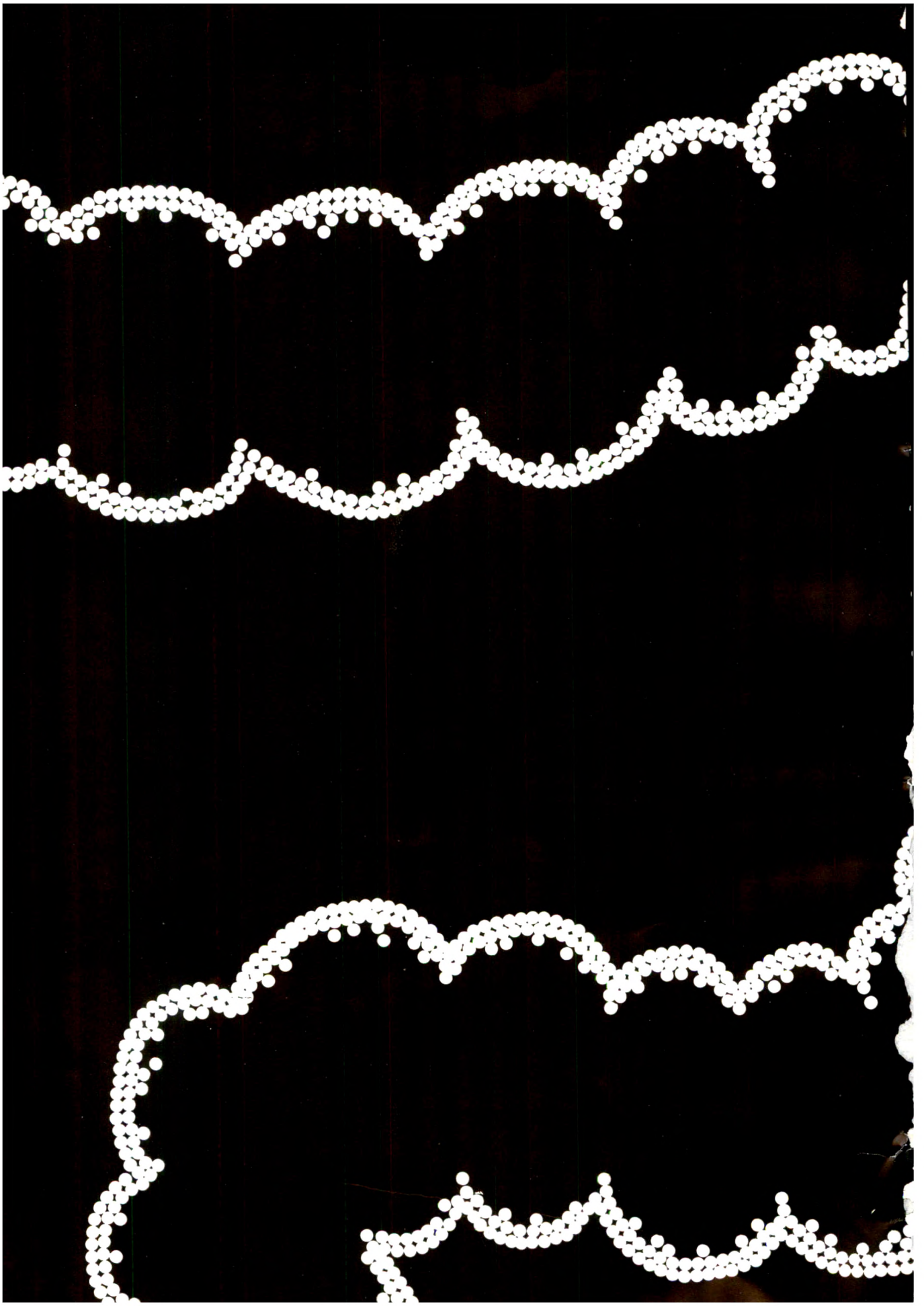
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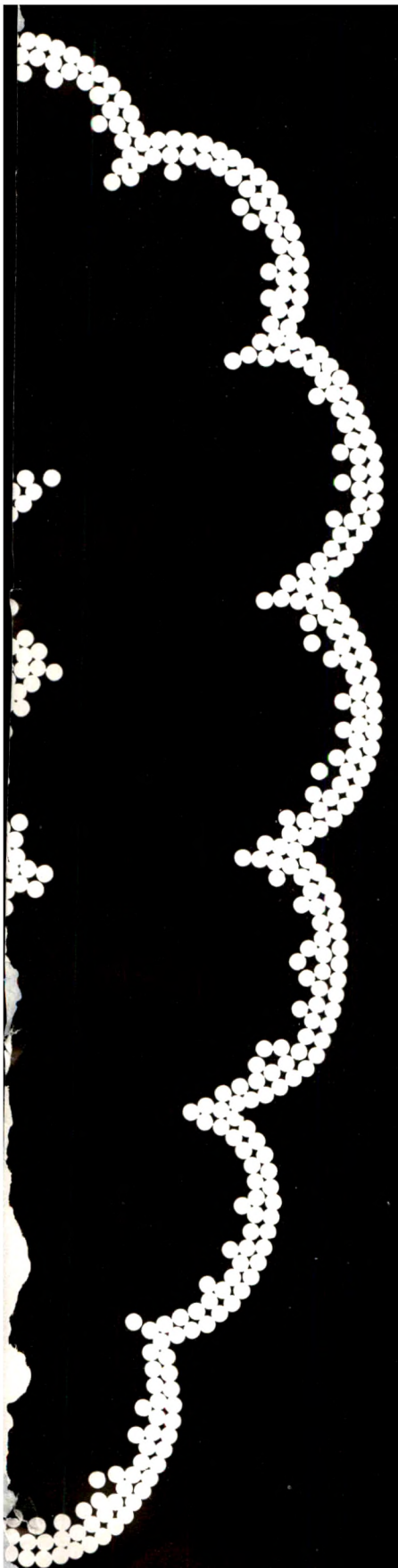
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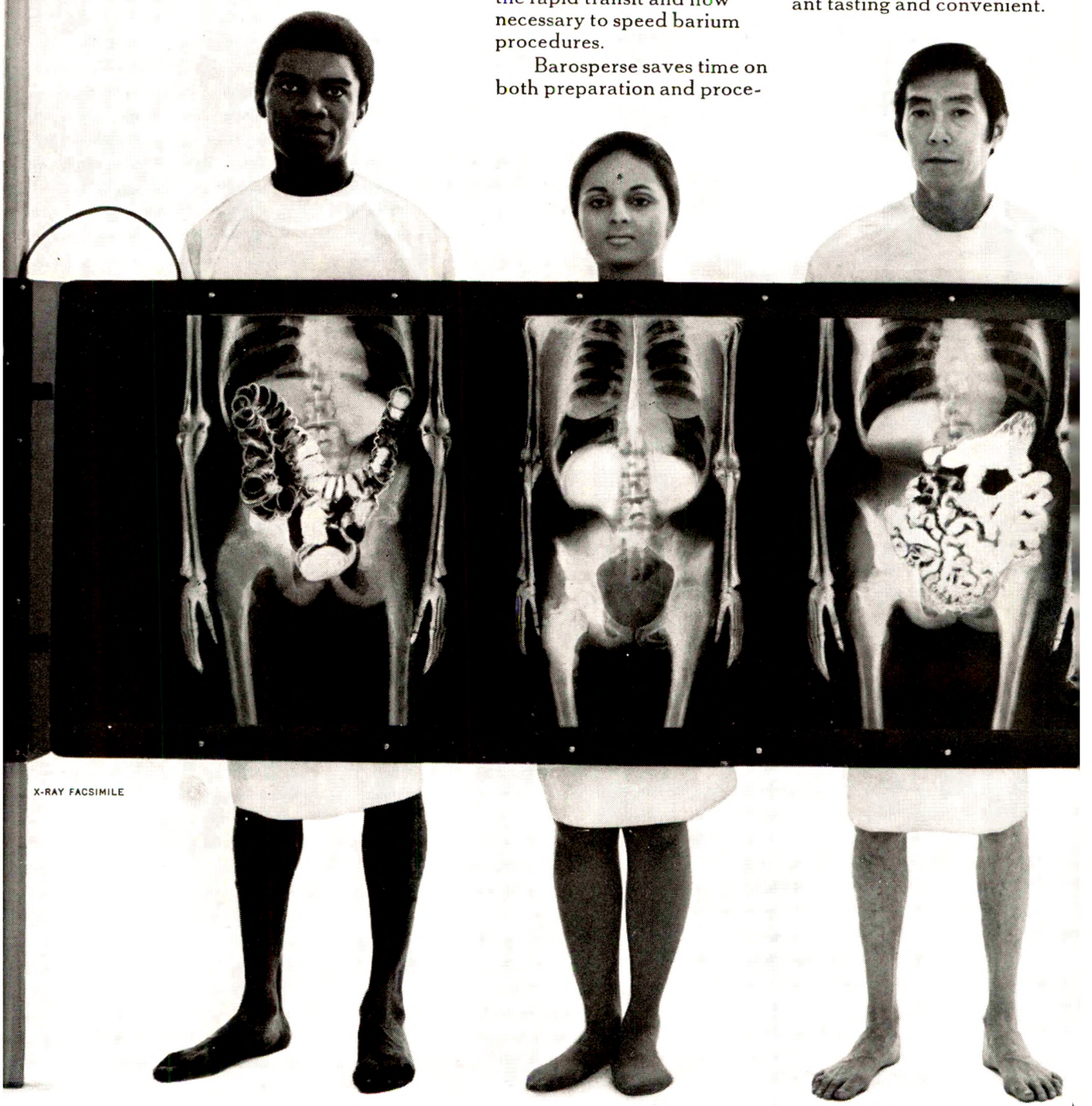
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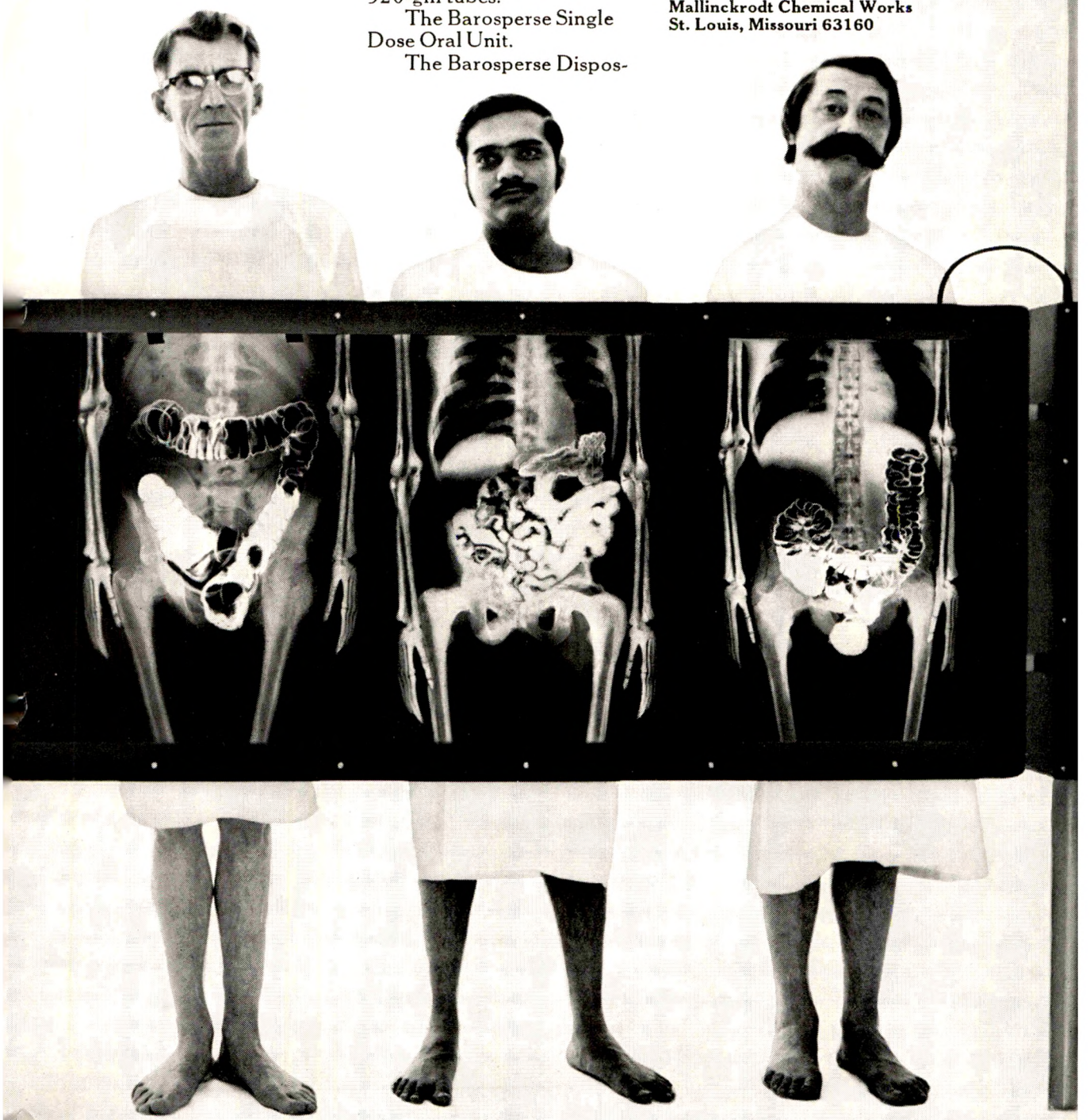
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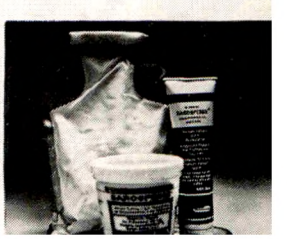
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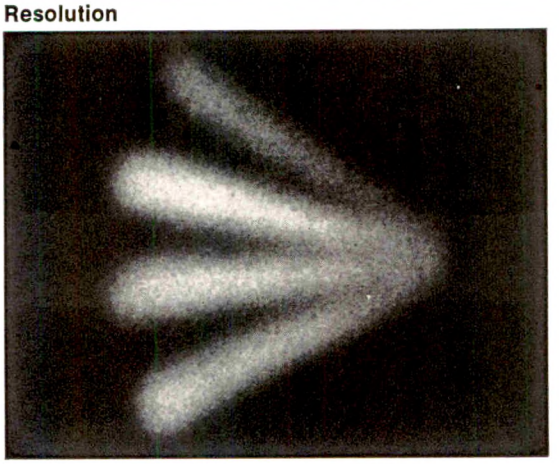


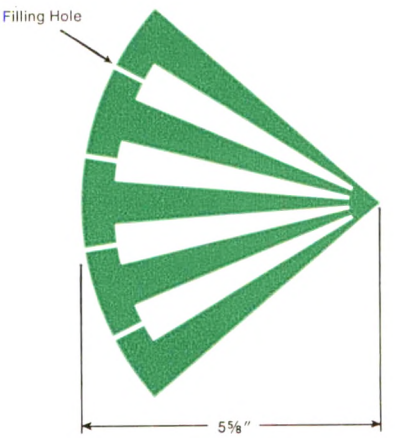
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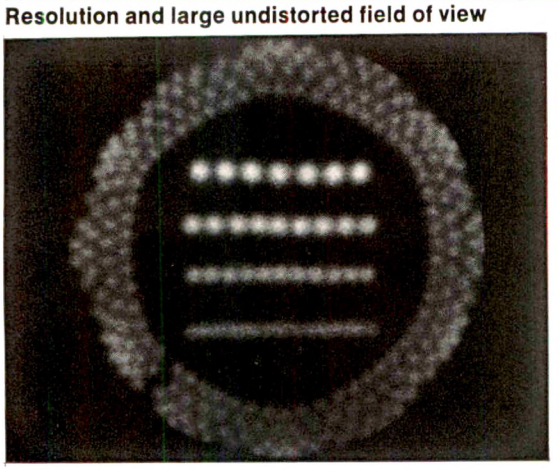
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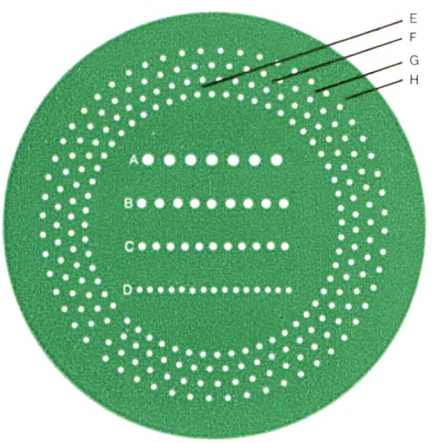




Phantom description: 3/8″ thick lucite with four 1/8″ thick radiating voids filled with activity.

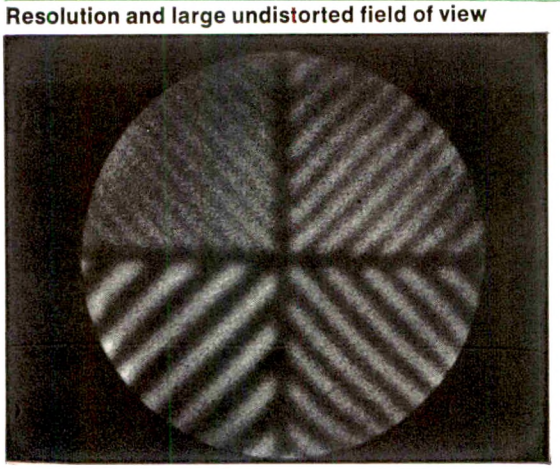
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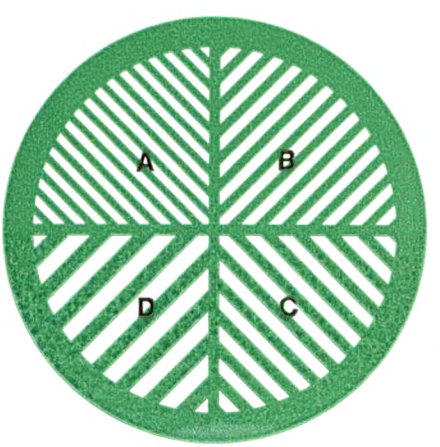




Phantom description: 1/8″ thick by 15″ dia. lead circle mounted between two circular pieces of 1/8″ thick lucite.
A. 3/8″ dia. 3/8″ space
B. 5/16″ dia., 5/16″ space
C. 1/4″ dia., 1/4″ space
D. 3/16″ dia., 3/16″ space
E. 3/16″ dia. holes with centers on 9″ dia. circle.
F. 3/16″ dia. holes with centers on 10″ dia. circle.
G. 3/16″ dia. holes with centers on 11″ dia. circle.
H. 3/16″ dia. holes with centers on 12″ dia. circle.

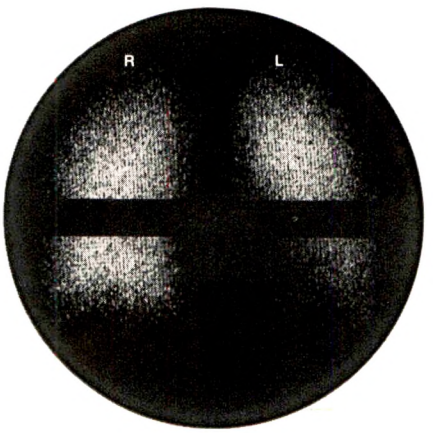
Resolution and large undistorted field of view

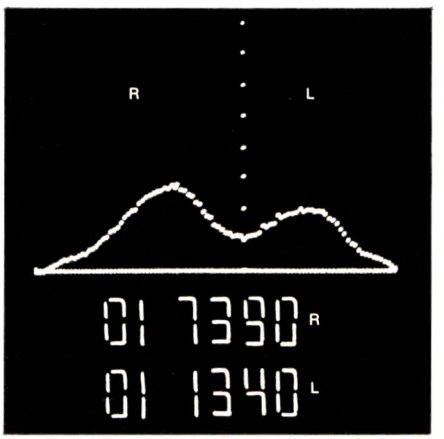




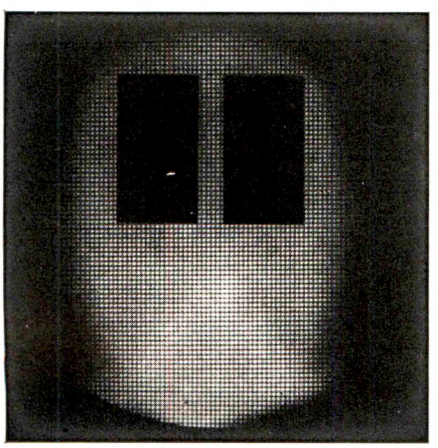
Phantom description: 1/8″ thick lead bars mounted between two circular pieces of 1/8″ thick lucite. A 14″ outside diameter, 1″ wide, lead ring surrounds the bars.
A. 1/4″ bars, 1/4″ spaces
B. 5/16″ bars, 5/16″ spaces
C. 3/8″ bars, 3/8″ spaces
D. 1/2″ bars, 1/2″ spaces

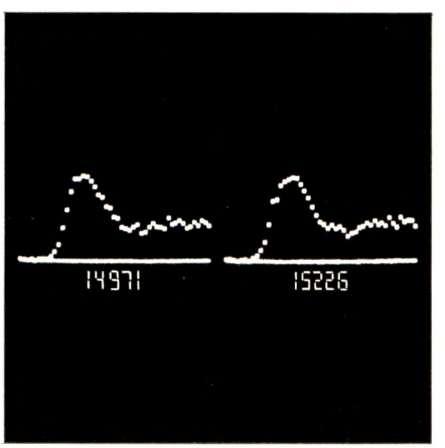
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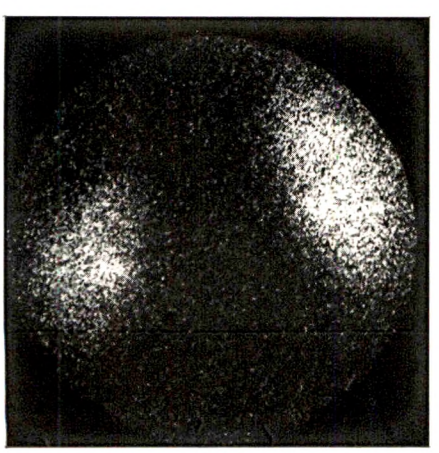


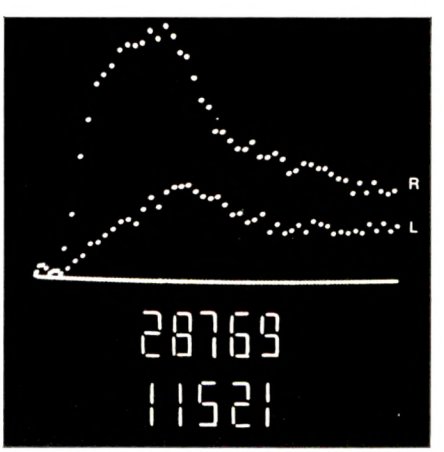
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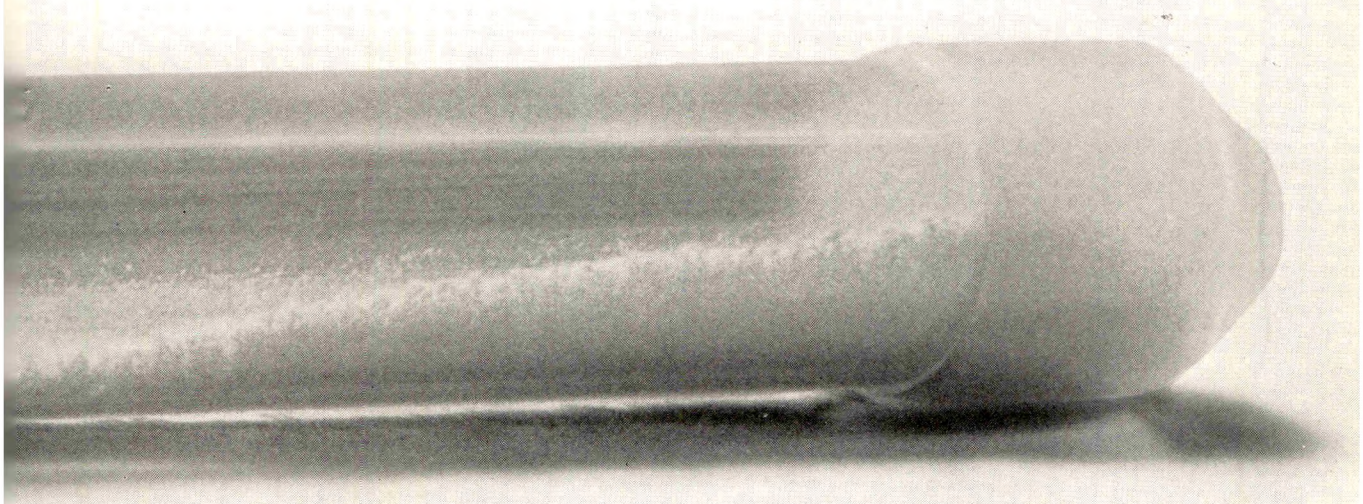
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IMPORTANT

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*Causes other than disease have occasionally been reported for nonvisualizing gallbladders.

Contraindications: Contraindicated in advanced hepatorenal disease or severe impairment of renal function, severe gastrointestinal disorders that prevent absorption, and in some patients sensitive to iodine compounds.

Precautions: Severe, advanced liver disease may interfere with metabolism of Telepaque, thus increasing the excretory load on the kidneys. Although renal difficulty has rarely been attributed to Telepaque, renal function should be assessed before cholecystography in severe, advanced liver disease, and renal output and hepatic function should be observed for a few days after the procedure. Patients with preexisting renal disease should not receive high doses of cholecystographic media. Possible renal irritation in susceptible individuals could result in reflex vascular spasm with partial or complete renal shutdown. Caution is advised in patients with coronary disorders, especially those with recent symptoms of coronary artery disease. Blood pressure should be observed after administration of cholecystographic media to these patients. Elevation of protein-bound iodine for several months and false positive urine albumin tests (for three days) may occur after ingestion of iodine-containing cholecystographic media.

Adverse Reactions: Most reactions are mild and transitory; serious side effects are very rare. Gastrointestinal effects (diarrhea, cramps, nausea, vomiting) are the most common. Usually the diarrhea consists merely of a few loose stools, although rarely it may be severe. A mild stinging sensation during urination may occur, and rarely, skin rash, urticaria, pruritus, and flushing. One case of thrombocytopenia has been reported in a patient with a history of conjunctival hemorrhages. Subjective complaints have been: dryness of throat, burning on swallowing, heartburn, sore throat, dizziness, and headache.

Usual Dosage: 3 Gm. (6 tablets) at night after a strictly fat-free supper.

Supplied: Tablets of 500 mg., envelopes of 6 tablets, boxes of 5 and 25 envelopes; also bottles of 500.

Telepaque® for precise oral cholecystography and cholangiography

brand of **iopanoic acid, USP**

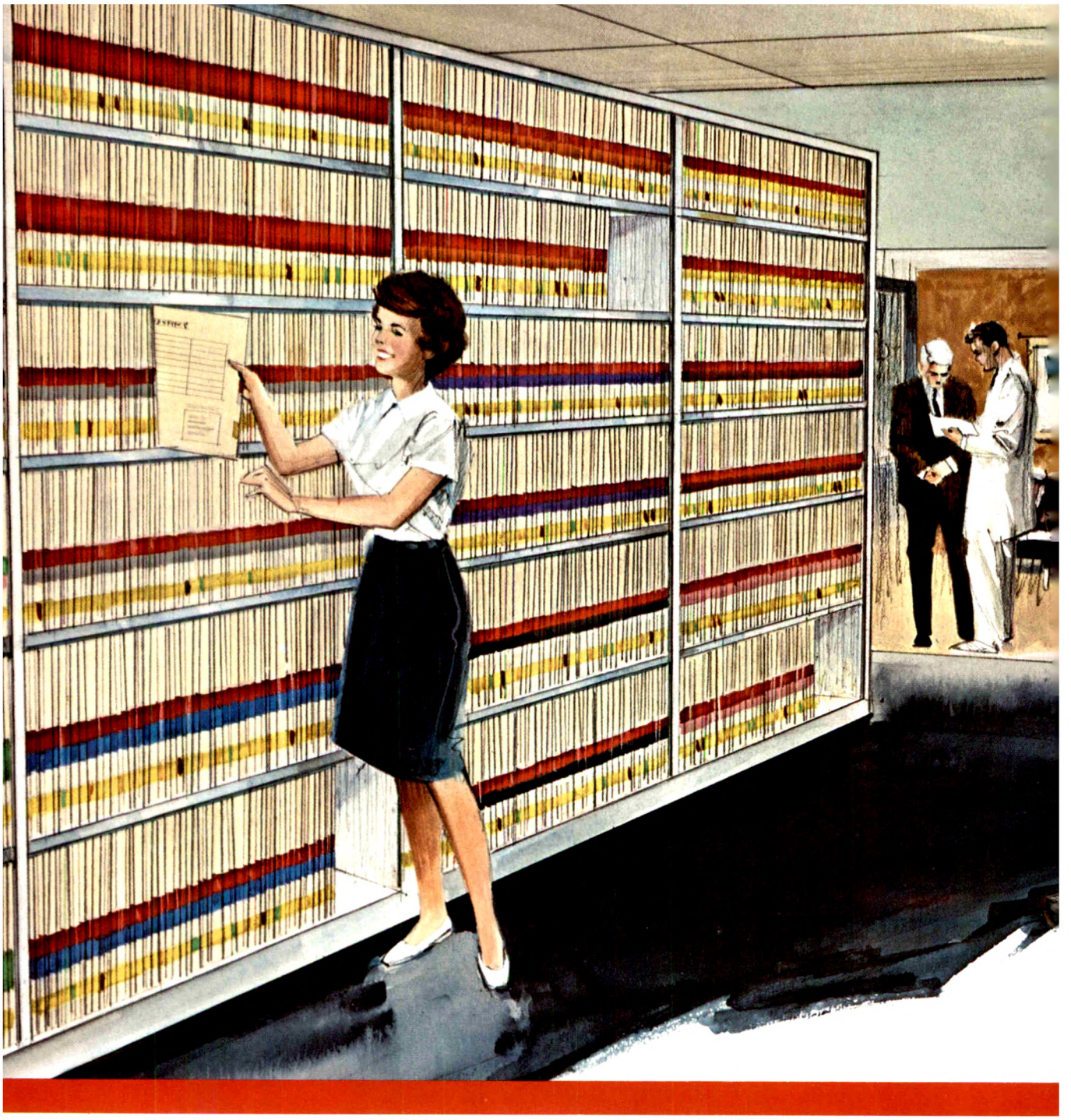
MORE THAN 40,000,000 DOSES OF TELEPAQUE HAVE BEEN ADMINISTERED* WITH AN UNPARALLELED RECORD OF CLINICAL SUCCESS

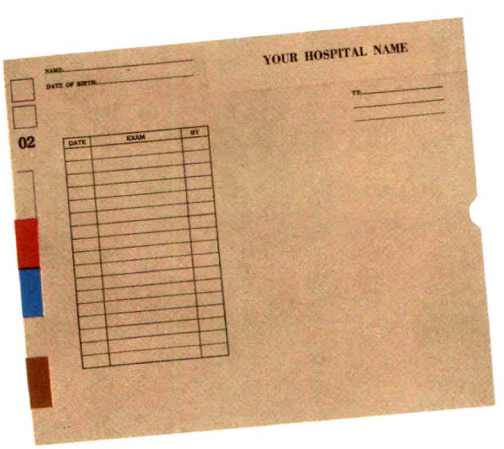
*Number of doses administered since the introduction of Telepaque

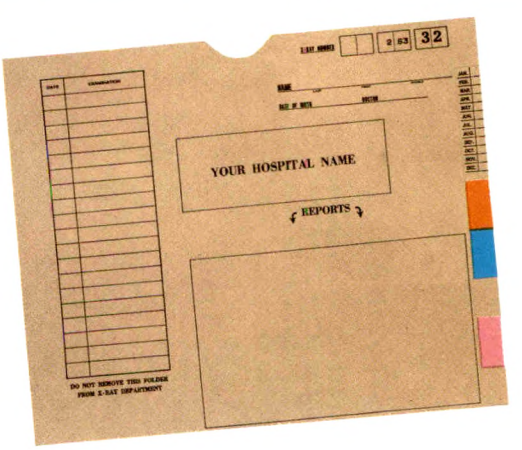
Winthrop **PIONEERS IN PRODUCTS FOR RADIOLOGIC DIAGNOSIS**

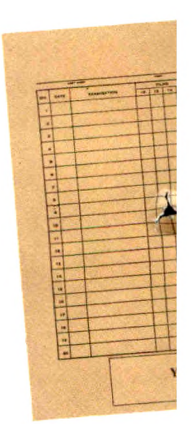
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NEW YORK, N.Y. 10016

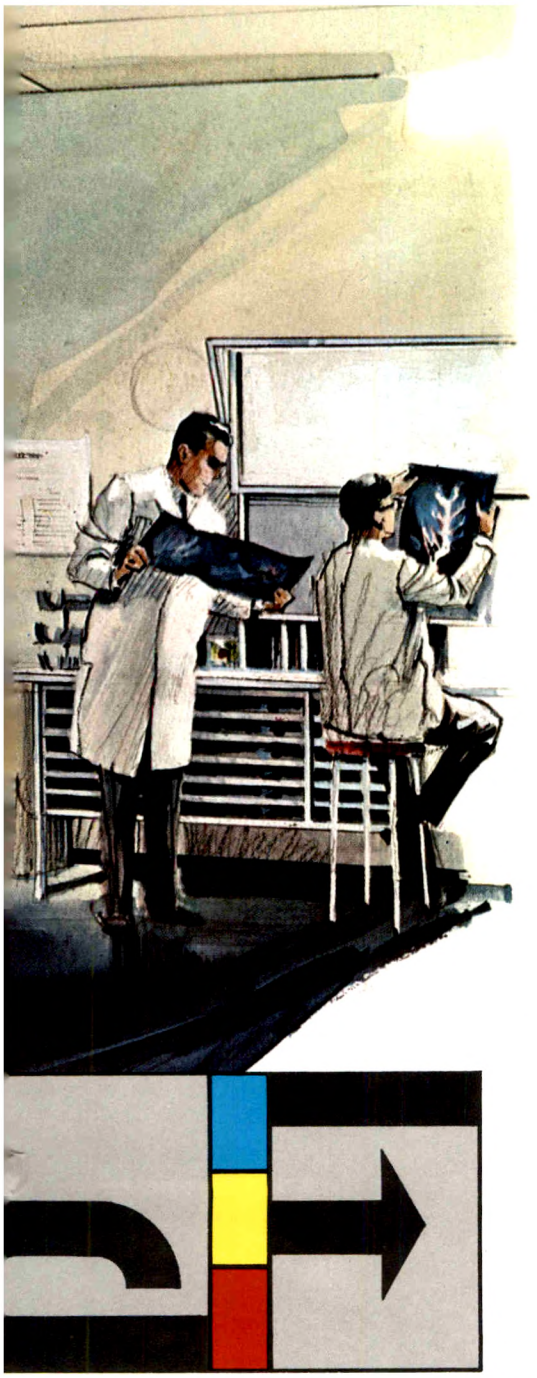
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AMES COLOR-FILE eliminates mis-files in the Radiology Department

Color Coded X-Ray Jackets

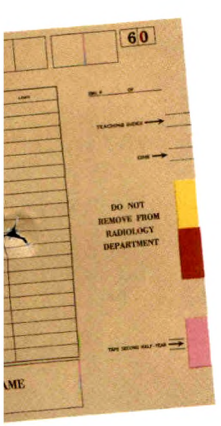
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Made from a durable tag, these jackets are printed in 10 colors in either two positions or three positions for terminal digit filing. The year code is also printed in color. Jackets are either pre-numbered with full unit number (when x-ray department issues its own number) or terminal digits only (when x-ray department uses hospital unit number). Terminal digits only may also be printed for use with Social Security numbers. Special pockets may be attached for reports.

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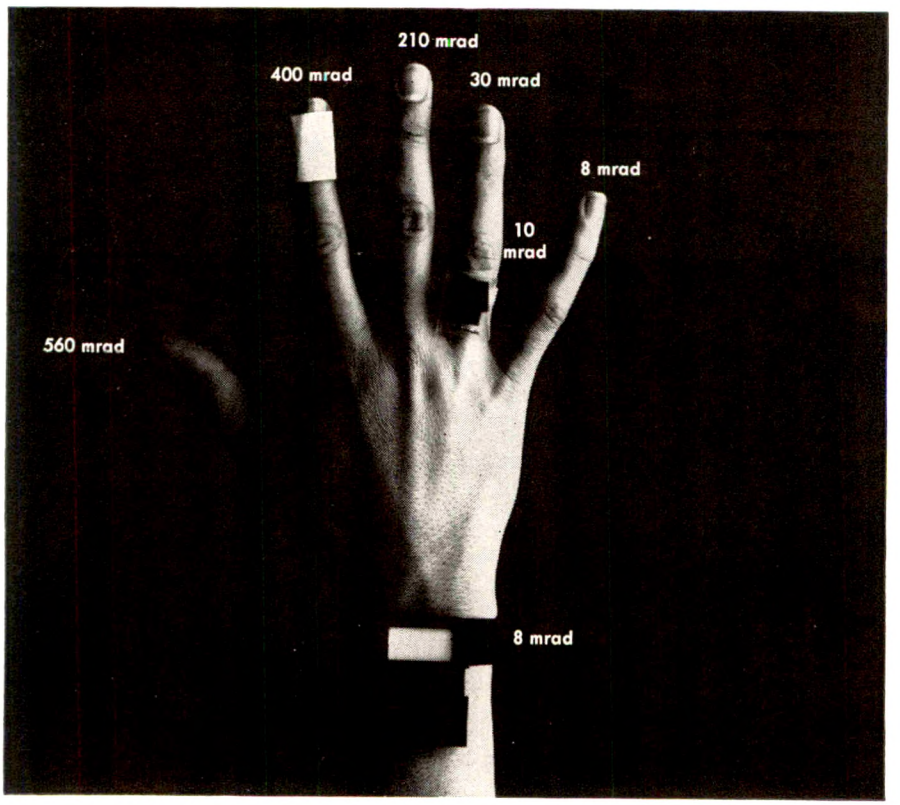
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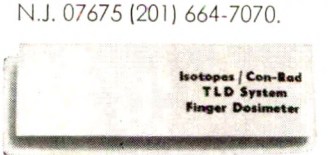
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Reasons enough, we think, to write or call ISOTOPES for full information. 50 Van Buren Avenue, Westwood, N.J. 07675 (201) 664-7070.

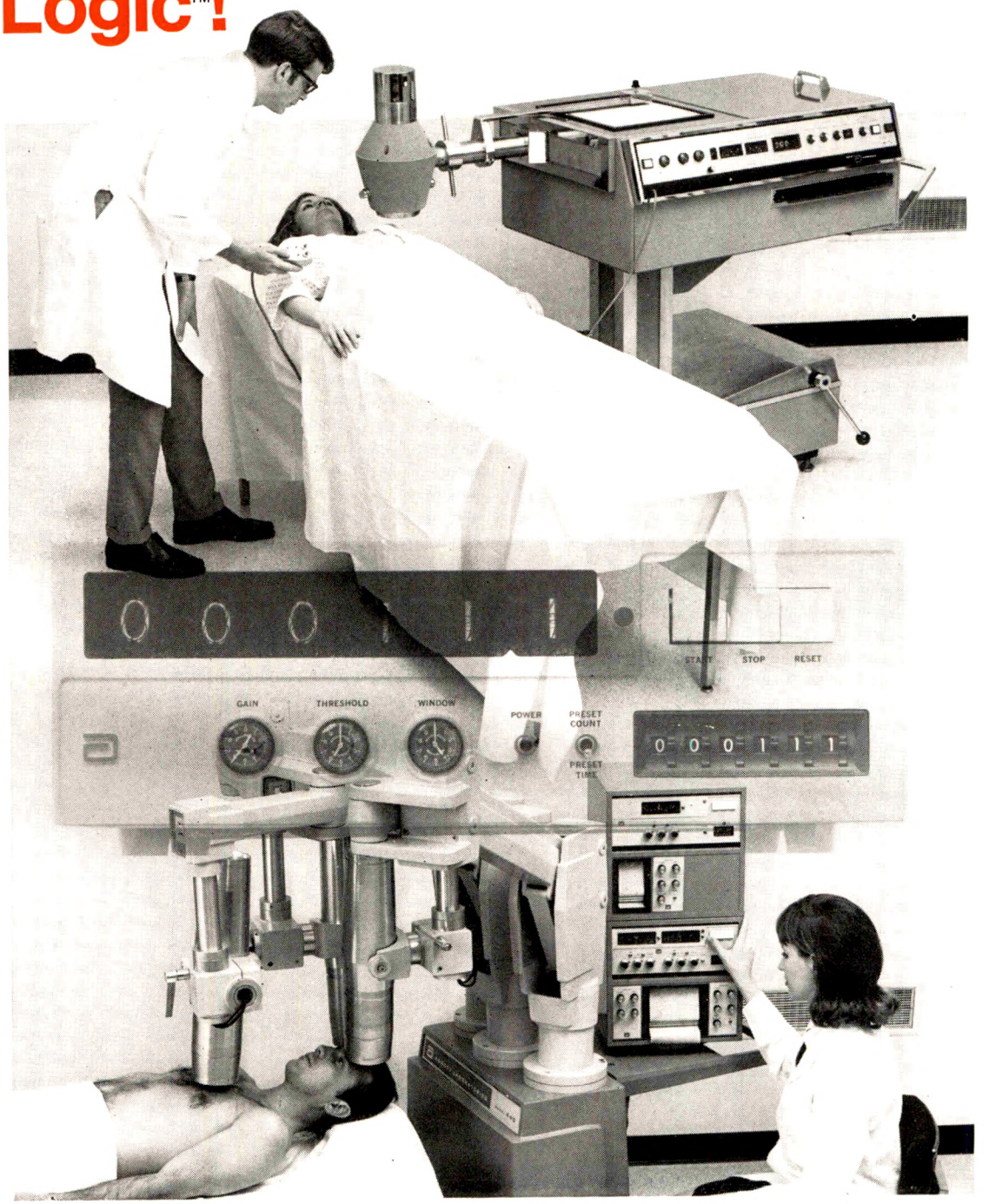


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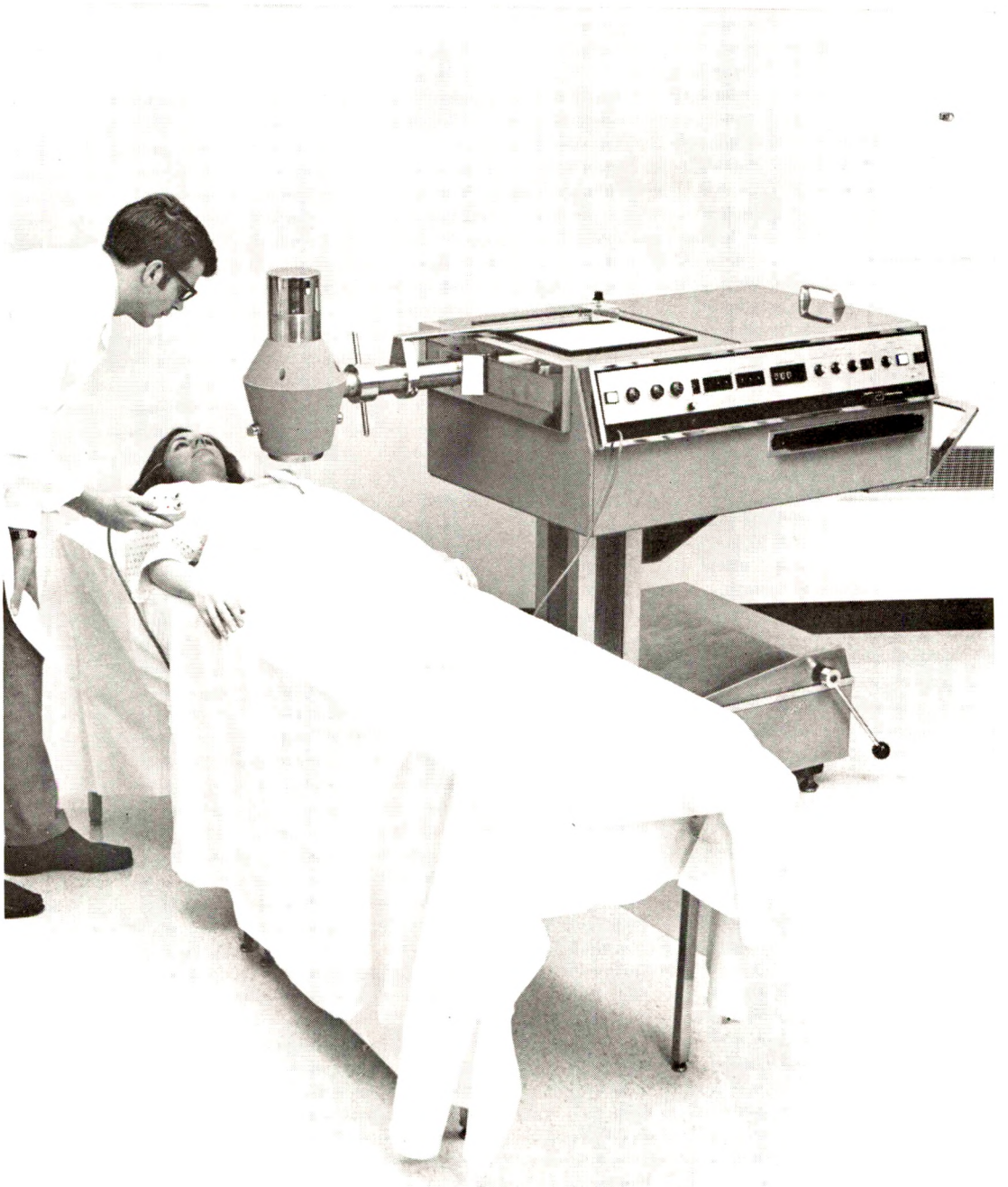
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. . . variable from 10 to 1,000 cm./minute with appropriate fixed index level. 1,000 cm. minute makes it the fastest scanner available. Portal to portal patient time may be less with some studies than with camera devices.

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. . . system is readily portable and can be easily moved on its 5″ casters. Will fit through any standard door opening. It is the only scanner that can easily be taken right to the patient in his hospital bed.

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. . . technician can master scanner operation within 30 minutes. Remote control for detector positioning. Detector angle may be adjusted 360°.

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. . . 17″ x 14″ in either direction. Allows for easier patient set-up to scan large lung fields, liver, spleen, etc. Can scan 17″ laterally or horizontally (important in obese patients). Uses standard 14″ x 17″ x-ray film.

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. . . the Graphic is available with either a 3″ or a 5″ detector head. Can be converted easily in the hospital from a 3″ to a 5″ scanner.

Versatile

. . . five levels of contrast enhancement including digital mode and six levels of background erase.

Integrated circuitry

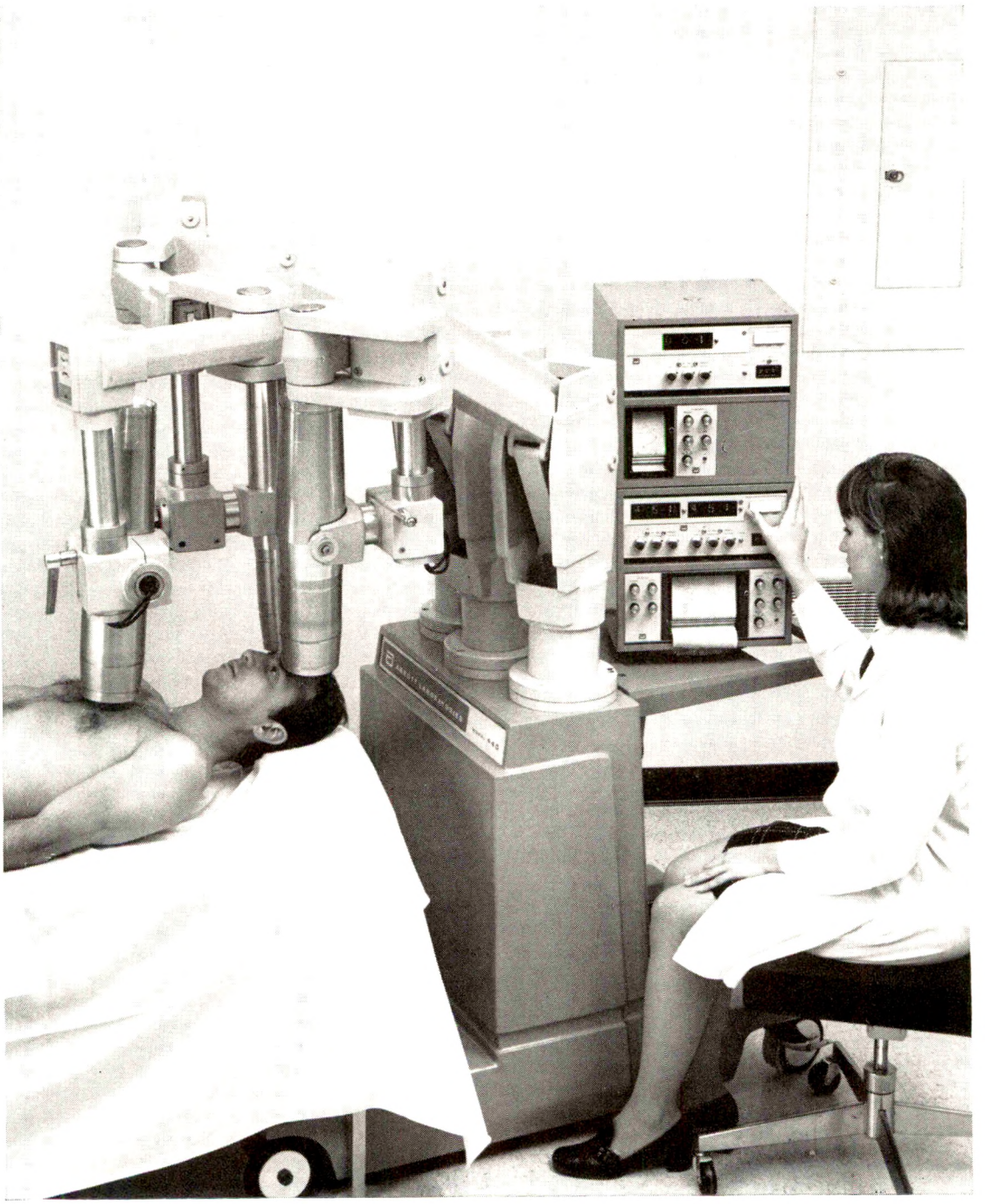
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. . . 0.1 millimeter/second, 1.0 millimeter/second, 10 millimeters/second, and 20 millimeters/second which are keyed to the circulation times relating to dynamic function studies of the important organs of the body. Included are the brain, lungs, heart, and kidneys.

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. . . the Dynamic system is entirely digital in operation.

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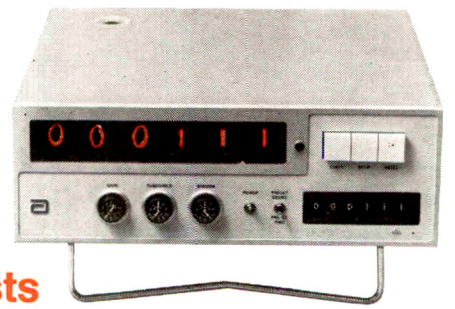
. . . in each of the strip chart recorders eliminates the messy chore of ink changing. This prevents blurred information as well as smudged fingers.

Choice

. . . of one, two, and three detector systems. 1.5 x 1.5 inch sodium iodide detectors mounted on electrically operated arms. This modular concept allows you to add on as your needs expand.

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...for in vitro and in vivo tests!



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. . . including T-3, T-4, thyroid uptake*, hepatic uptake*, plasma volume, fecal Rose Bengal excretion, iron binding, fat absorption, and placenta localization*.

Speed of electronics

. . . count and display in excess of 15,000,000 counts per minute.

Solid state integrated circuitry

. . . assures higher reliability; less down time.

Simple to operate

. . . minimum of controls with Direct Ratio Readout in %.

Choice of 3 models

. . . 101 and 111 have spectrometer and well in one instrument. The 121 has an external well.

Fast service

. . . with easy-to-use service manual; replacement boards in 24 hours. There's no waiting for servicemen.

Modular concept

. . . with built-in versatility. Protect your investment by adding components as the need arises.

*May be done by adding medical stand, external probe (shield and collimator).

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This product diversity is not a happenstance. The Picker Medical Products Division —we've chosen a new name to reflect the breadth of our involvement—will continue to expand into whatever areas provide useful tools for health care, be they complex diagnostic instruments or everyday supply items. The point is that we're neither awed by the design, manufacturing, and servicing demands of the most sophisticated equipment, nor are we unaware of the basic importance of the less prestigious tools of the trade.

But what does such a product panorama mean to the user? Is this just diversity for diversity's sake?

Hardly. Product diversity leads to more than a thick catalog: it makes *systems* possible. Individual products can begin to coalesce into interrelated, coordinated, compatible *systems*. This voids the traditional user problem of integrating separate products of dubious compatibility. Likewise, it voids the need for multiple servicemen working on multiple products with no one accepting the total responsibility for the *only* thing the user really cares about: an efficient, functioning, problem-solving system. An unobtrusive system, if you will.

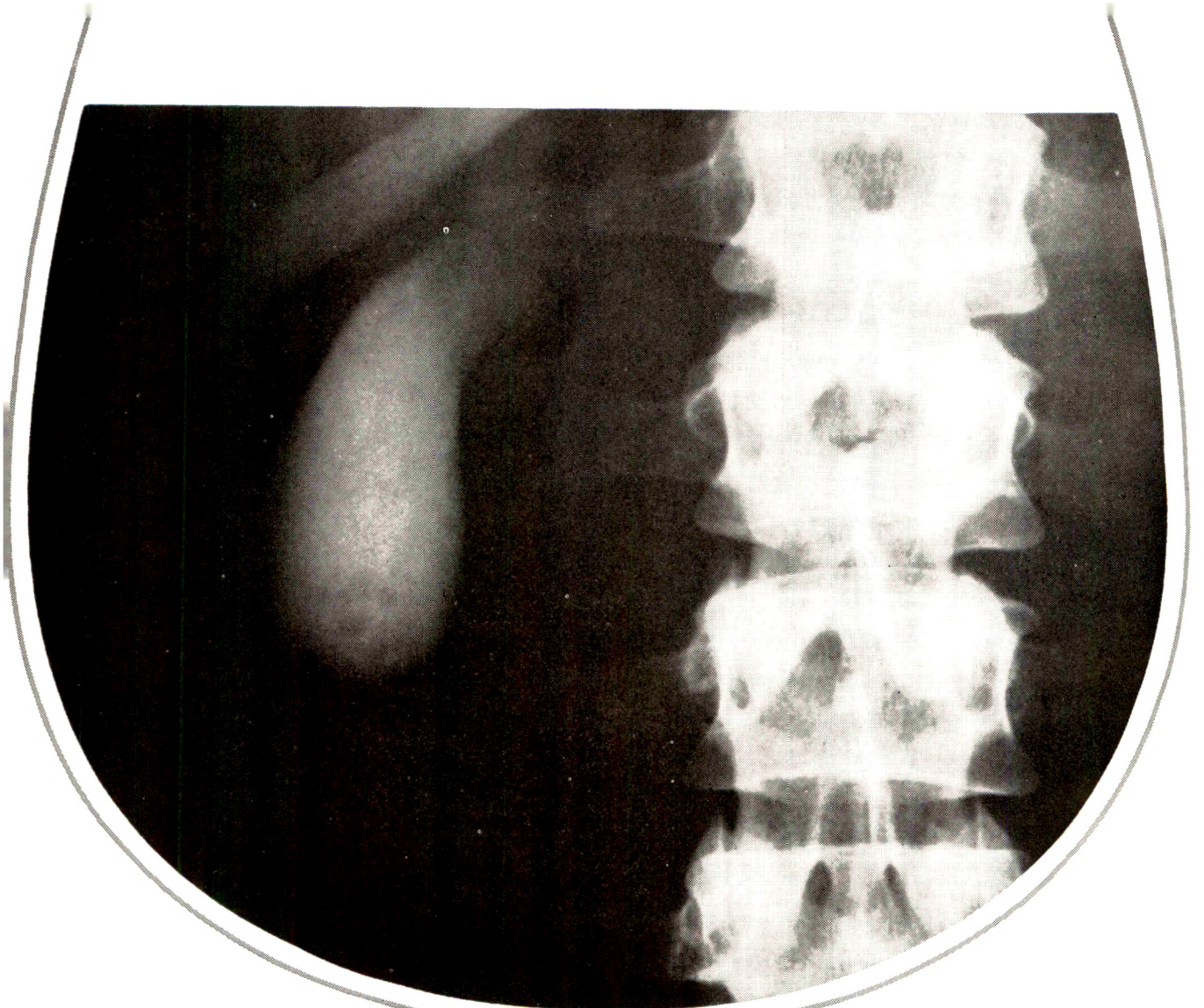
So we acknowledge the user's desire for *total* solutions to problems rather than for a miscellany of individual products. Accordingly, we point ourselves in this direction:

> *To design and build complete systems that will satisfy the user's needs for information, simplicity, practicality, economy, and reliability.*
> *To test those systems as systems*
> *To install and to instruct as needed.*
> *And, finally, to take the sole responsibility for servicing everything we sell.*

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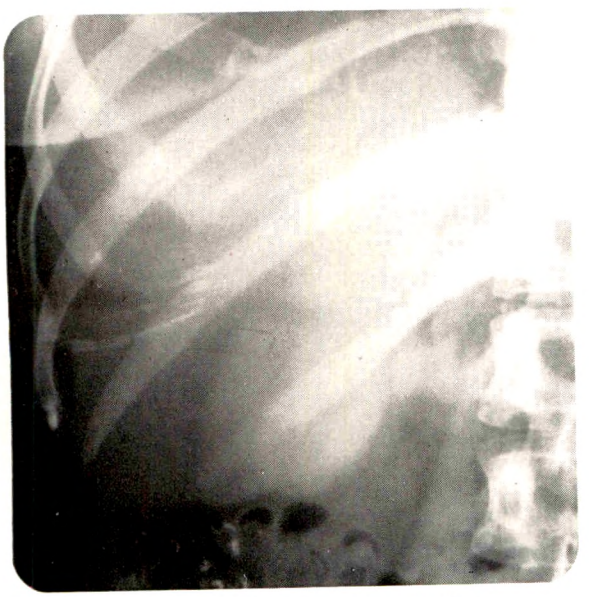
LXC

A nonvisualized gallbladder...



can often be visualized with Oragrafin® Calcium Granules

Calcium Ipodate



Cholecystogram 12 hours after routine dose of iopanoic acid.[1]



Cholecystogram 3 hours later after dose of 6 Gm. of calcium ipodate granules. Note filling of the common duct—from Root[1], discussion on the following page.

When surgery hangs on diagnostic films...

Calcium and sodium ipodate are rapidly absorbed from the gastrointestinal tract and begin to appear in the bile with striking rapidity. The abundant delivery to the liver and biliary system permits visualization even in a poorly functioning gallbladder. In addition, use of calcium ipodate granules permits same-day re-examination, should a second visualization be deemed necessary.

See next page for "Brief Summary."

Recent clinical study[1] demonstrated improved diagnoses

"A group of 114 patients with poorly filled gallbladders after routine administration of iopanoic acid were given 6 gm. of [calcium] ipodate granules immediately following their routine cholecystography. In 66 per cent of these patients opacification of the gallbladder was obtained within 3 hours, allowing a definite diagnosis. Of the remaining 33 per cent, one-half were ultimately proven diseased."[1] For one of the patients in this group, see x-ray pictures on previous page.

"It is apparent that many poorly visualized gallbladders can be rendered visible by the ingestion of ipodate granules. In most instances, definite diagnosis can be made within 18 hours of the original administration of opaque medium [iopanoic acid] rather than waiting 36 hours. A whole day may be saved and other diagnostic studies may be performed."[1]

plus other important advantages

Less discomfort—In a study utilizing iopanoic acid and ORAGRAFIN Sodium Capsules (Sodium Ipodate) nausea, vomiting, diarrhea, cramps, and dysuria—all were less frequent with Oragrafin Sodium Capsules.[2] *Few side reactions*—McCrory[3] reported that Oragrafin Sodium and Calcium were "...used routinely in cholecystographic studies in approximately 2000 patients with excellent diagnostic films and only rare and mild reactions." *Greater convenience*—Routine overnight procedure with Oragrafin Sodium Capsules is easy for patients to follow. If necessary, rapid, same-day re-examination procedures possible with the calcium granules reduce the need for I.V. studies.

Contraindications: Contraindicated for persons sensitive to oral iodine compounds or for patients with *combined* renal and hepatic disease or *severe* kidney impairment. Gastrointestinal disorders, which may interfere with absorption, or liver dysfunction, which may result in inadequate biliary secretion of medium, are likely to result in unsatisfactory visualization.

Precautions and Side Effects: Mild and transient nausea, vomiting, or diarrhea sometimes occur; but the incidence can be reduced by using the calcium granules and restricting the dosage to 3 Gm. Transient headache, dysuria, or abdominal pains may occur.

Hypersensitivity reactions may include urticaria, serum sickness-like reactions, and rarely anaphylactoid shock. They are more likely to occur in the individual with a history of allergy, asthma, hay fever, or urticaria and in the individual who is known to be hypersensitive to iodine compounds. Antihistamines and corticosteroids are used to control hypersensitivity reactions; but the occasional serious anaphylactoid reactions require the immediate use of epinephrine or phenylephrine, oxygen, and intravenous corticosteroids.

For full information see Package Insert.

Supply: The calcium salt (Oragrafin Calcium Granules) is available in single-dose foil packets providing 3 Gm. of calcium ipodate as granules dispersed in flavored sucrose. The sodium salt is available in capsule form (Oragrafin Sodium Capsules) providing 0.5 Gm. sodium ipodate per capsule.

References: (1) Root, H. F., Jr.: Amer. J. Roentgen. *101*:621 (Nov.) 1967. (2) White, W. W., and Fischer, H. W.: Amer. J. Roentgen. *87*:745 (April) 1962. (3) McCrory, E.: J. Tenn. Med. Ass. *58*:258 (Aug.) 1965.

Oragrafin® Calcium Granules
Calcium Ipodate

Oragrafin® Sodium Capsules
Sodium Ipodate

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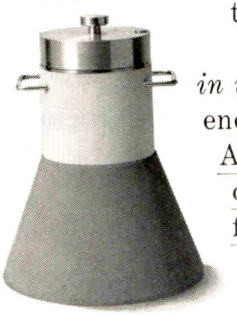
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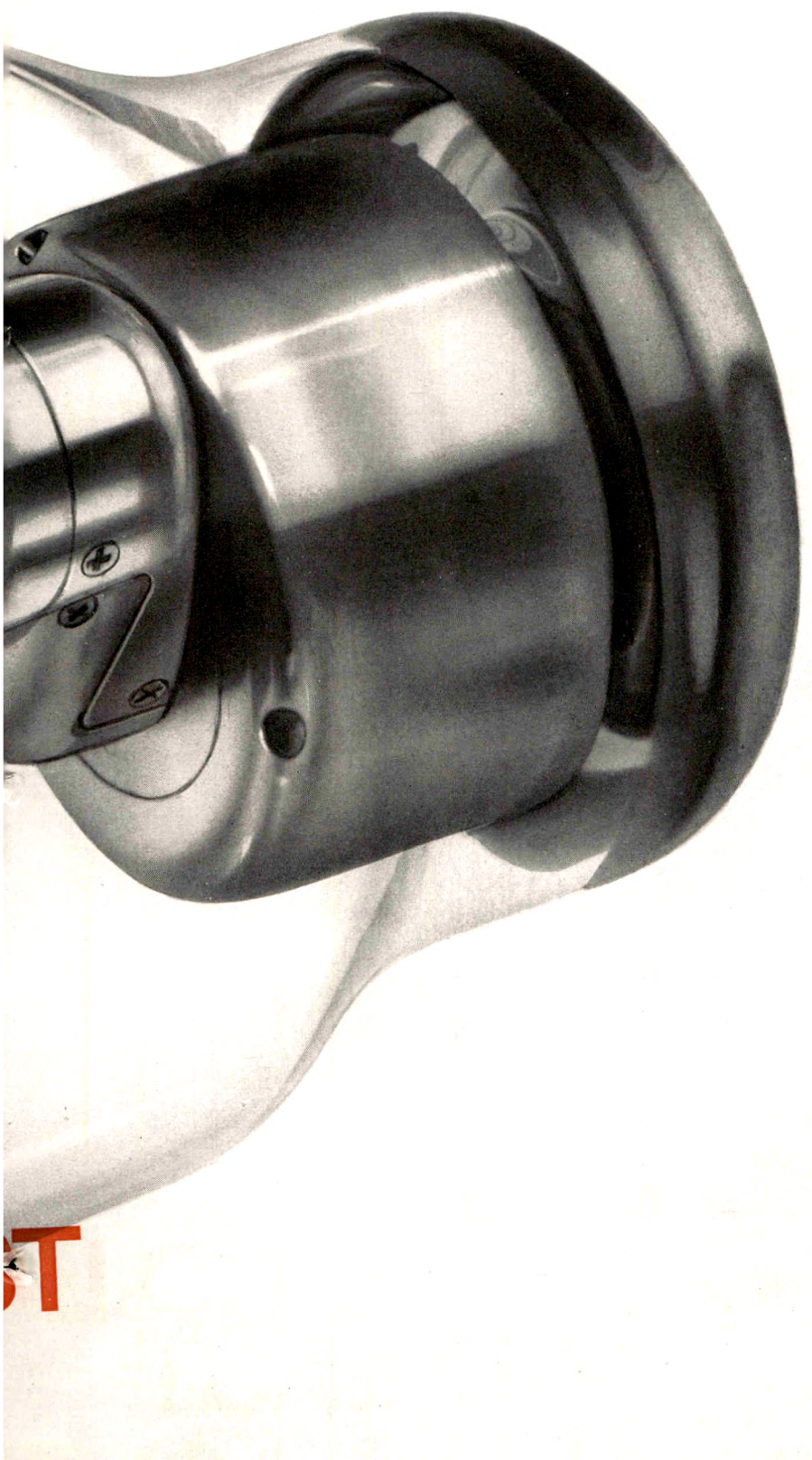
A NEW LINE OF HEAVY
ROTATING ANODE X-RAY
THAT LAST AND LAST

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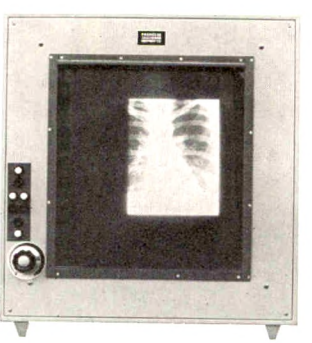
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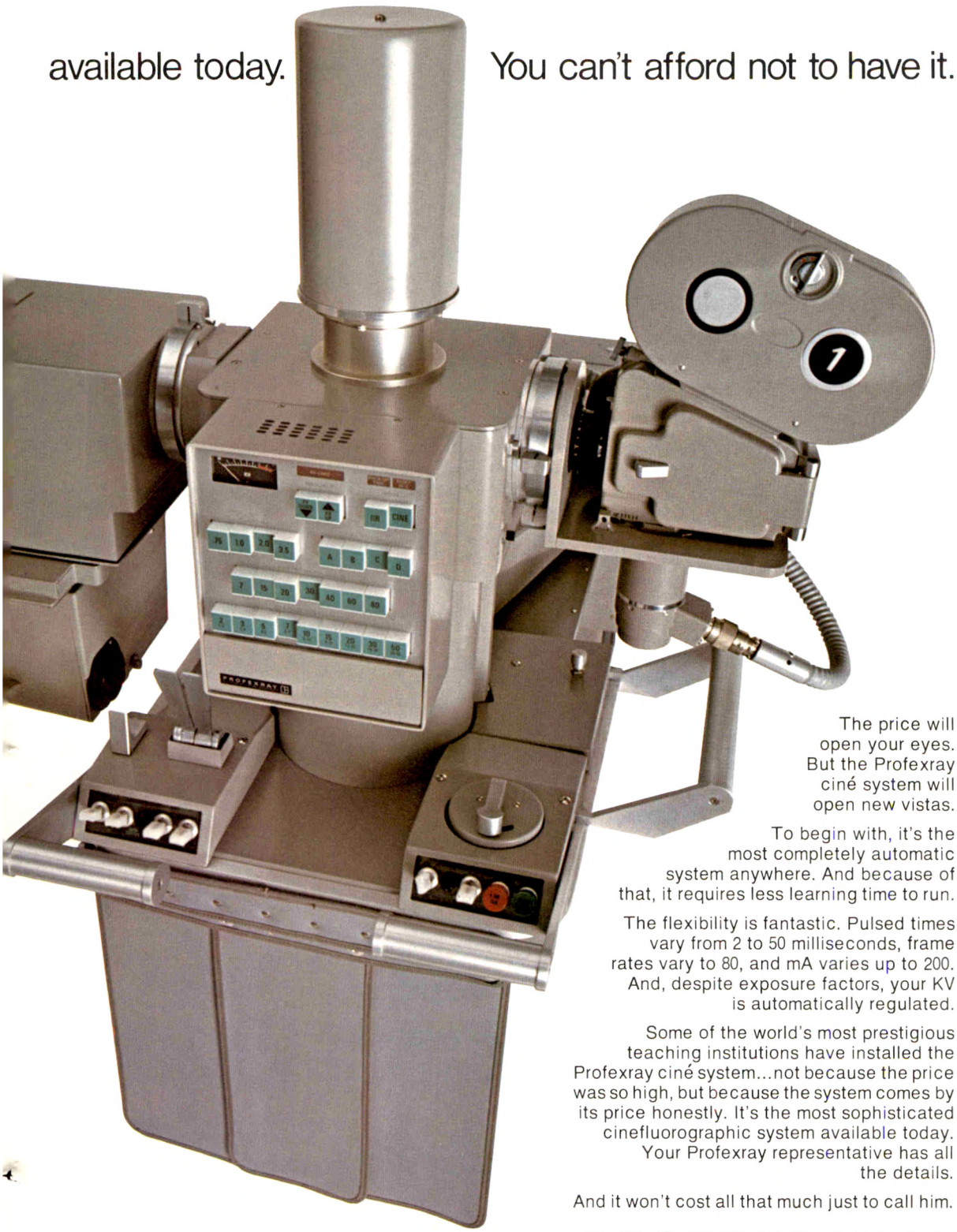
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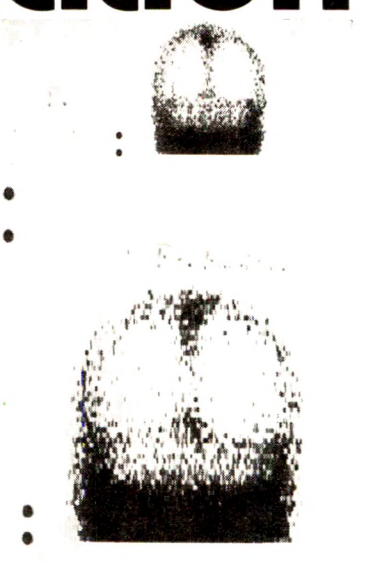
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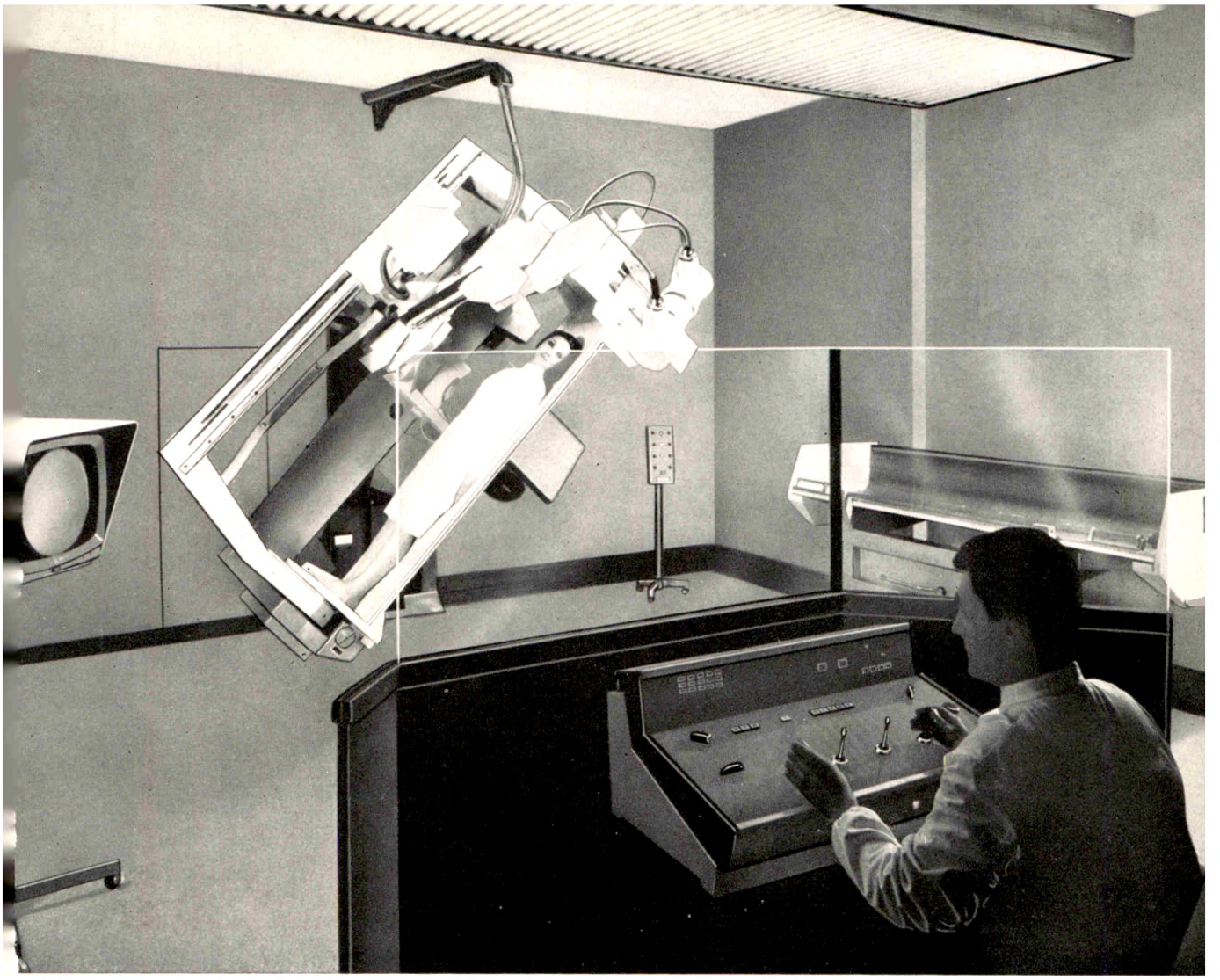
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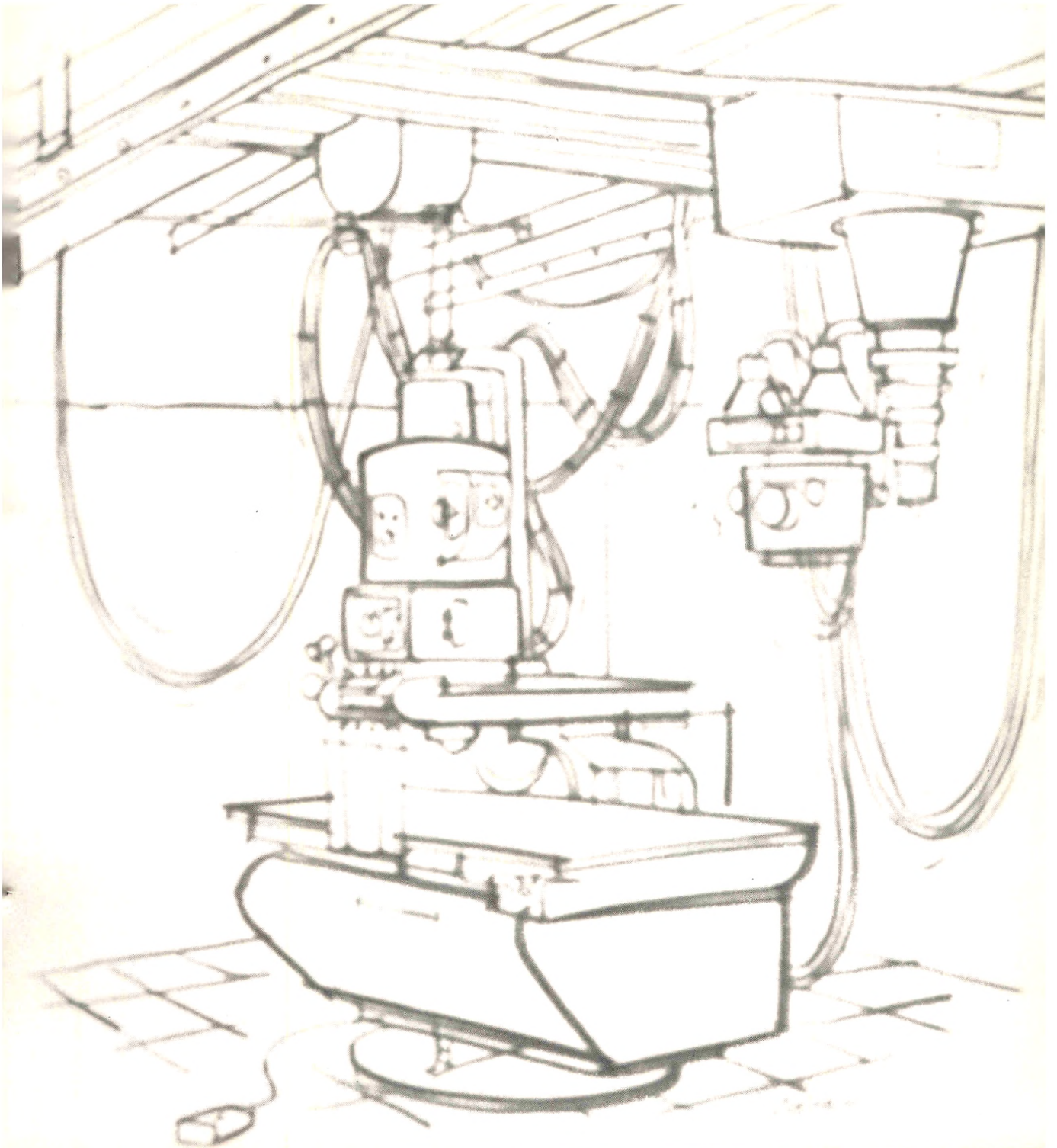
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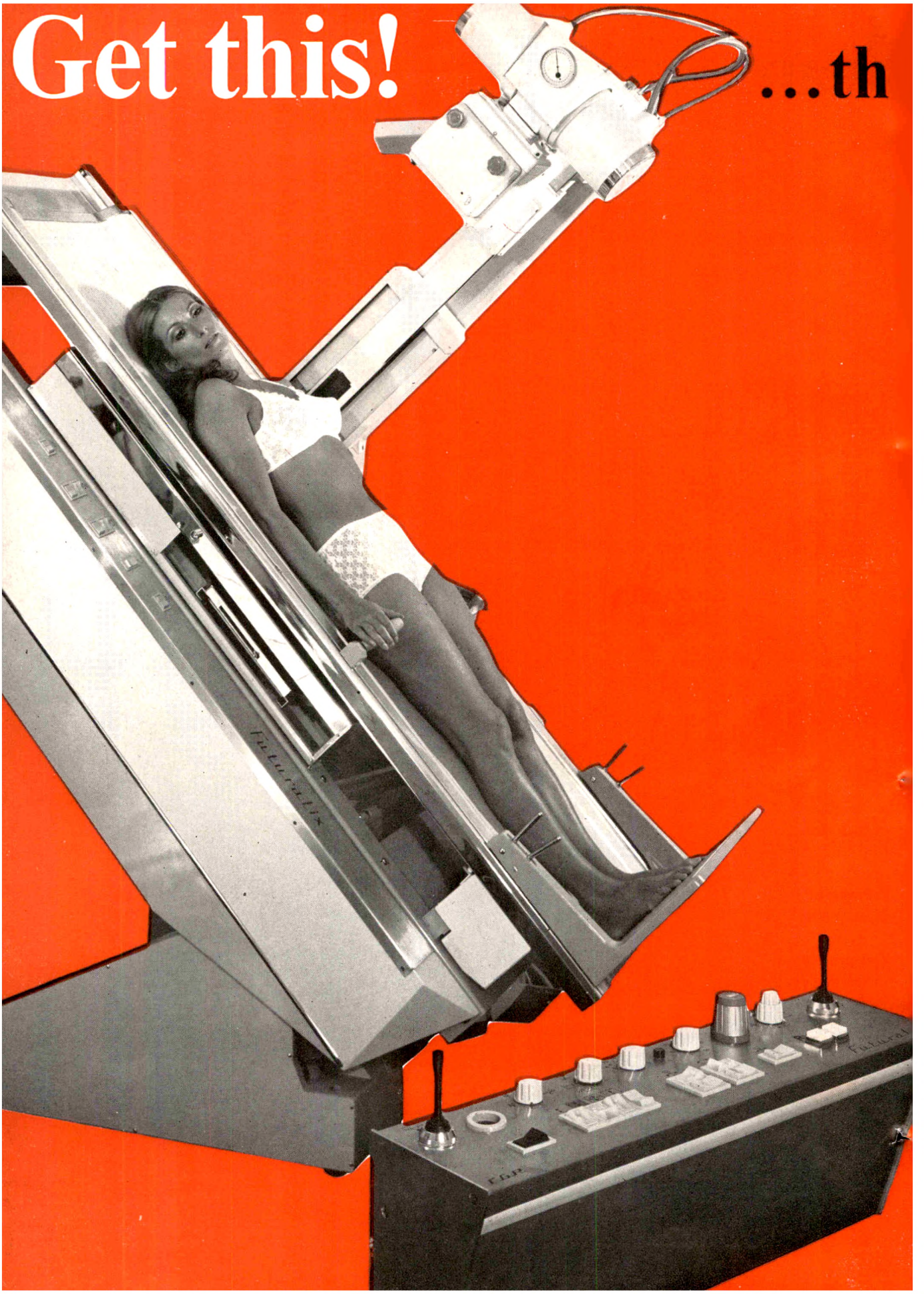
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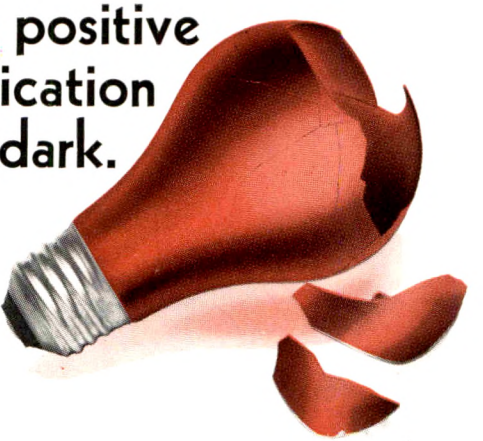
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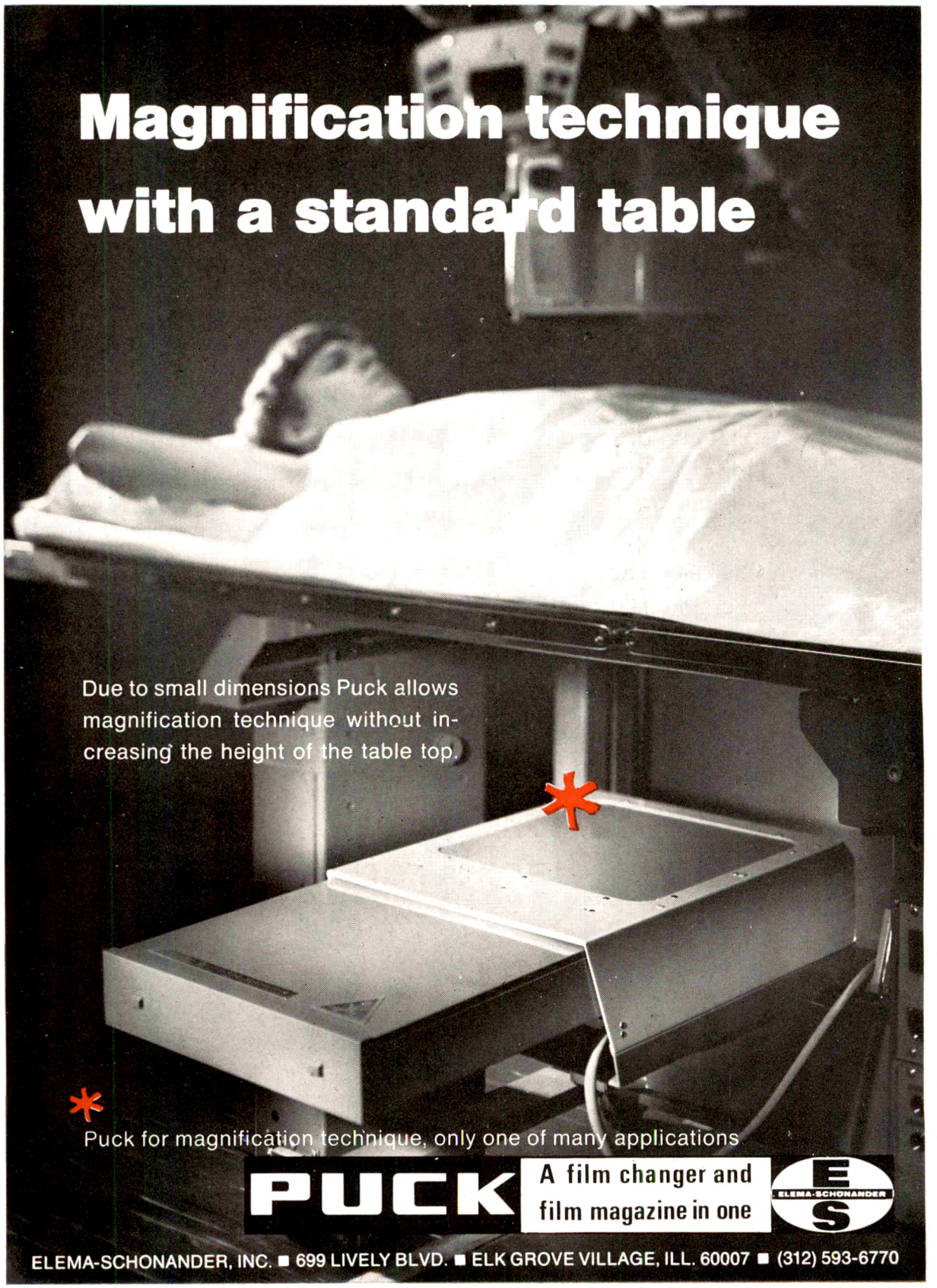
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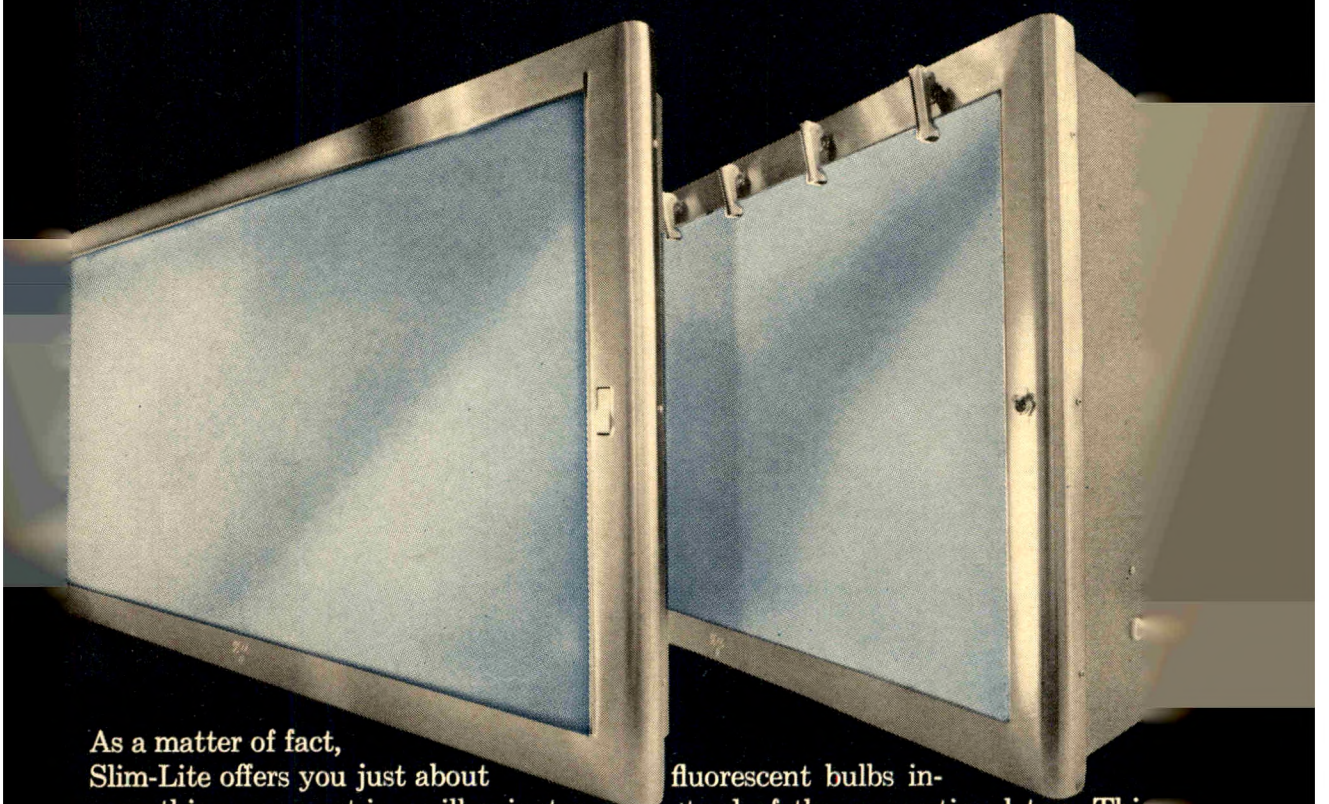
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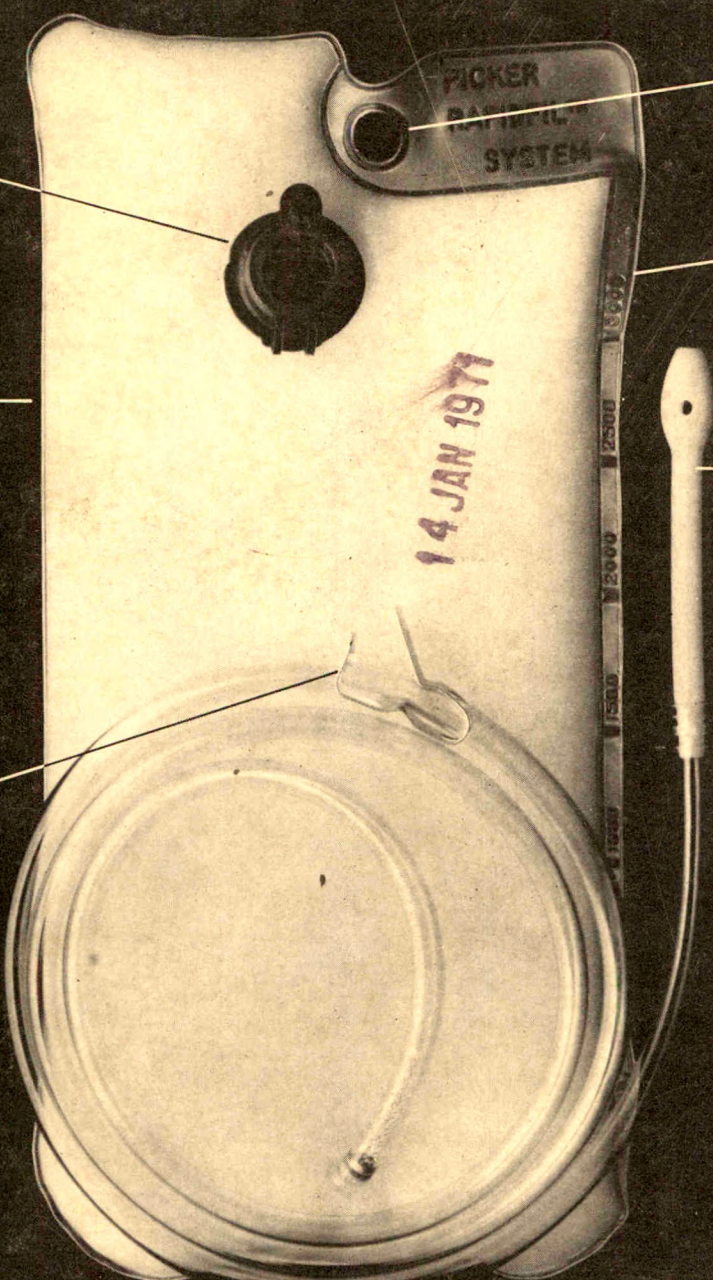
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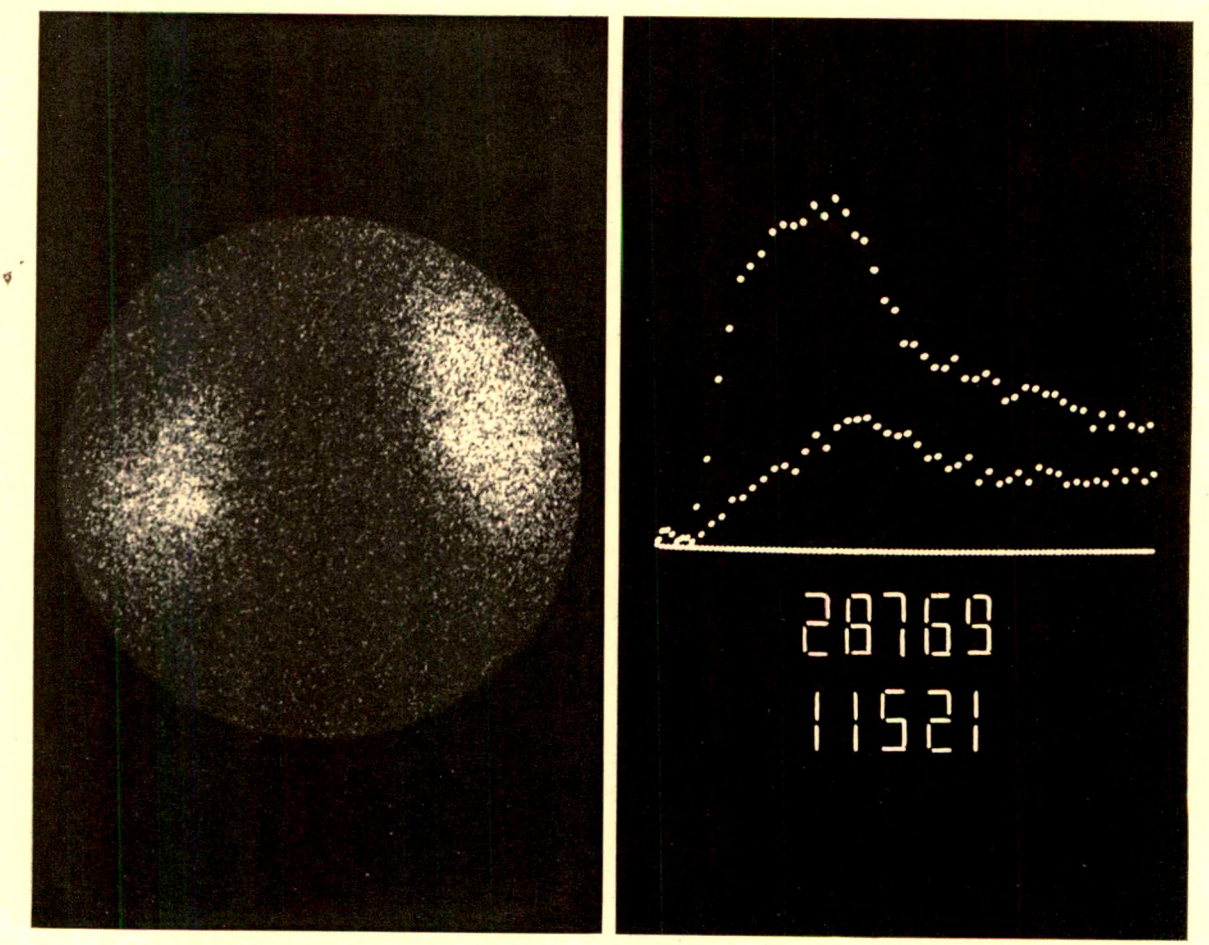
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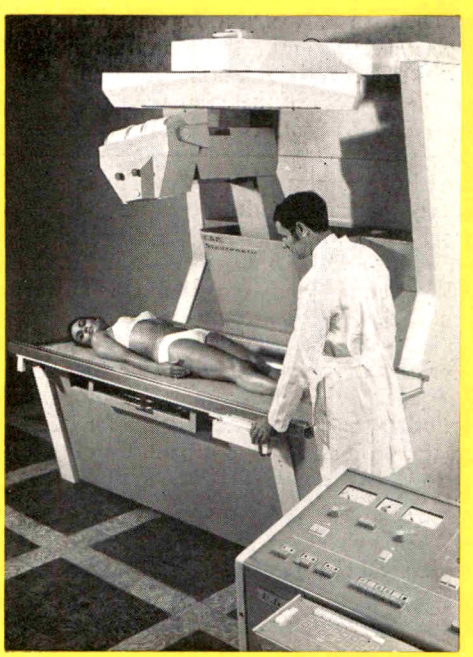
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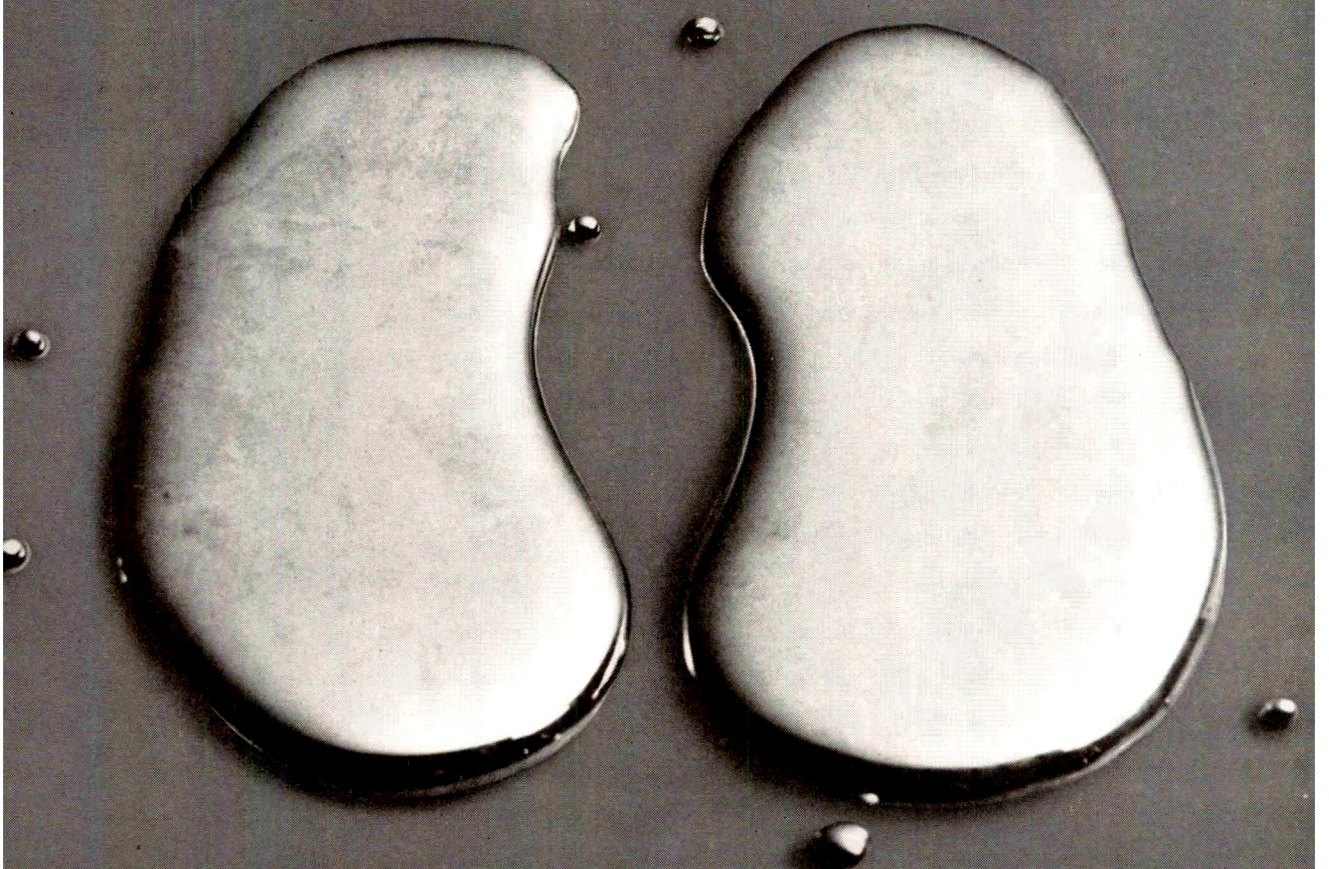
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TECHNETOPE II (Technetium 99m) Sterile Generator provides a means of obtaining a sterile, non-pyrogenic supply of technetium 99m as sodium pertechnetate.

Warnings: The contents of the syringes in the Renotec Kit and the Tesuloid Kit should **not** be injected directly into a patient.

Usage in pregnancy—These agents should not be administered to women who are pregnant or who may become pregnant and during lactation unless the indications are exceptional and the need for the agent outweighs the possible potential risk from the radiation exposure involved.

Since sodium pertechnetate ^{99m}Tc may be taken up by the fetus and excreted in human milk, administration of the preparation during pregnancy and lactation is not recommended.

Formula feedings should be substituted for breast feedings if these agents must be administered to the mother during lactation.

^{99m}Tc-DTPA, ^{99m}Tc-S colloid, and sodium pertechnetate ^{99m}Tc should not be administered to persons less than 18 years of age unless the expected benefit outweighs the hazards. It should be noted that although radiopharmaceuticals are not generally used in individuals under 18, procedures using ^{99m}Tc-DTPA or ^{99m}Tc-S colloid are occasionally necessary in such patients. The low internal radiation dosage of ^{99m}Tc-DTPA makes it a very satisfactory agent when scans of the kidney, brain, or blood vessels are necessary in young patients. The low internal radiation dosage of ^{99m}Tc-S colloid makes it a very satisfactory agent when liver or spleen scans are necessary in young patients.

Radiopharmaceuticals, produced by nuclear reactor or cyclotron, should be used only by physicians who are qualified by specific training in the safe use and safe handling of radioisotopes and whose experience and training have been approved by the appropriate federal or state agency authorized to license the use of radioisotopes.

When obtaining elutions from Technetope II (Technetium 99m) Sterile Generator, proper radiation safety precautions should be maintained at all times. The column containing ^{99}Mo need not be removed from the lead shield at any time. There is a high radiation field surrounding an unshielded column. Solutions of sodium pertechnetate ^{99m}Tc withdrawn from the generator should always be adequately shielded. The early elutions from the generator are highly radioactive. **Important:** Since material obtained from the generator may be intended for intravenous administration, aseptic technique must be strictly observed in all handling. The stoppers of the eluent bottle, of the elution tube, and of the collecting vial, as well as both rubber closures in the generator column, should be swabbed with a suitable germicide before each entry. All entries into the generator column must be made aseptically with sterile needles. Only the eluent provided should be used to elute the generator. Use a fresh milking tube and collecting vial for each elution; sufficient equipment is provided for this purpose. All equipment used to collect or administer sodium pertechnetate ^{99m}Tc must be sterile. Do not administer material eluted from the generator if there is any evidence of foreign matter.

NOTE: The Renotec Kit and the Tesuloid Kit are not radioactive. However, after the eluted ^{99m}Tc is added, adequate shielding of the resulting preparation should be maintained.

Precautions: When using radioactive material, care should be taken to insure minimum radiation exposure to the patient (*i.e.*, by using the smallest dose of radioactivity consistent with safety and validity of data) as well as to all personnel directly or indirectly involved with the patient. Before a test is repeated in the same patient, the need should be carefully evaluated; this is especially true in younger patients.

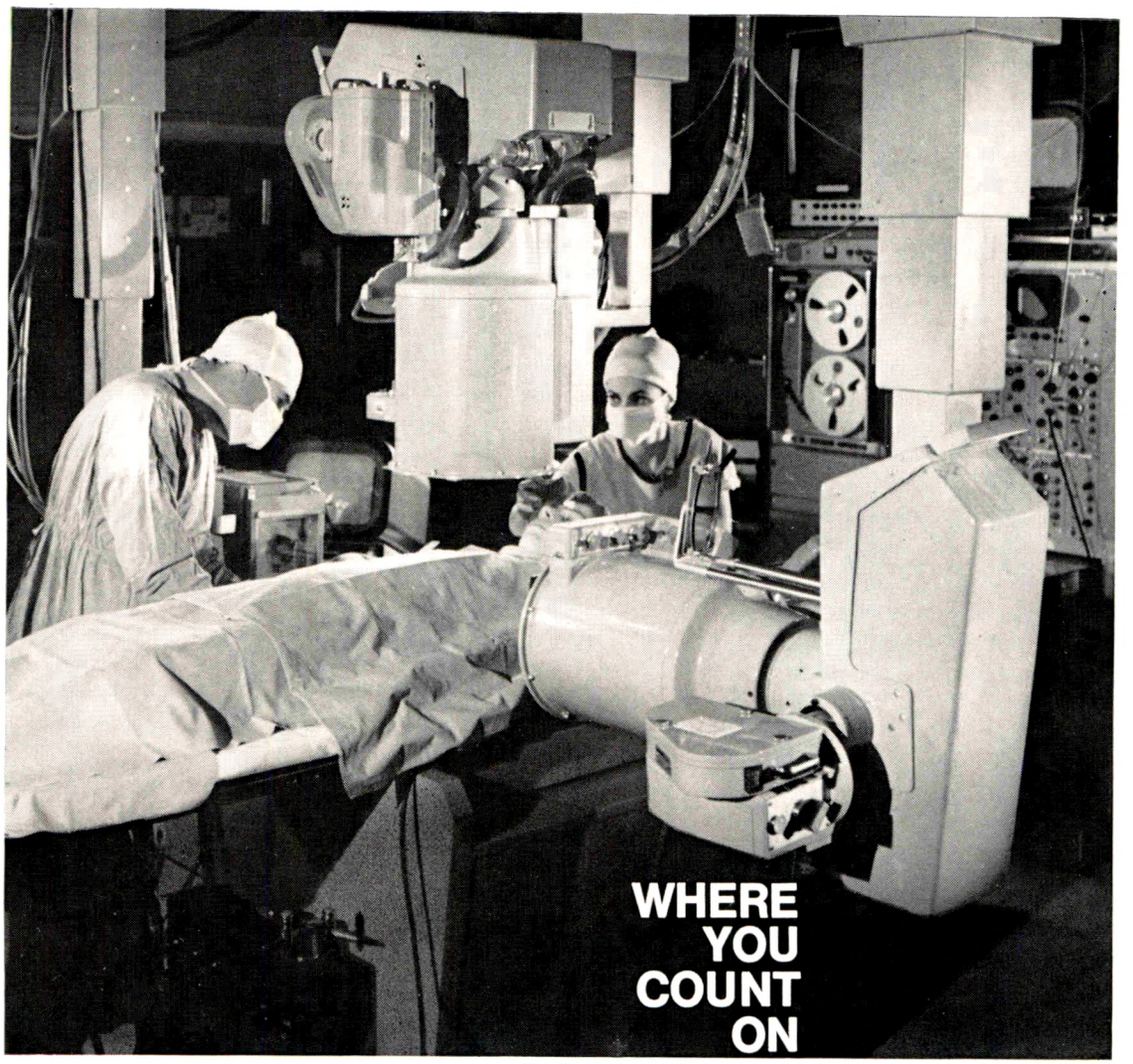
Each elution from Technetope II (Technetium 99m) Sterile Generator should be assayed before use for ^{99m}Tc activity and for the possible presence of ^{99}Mo. Material containing more than 5 microcuries of ^{99}Mo per dose of ^{99m}Tc pertechnetate exceeds Atomic Energy Commission limits and should not be administered. Poor gastrointestinal absorption of an oral dose of pertechnetate and resultant low blood radioactivity levels have been observed in the postprandial state, in seriously ill patients, and in a small number of normal, fasting individuals. Since pertechnetate is concentrated by the gastric mucosa and the salivary glands, secretions of the digestive tract are radioactive and may cause artifacts on the cranial scan. Therefore, all possible care should be taken to avoid extracranial contamination, not only for the protection of patients and of hospital personnel but also to avoid obtaining a falsely positive scan due to extracranial radiation. Any condition which alters the blood-brain barrier or the normal cranial vasculature may cause abnormal areas of increased radioactivity. The brain scan with sodium pertechnetate ^{99m}Tc is therefore likely to be abnormal in patients with scalp contusions or acute head injuries. Following a craniotomy, uptake of radioactivity is increased throughout the operative field, usually for only a few weeks but in some instances for prolonged periods. Since cerebral radiographic techniques temporarily affect the blood-brain barrier, brain scanning with sodium pertechnetate ^{99m}Tc should precede cerebral angiography when possible, or should be postponed for several days thereafter. A negative brain scan does not rule out the possibility of a lesion and should therefore never be considered diagnostically conclusive. Because the normal vascular structures are more apparent on a ^{99m}Tc pertechnetate scan than on a radiochlormerodrin scan, and because the choroid plexus may be visible, it is particularly important to recognize the appearance of a normal brain scan when ^{99m}Tc pertechnetate is used, in order to avoid incorrect interpretation.

NOTE: The Renotec Kit and the Tesuloid Kit were designed for use with the sodium pertechnetate eluate obtained from a Technetope II Sterile Generator. It is recommended that only Technetope II be used as the source of sodium pertechnetate with the Renotec Kit and the Tesuloid Kit unless the user has demonstrated that other sources of ^{99m}Tc are consistently compatible and meet the standards of Technetope II.

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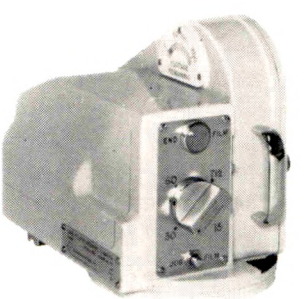


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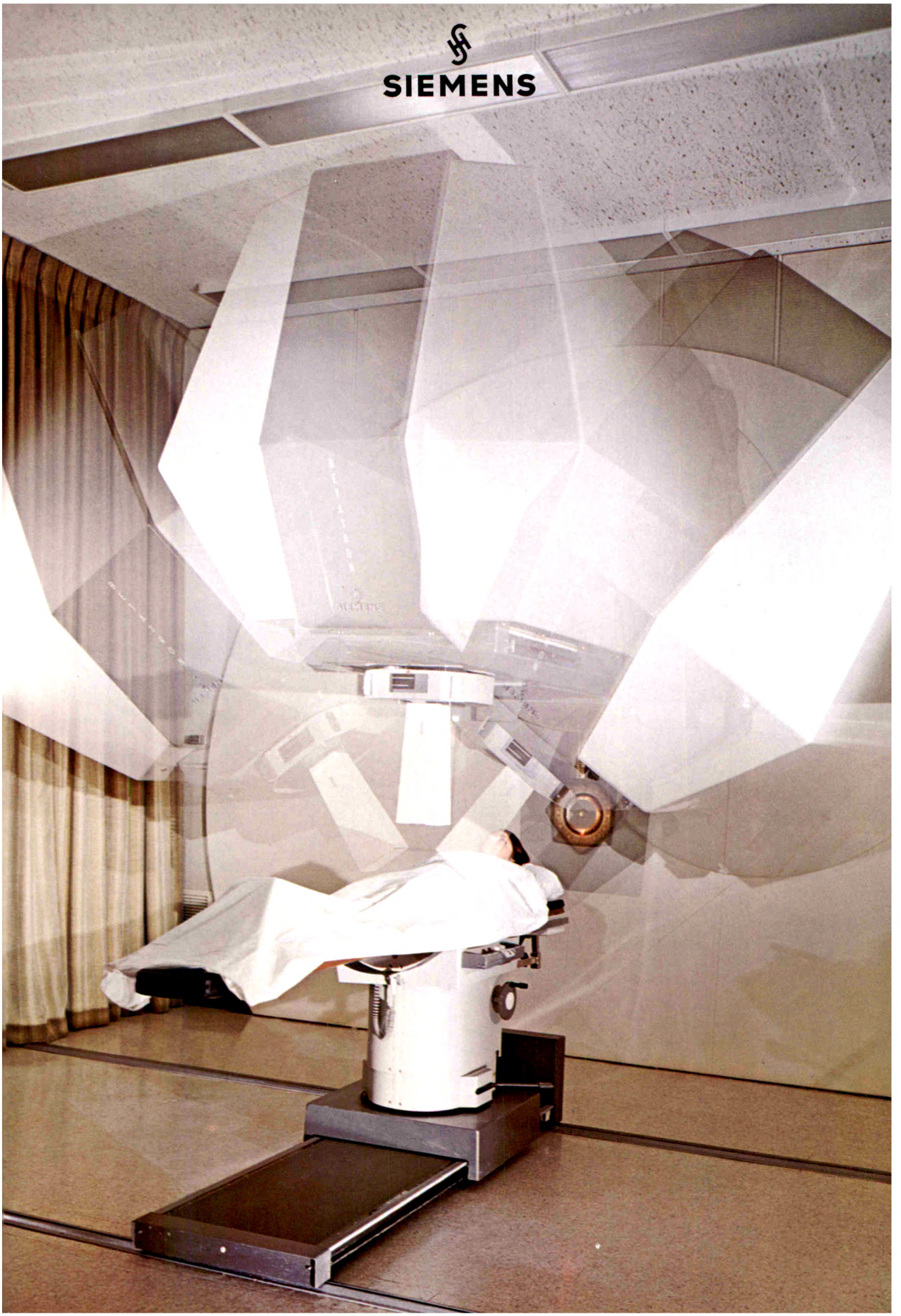
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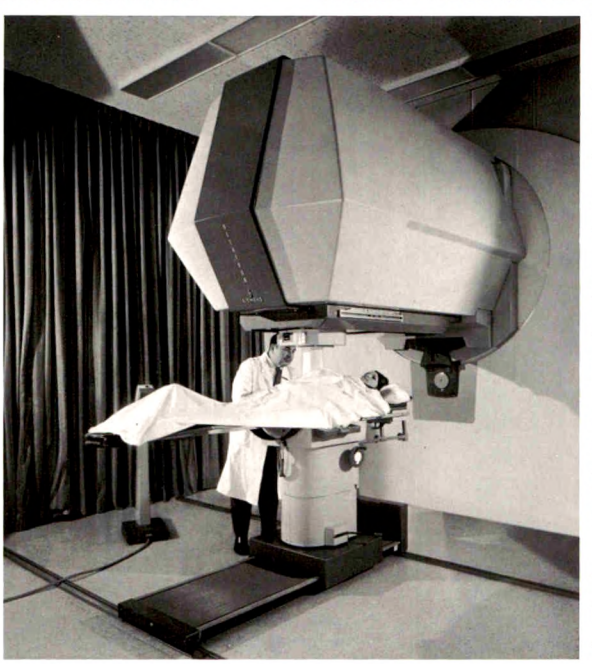
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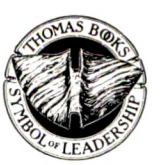
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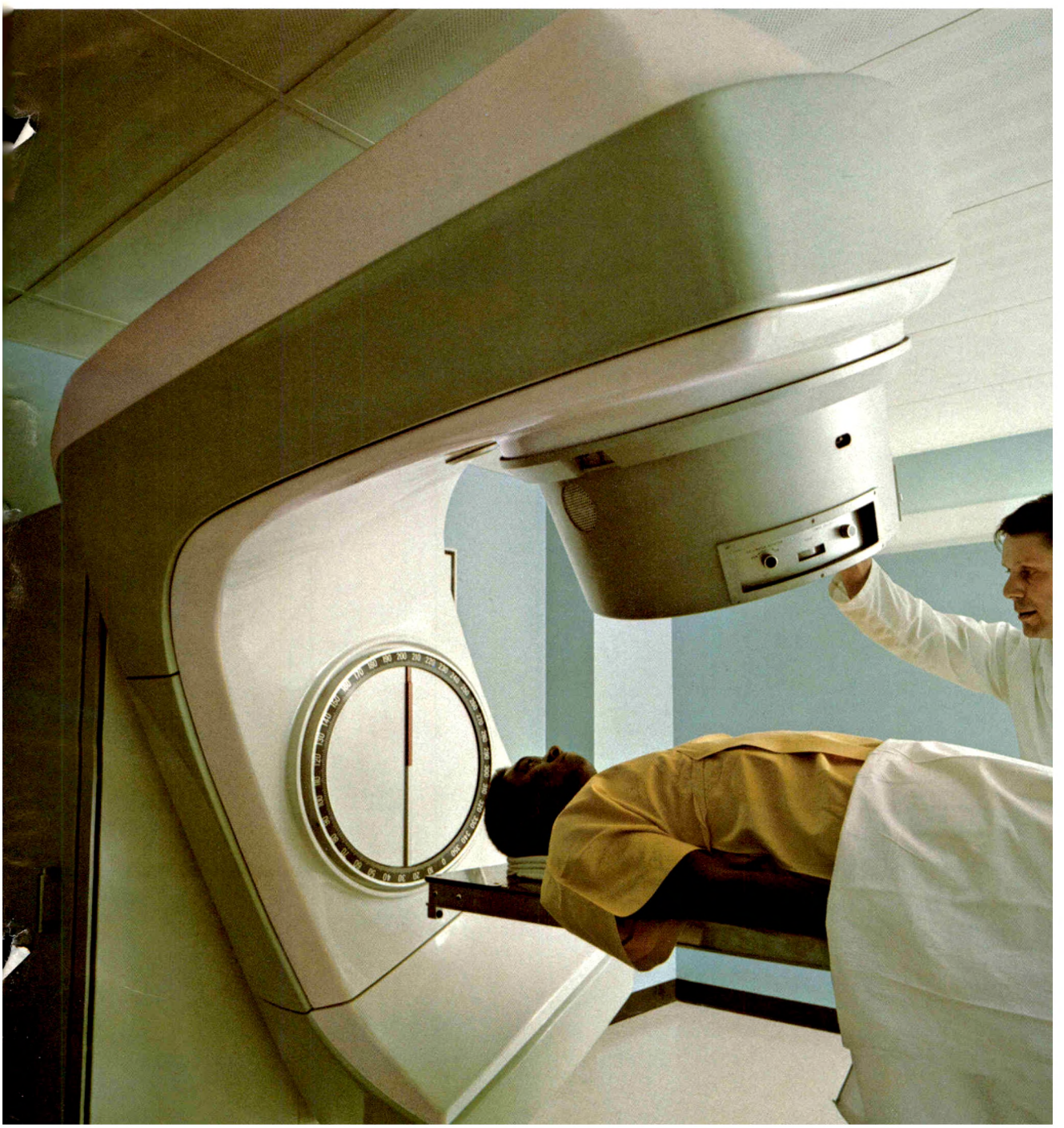
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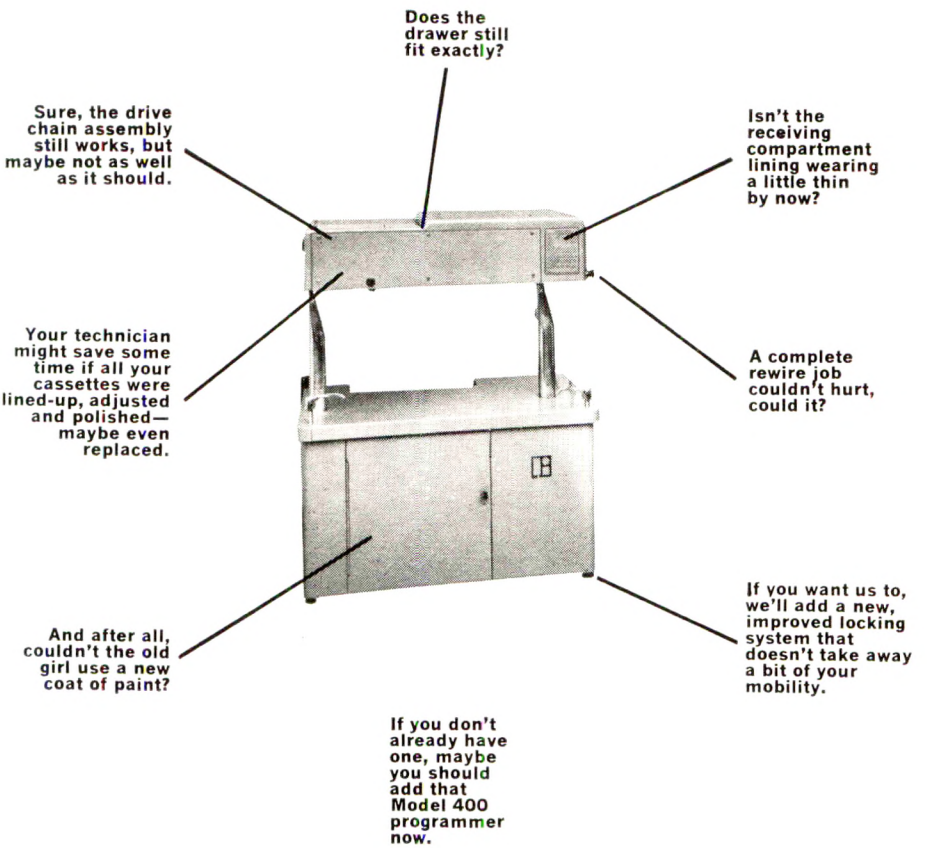
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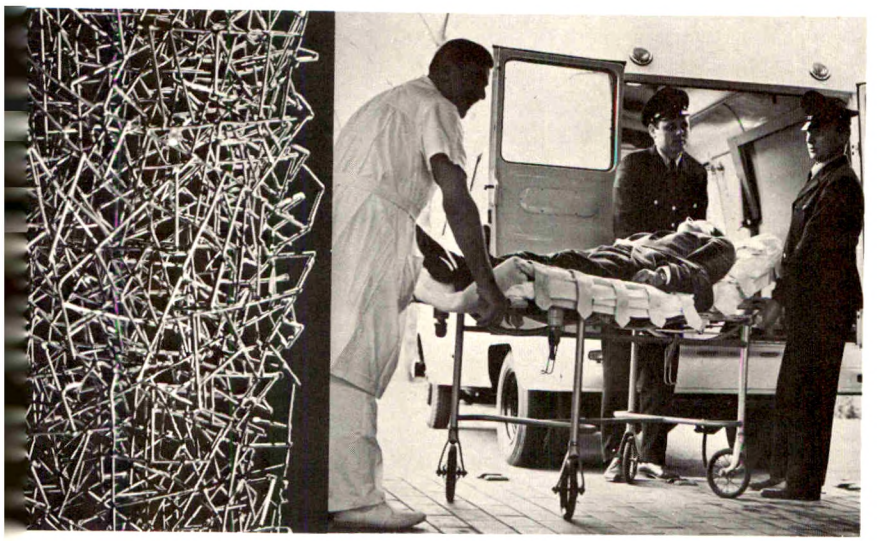


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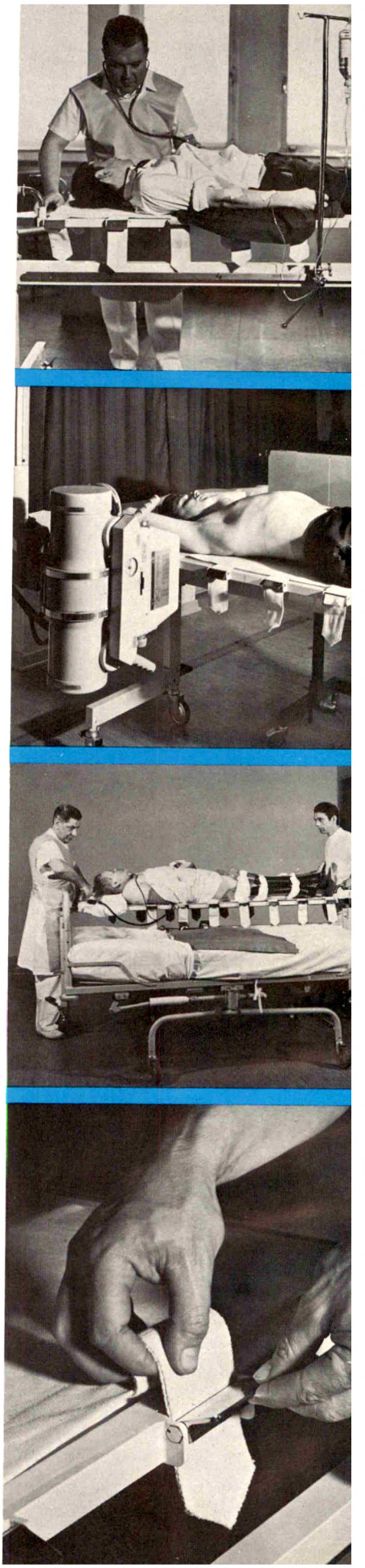
M-10

PHILIPS®

PHILIPS MEDICAL SYSTEMS, INC.
A NORTH AMERICAN PHILIPS COMPANY

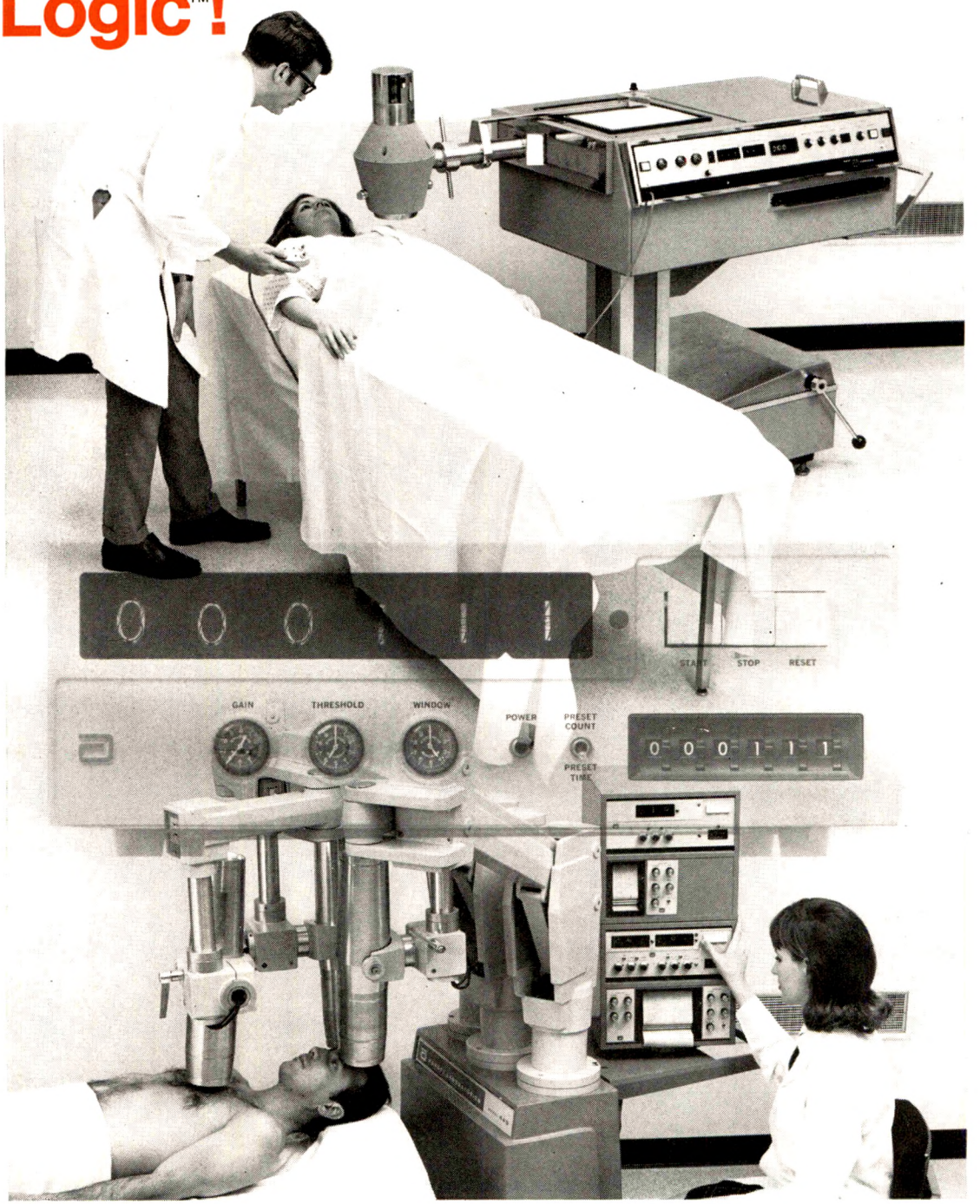
100 East 42nd Street, New York, N. Y. 10017

Outside U.S.A.: N. V. Philips • Eindhoven, The Netherlands



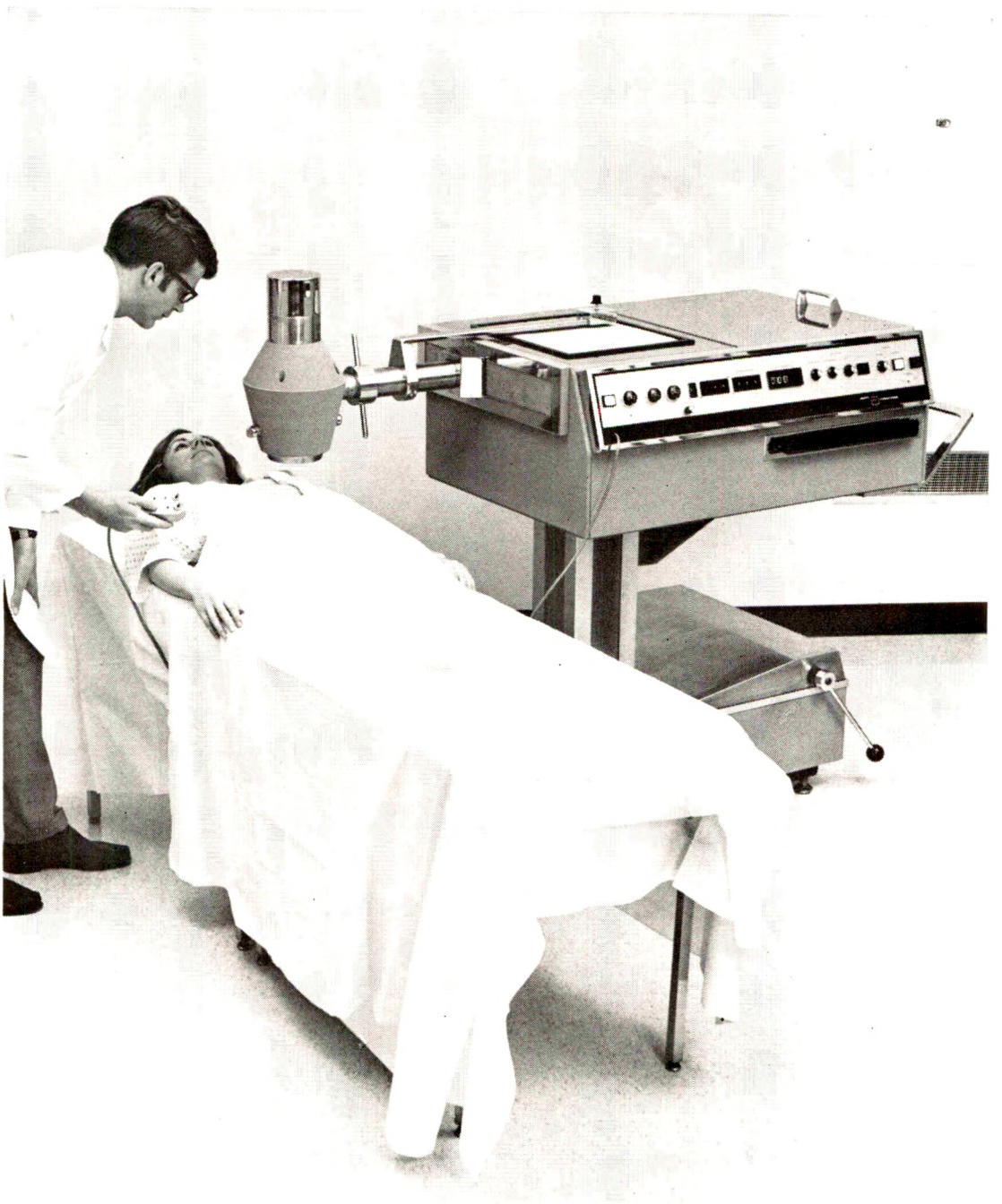
Graphic™! Dynamic™! Logic™!

To test, to "see" and to learn what goes on inside the patient!



Announcing

Graphic...



...a new concept in scanners!

Speed!
Portability!
Simplicity of Operation!

Scan speed ranges to 1,000 cm./min.

. . . variable from 10 to 1,000 cm./minute with appropriate fixed index level. 1,000 cm. minute makes it the fastest scanner available. Portal to portal patient time may be less with some studies than with camera devices.

Portable

. . . system is readily portable and can be easily moved on its 5″ casters. Will fit through any standard door opening. It is the only scanner that can easily be taken right to the patient in his hospital bed.

Simple to operate

. . . technician can master scanner operation within 30 minutes. Remote control for detector positioning. Detector angle may be adjusted 360°.

Scan area 17″ either way

. . . 17″ x 14″ in either direction. Allows for easier patient set-up to scan large lung fields, liver, spleen, etc. Can scan 17″ laterally or horizontally (important in obese patients). Uses standard 14″ x 17″ x-ray film.

Convertible

. . . the Graphic is available with either a 3″ or a 5″ detector head. Can be converted easily in the hospital from a 3″ to a 5″ scanner.

Versatile

. . . five levels of contrast enhancement including digital mode and six levels of background erase.

Integrated circuitry

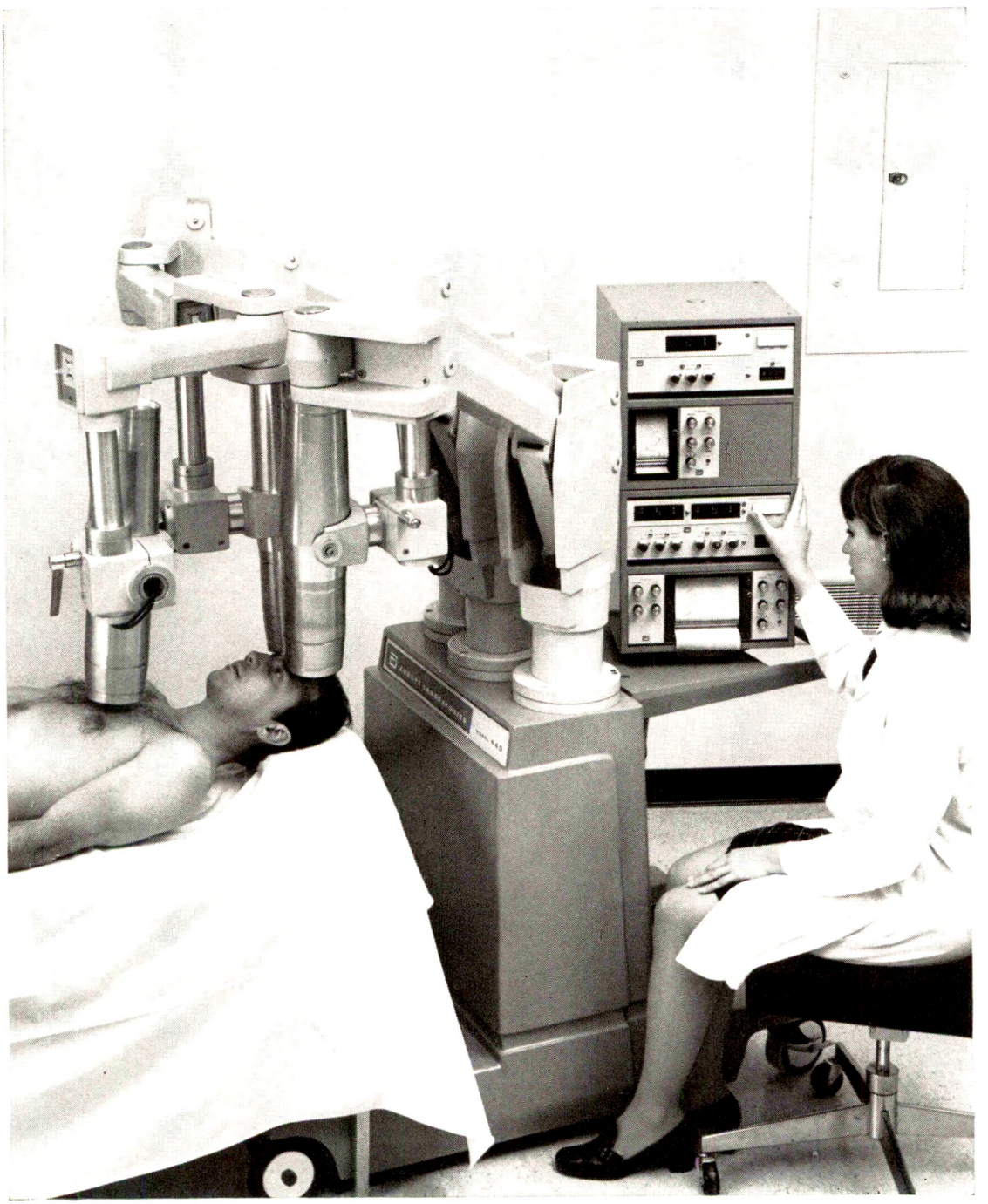
. . . allows fast, reliable photoscans in the shortest possible time with less instrument down time.

Ask about Abbott's unique service concept!

TM—Trademark.

Announcing

Dynamic...



...to study the life systems!

Four chart speeds

. . . 0.1 millimeter/second, 1.0 millimeter/second, 10 millimeters/second, and 20 millimeters/second which are keyed to the circulation times relating to dynamic function studies of the important organs of the body. Included are the brain, lungs, heart, and kidneys.

Digital

. . . the Dynamic system is entirely digital in operation.

"Foldover" capability

. . . this unique feature of the strip chart recorder assures you that no data will be lost. A Dynamic exclusive.

Heated stylus

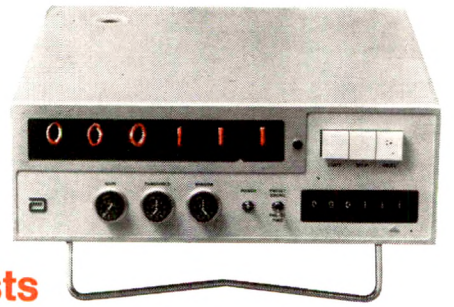
. . . in each of the strip chart recorders eliminates the messy chore of ink changing. This prevents blurred information as well as smudged fingers.

Choice

. . . of one, two, and three detector systems. 1.5 x 1.5 inch sodium iodide detectors mounted on electrically operated arms. This modular concept allows you to add on as your needs expand.

Logic...

...for in vitro and in vivo tests!



Radioisotope tests

. . . including T-3, T-4, thyroid uptake*, hepatic uptake*, plasma volume, fecal Rose Bengal excretion, iron binding, fat absorption, and placenta localization*.

Speed of electronics

. . . count and display in excess of 15,000,000 counts per minute.

Solid state integrated circuitry

. . . assures higher reliability; less down time.

Simple to operate

. . . minimum of controls with Direct Ratio Readout in %.

Choice of 3 models

. . . 101 and 111 have spectrometer and well in one instrument. The 121 has an external well.

Fast service

. . . with easy-to-use service manual; replacement boards in 24 hours. There's no waiting for servicemen.

Modular concept

. . . with built-in versatility. Protect your investment by adding components as the need arises.

*May be done by adding medical stand, external probe (shield and collimator).

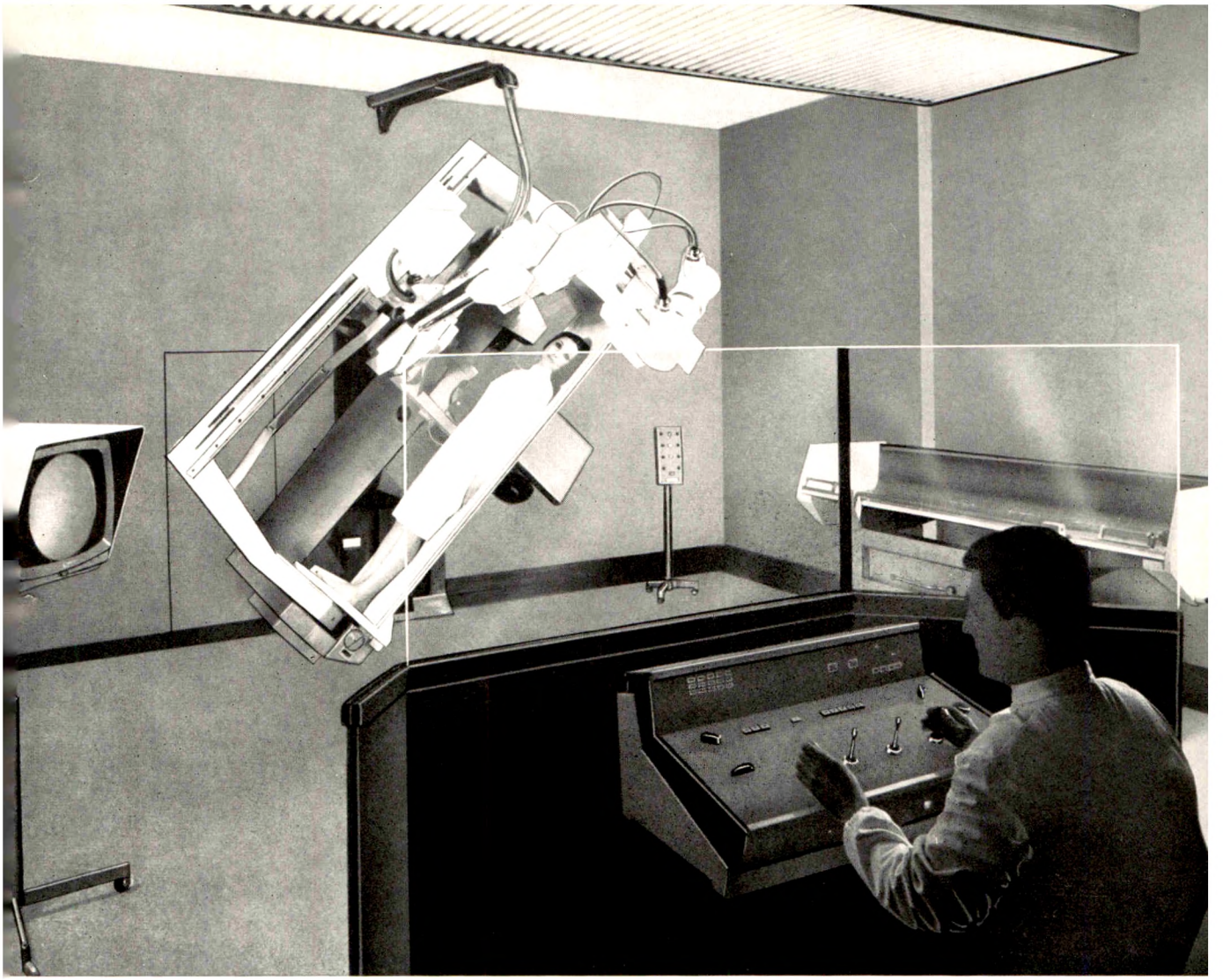
The Full Line Nuclear Medical Instrument Company

ABBOTT LABORATORIES North Chicago, Illinois 60064

Nuclear Instruments You Can Count On

Vertretung für Europa: Labor-Service GmbH, Abt. Radiopharmazeutika, 6236 Eschborn/Ts, Germany, Postfach 1245

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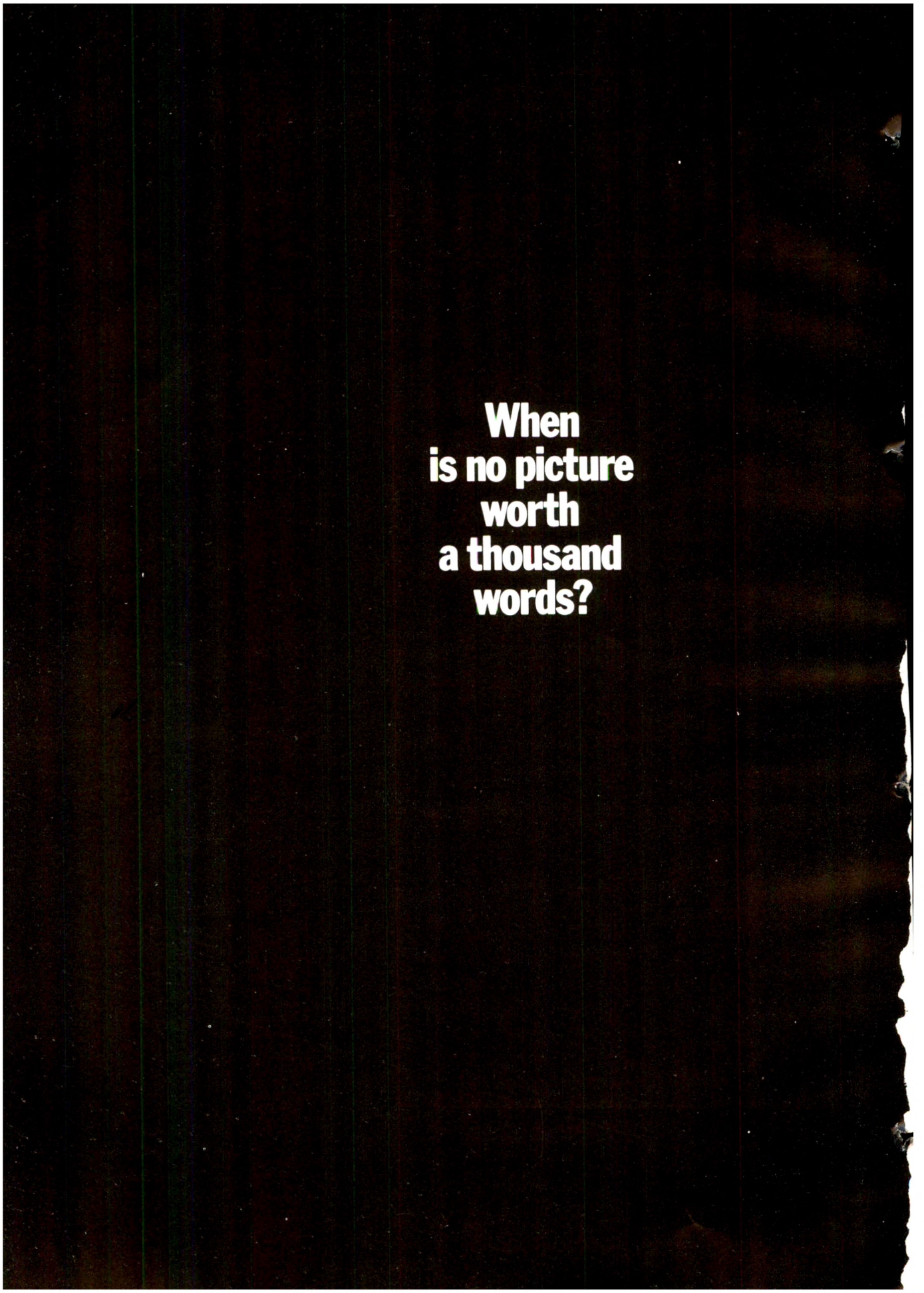
DIRIGON

Universal remote controlled diagnostic table for television screening, radiography, tomography, with the patient in any position.

GENERAY, Generale Radiologica S.p.A.
Via Monte Albenza 9
20052 MONZA, Italy.

Generay

Distributors for the United States:
Keleket/C.G.R. Corp.
1603 Trapelo Road
WALTHAM, Mass. 02154


When
is no picture
worth
a thousand
words?

What does it mean if you *don't* achieve gallbladder visualization after a repeat study with TELEPAQUE? Just *two words* sum it up—two very important words: NONFUNCTIONING GALLBLADDER—a condition ordinarily indicative of disease.* Thus the absence of gallbladder visualization is of extreme diagnostic significance. Though other radiopaque agents may opacify the gallbladder regardless of its functional status, only with TELEPAQUE is it possible to detect those patients with cholecystitis and loss of function.

But TELEPAQUE does more than produce "nonpictures." If the gallbladder is normal or is partially functioning but contains stones, these conditions are precisely defined.

So, with TELEPAQUE, you get "the complete picture"—whether you get a picture or not. Because of its diagnostic dependability and excellent tolerance, it is the preferred oral contrast agent for cholecystography. And there's still another advantage with TELEPAQUE: presence of an intestinal residue assures you that TELEPAQUE was ingested!

*Causes other than disease have occasionally been reported for nonvisualizing gallbladders.

Contraindications: Contraindicated in advanced hepatorenal disease or severe impairment of renal function, severe gastrointestinal disorders that prevent absorption, and in some patients sensitive to iodine compounds.

Precautions: Severe, advanced liver disease may interfere with metabolism of Telepaque, thus increasing the excretory load on the kidneys. Although renal difficulty has rarely been attributed to Telepaque, renal function should be assessed before cholecystography in severe, advanced liver disease, and renal output and hepatic function should be observed for a few days after the procedure. Patients with preexisting renal disease should not receive high doses of cholecystographic media. Possible renal irritation in susceptible individuals could result in reflex vascular spasm with partial or complete renal shutdown. Caution is advised in patients with coronary disorders, especially those with recent symptoms of coronary artery disease. Blood pressure should be observed after administration of cholecystographic media to these patients. Elevation of protein-bound iodine for several months and false positive urine albumin tests (for three days) may occur after ingestion of iodine-containing cholecystographic media.

Adverse Reactions: Most reactions are mild and transitory; serious side effects are very rare. Gastrointestinal effects (diarrhea, cramps, nausea, vomiting) are the most common. Usually the diarrhea consists merely of a few loose stools, although rarely it may be severe. A mild stinging sensation during urination may occur, and rarely, skin rash, urticaria, pruritus, and flushing. One case of thrombocytopenia has been reported in a patient with a history of conjunctival hemorrhages. Subjective complaints have been: dryness of throat, burning on swallowing, heartburn, sore throat, dizziness, and headache.

Usual Dosage: 3 Gm. (6 tablets) at night after a strictly fat-free supper.

Supplied: Tablets of 500 mg., envelopes of 6 tablets, boxes of 5 and 25 envelopes; also bottles of 500.

Telepaque® for precise oral cholecystography and cholangiography

brand of **iopanoic acid, USP**

MORE THAN 40,000,000 DOSES OF TELEPAQUE HAVE BEEN ADMINISTERED* WITH AN UNPARALLELED RECORD OF CLINICAL SUCCESS

*Number of doses administered since the introduction of Telepaque

Winthrop **PIONEERS IN PRODUCTS FOR RADIOLOGIC DIAGNOSIS**

WINTHROP LABORATORIES
NEW YORK, N.Y. 10016

1410M



How do you put together an x-ray department?

For some people, the currently fashionable way is piecemeal. A little bit from here. A little bit from there. A dab of this, a dash of that. The fact that such a system actually works—often quite well—is an enormous tribute to the state of our art, and to the integrity of the various equipment manufacturers (including us, thank you).

But there's still a chance that instead of winding up with an x-ray department you can get your teeth into, you'll wind up with hash. After all, not every link can be the strongest one in the chain. How many extra factors can you afford to include before the confusion begins?

So we'd like to say a kind word about the idea of buying everything from a single source: namely, Litton's Profexray Division. It makes sense to start with us, because we offer the broadest line in the industry—from 13 MeV linear accelerators to 3-phase generators to processors to heart cath electronics. And it makes sense to stay with us because then you've got only one tail to twist in case something doesn't work. Ours. Next time you put together an x-ray department, phone your Profexray representative.

And see what he can cook up for you.

PROFEXRAY
DIVISION OF LITTON INDUSTRIES
515 East Touhy Avenue, Des Plaines, Illinois 60018



This particle of difference in TRESITOPE®

makes a big difference in your in vitro thyroid function tests*



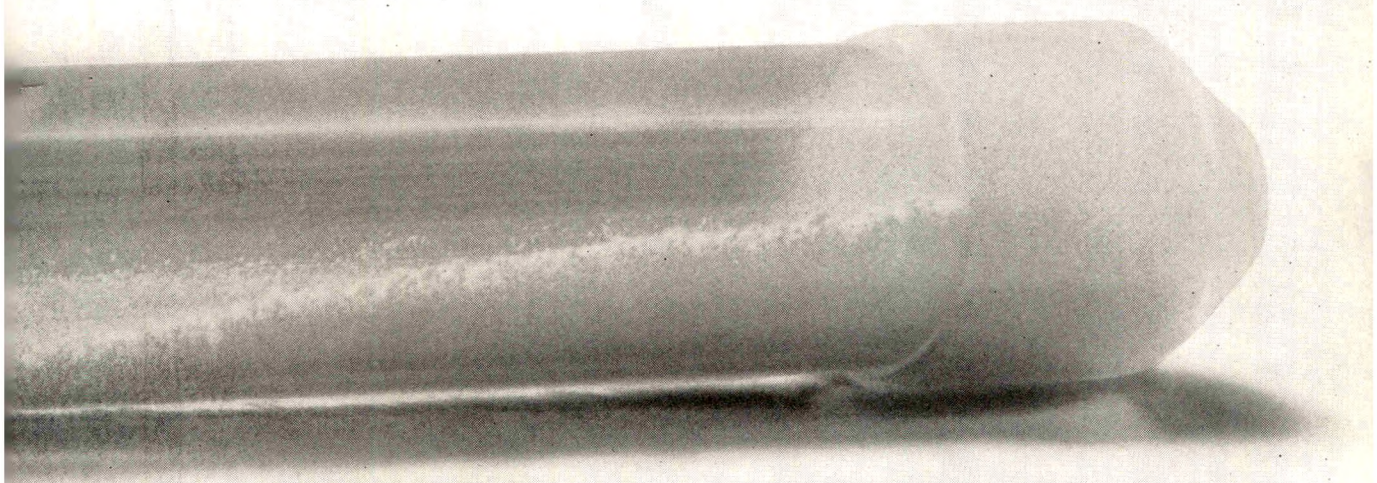
Magnification 10X

Now the resin powder is granulated for more reliable, reproducible results than ever before

The new resin particles in our Tresitope Diagnostic Kit provide a more effectual secondary binding site for the T_3 hormone.

The resin uptake powder uniformly absorbs the serum-buffer solution, facilitates simplicity of test procedures and is a key factor in yielding reliable, reproducible results.

***NOTE:** While the resin uptake test is a very useful aid in the evaluation of thyroid func-



tion, it should not be used as the sole basis for such an evaluation. In any patient, the clinical state is probably the best indication of thyroid status, and *any* laboratory test must be interpreted with caution when test results do not agree with clinical evidence.

There is a Tresitope Diagnostic Kit to meet your needs. The 12-test kit containing 10 light-resistant (amber) vials of solution for serum testing, plus 2 vials for use with reference samples, is designed to save refrigerator space. The vials of radioactive test solution are packaged separately and are the only parts requiring refrigeration. A handy styrofoam platform holds the vials. One end of the platform is modified to facilitate suction washings of the resin powder.

The Tresitope Diagnostic Kit is also available as a 105-test kit and a bulk vial kit. The 105-test kit contains 100 light-resistant (amber) vials of solution for serum testing, plus 5 vials for use with reference samples. The vials of radioactive test solution are packaged separately with these two kits and are the only parts requiring refrigeration. Included is a sufficient supply of tubes of resin powder and individual droppers for each test.

The bulk vial kit contains a 60 ml. bottle of test solution with a sufficient number of plastic tubes of resin powder to perform at least 105 tests.

IMPORTANT

Use appropriate radiation precautions in handling, identifying and discarding all radioactive material. Remember that minute amounts of radioactivity remain on components used in the test, including the styrofoam platform when it is used in performing the test, and particularly when the Tresitope Suction Method is used for a number of tests.

SQUIBB
Division of Nuclear Medicine
New Brunswick,
New Jersey 08903

Medotopes®

TRESITOPE® DIAGNOSTIC KIT

Resin Uptake Kit with Liothyronine I 125 Buffer Solution

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FUJI

Unsurpassed X-ray film. Type Rx for 90-second processing.

PYNE XRAY CORPORATION

FUJI FILM

Dealers of FUJI Kx and Rx film in all cities throughout the country.



Don't buy Nuclear Instruments—invest in them! The Abbott Modular Concept lets you do just that.

The Abbott Modular Concept is an ideal way to utilize nuclear instruments. With a minimum investment, you get the LOGIC™ 111A. This is an integrated Spectrometer and Well Counter in a single package.

As your investment pays for itself, you can add other components to your basic unit. Thus, with the Abbott Modular Concept, you get the capability you need today—with the option to add on as your needs grow. And because these units employ advanced technology, you don't have to be concerned about obsolescence. You are getting tomorrow's Nuclear Instruments today.


External Probe

When you're ready to do *in vivo* studies such as thyroid uptakes, you can add an External Probe, Collimators, and the Mechanical Medical Stand. What's more, this total system actually costs you thousands of dollars less than any competitive hardware.


Medical Stand


External Well

As your needs continue to grow, you can add an External Well to run *in vitro* studies with high energy nuclides. Only the Abbott Modular Concept offers you this flexibility and economy.

TM—Trademark

003191

The Full-Line Nuclear Medical Instrument Company

ABBOTT LABORATORIES NORTH CHICAGO, ILLINOIS 60064

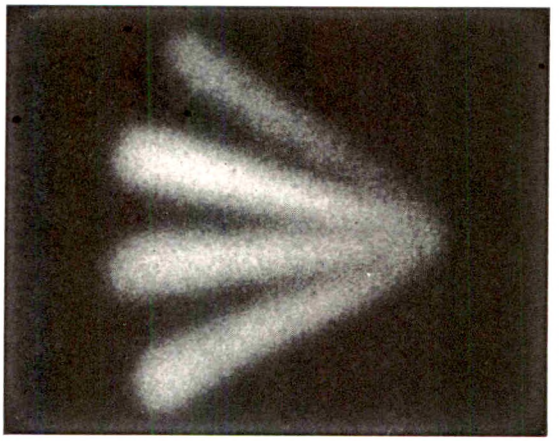
Nuclear Instruments You Can Count On

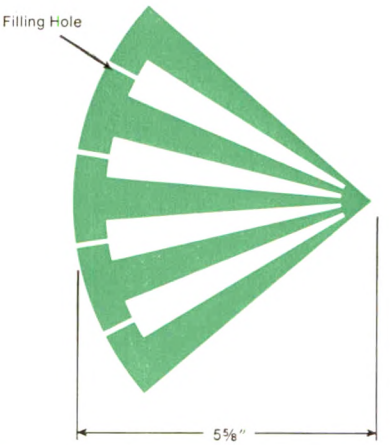
Vertretung für Europa: Labor-Service GmbH, Abt. Radiopharmazeutika, 6236 Eschborn/Ts, Germany, Postfach 1245

The Picker Dynacamera 2:

The scintillation camera with both high resolution and a large *undistorted* field of view:

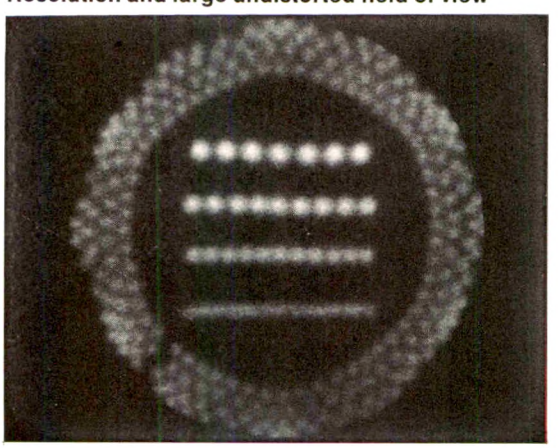
Resolution

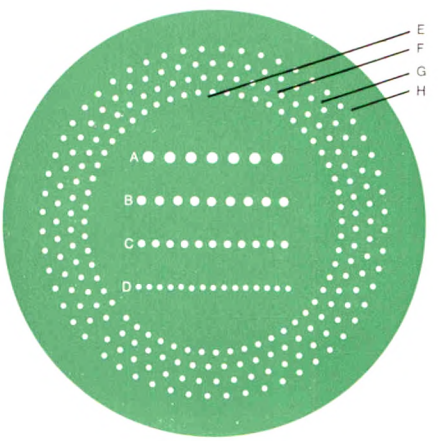




Phantom description: 3/8″ thick lucite with four 1/8″ thick radiating voids filled with activity.

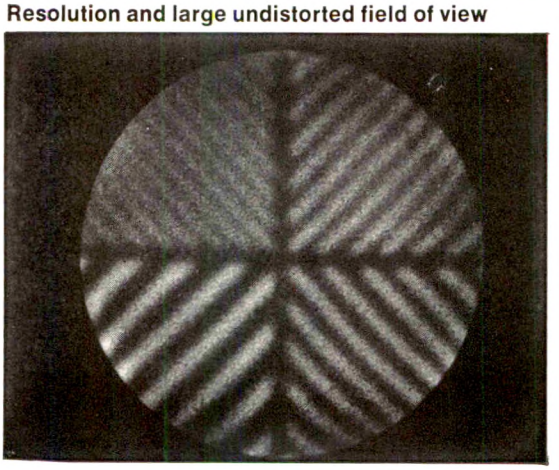
Resolution and large undistorted field of view

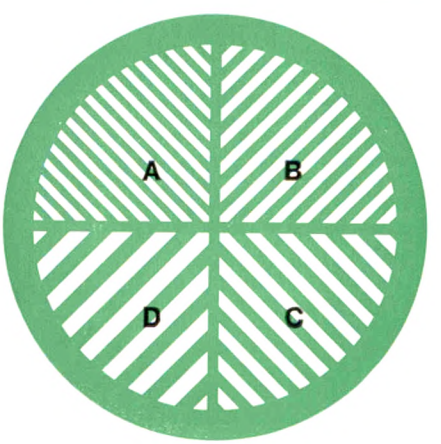




Phantom description: 1/8″ thick by 15″ dia. lead circle mounted between two circular pieces of 1/8″ thick lucite.
A. 3/8″ dia. 3/8″ space
B. 5/16″ dia., 5/16″ space
C. 1/4″ dia., 1/4″ space
D. 3/16″ dia., 3/16″ space
E. 3/16″ dia. holes with centers on 9″ dia. circle.
F. 3/16″ dia. holes with centers on 10″ dia. circle.
G. 3/16″ dia. holes with centers on 11″ dia. circle.
H. 3/16″ dia. holes with centers on 12″ dia. circle.

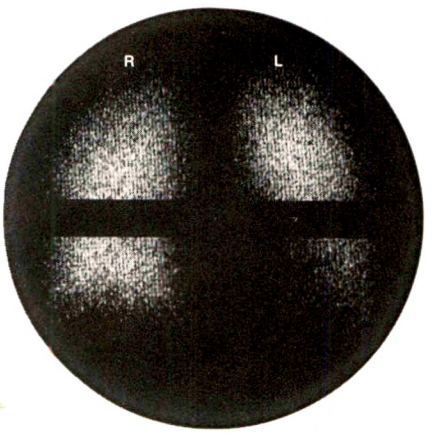
Resolution and large undistorted field of view

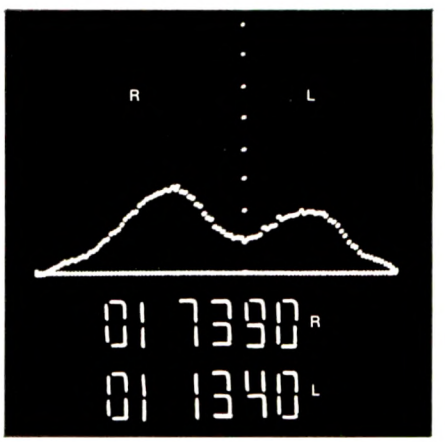




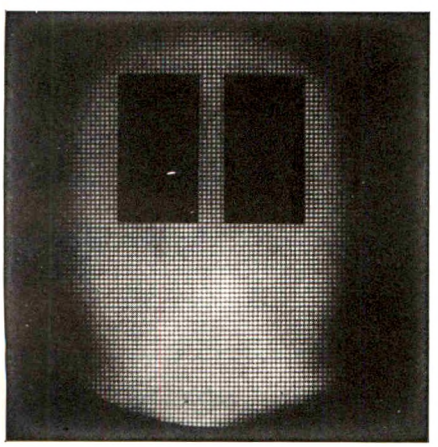
Phantom description: 1/8″ thick lead bars mounted between two circular pieces of 1/8″ thick lucite. A 14″ outside diameter, 1″ wide, lead ring surrounds the bars.
A. 1/4″ bars, 1/4″ spaces
B. 5/16″ bars, 5/16″ spaces
C. 3/8″ bars, 3/8″ spaces
D. 1/2″ bars, 1/2″ spaces

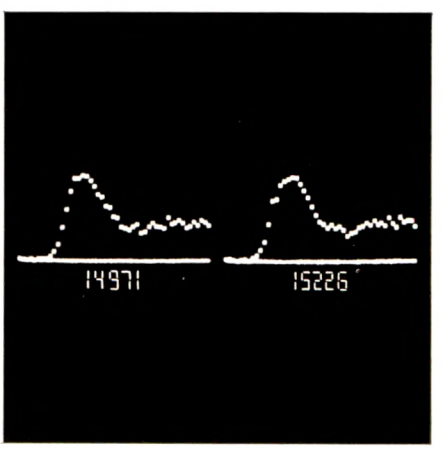
The scintillation camera with more clinically useful and proven capabilities:



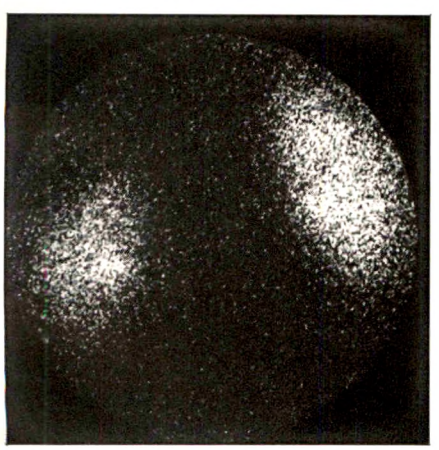


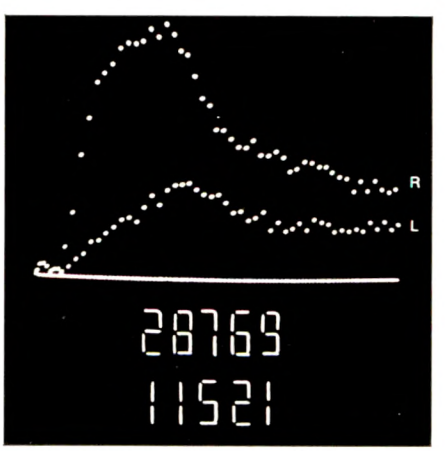
Quantification of static studies
(a built-in capability)
Dynacamera 2 is the scintillation camera that provides both Scintigrams and the total count in an organ or any portion of it.





Quantitative regions of interest
(a built-in capability)
Dynacamera 2 permits the selection of two regions of interest and simultaneously displays both count rate vs. time and total integrated counts in both regions.





Quantitative dynamic studies
(a built-in capability)
Dynacamera 2 performs quantitative dynamic function studies in selected regions without the need for modifications, accessory systems, or extra cost and produces digital histograms simultaneously for quantification of each discrete phase.

Please call your local Picker technical specialist for information about other Dynacamera 2 features or to learn about Dynacamera 3, the scintillation camera with a built-in image enhancement system. Or write Picker Medical Products Division, Dept. N, 595 Miner Road, Cleveland, Ohio 44143.

PICKER

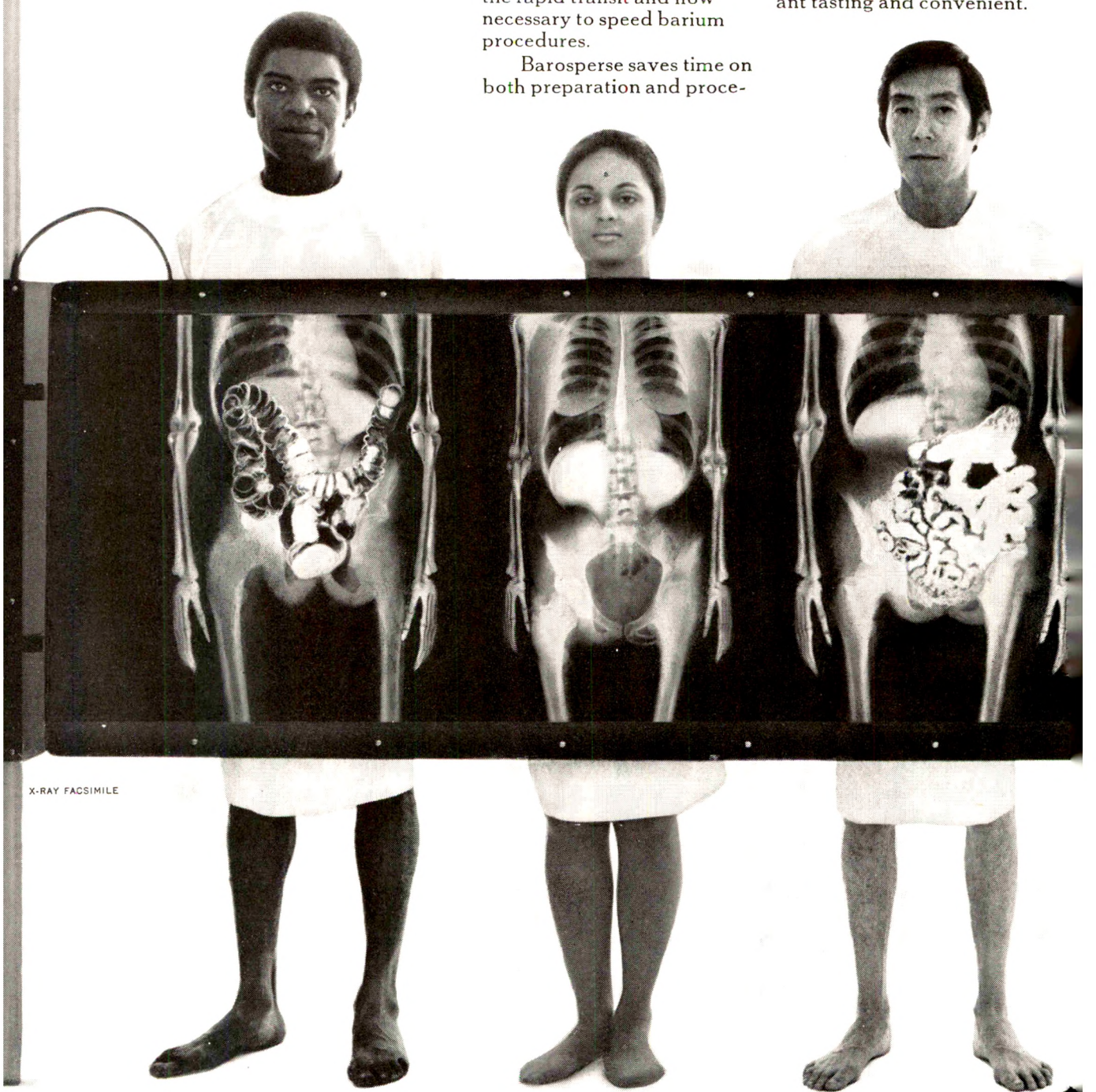
The "single source responsibility" company.

To be more exact, Barosperse has been used in millions of G.I. examinations in more than 30 countries around the world. Which makes Barosperse the most widely accepted of all barium sulfate formulations by far. And with good reason.

With Barosperse, you get films of unexcelled diagnostic quality and definition of detail. Used for upper or lower G.I. series, this formulation has the fluidity and coatability to fill in the folds and convolutions of the entire G.I. tract. The opacity to provide sharp, accurate, readable visualizations. And the rapid transit and flow necessary to speed barium procedures.

Barosperse saves time on both preparation and procedure. It mixes with water and is dispersed in 20 to 30 seconds. Barosperse requires minimum fluoroscopic time resulting in minimum radiation. Superb small bowel delineation is normally accomplished in 30 to 90 minutes.

Barosperse is both pleasant tasting and convenient.



X-RAY FACSIMILE

Barosperse® gets into more G.I. tracts in more countries

(barium sulfate formulation)

Most patients readily accept the smooth, pleasantly flavored mixture; and both single dose and bulk packaging permit greater flexibility and convenience in the lab.

So when you think of barium sulfate, think of the formulation more radiologists and gastroenterologists in more than 30 countries use. Barosperse.

Barosperse® (Barium Sulfate Formulation) is available in the following convenient packages.

Barosperse Esophageal Cream in 100-gm and 320-gm tubes.

The Barosperse Single Dose Oral Unit.

The Barosperse Disposable Barium Enema Unit.

The Barosperse Disposable Direct Air Contrast Barium Enema Unit.

Barosperse, for bulk use, in 10 lb., 25 lb., and 100 lb. drums.

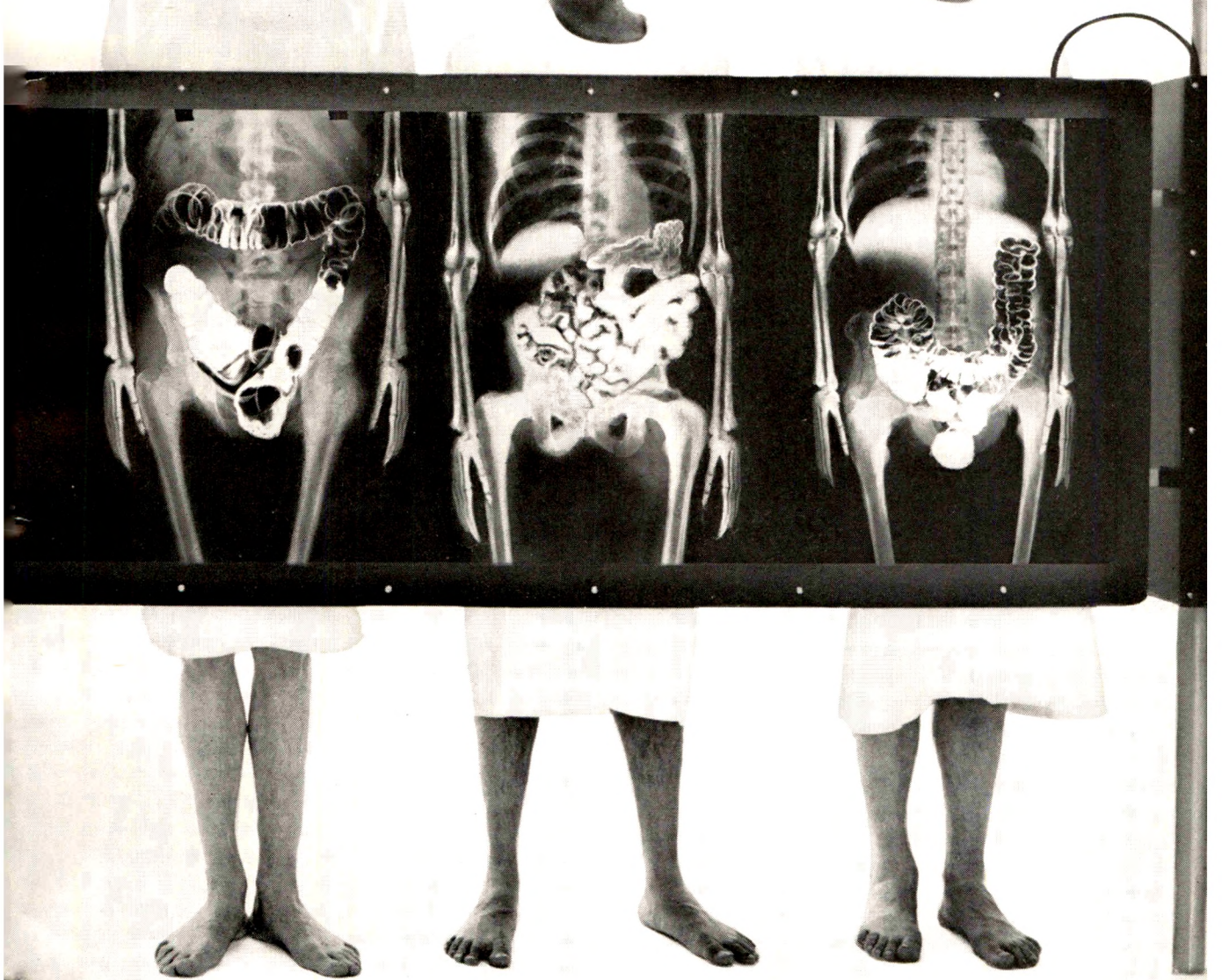
Mallinckrodt Pharmaceuticals

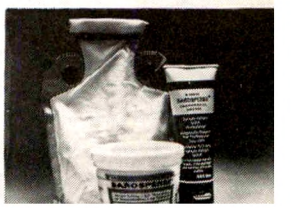
Mallinckrodt Chemical Works
St. Louis, Missouri 63160





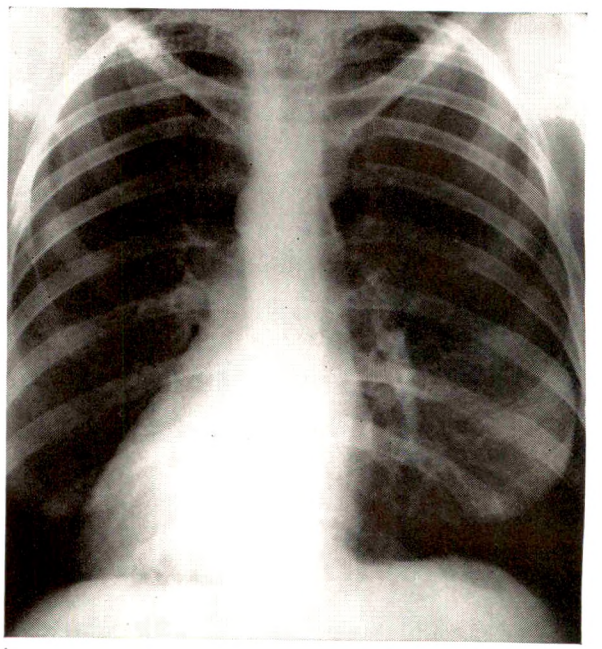




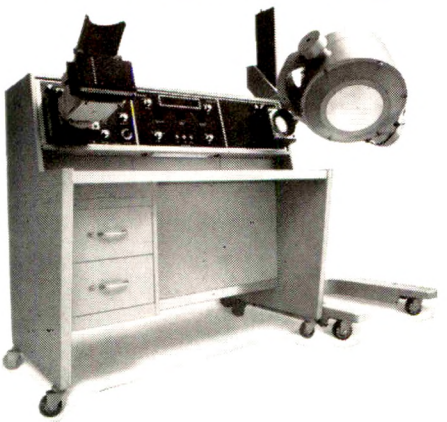


than any other contrast medium of its kind.

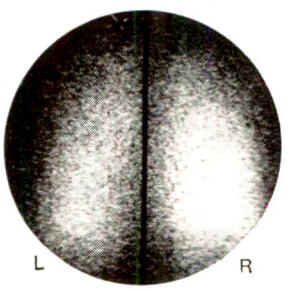
STANDARD CHEST RADIOGRAPH.
INVERTED AP VIEW.



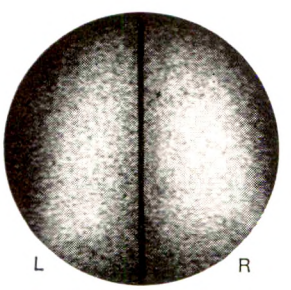
THE PHO/GAMMA SCINTILLATION CAMERA.



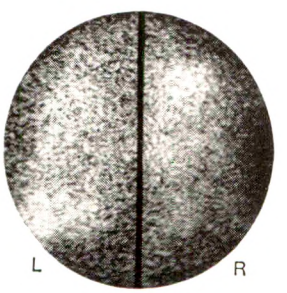
SERIAL SCINTIPHOTOS. POSTERIOR VIEW.



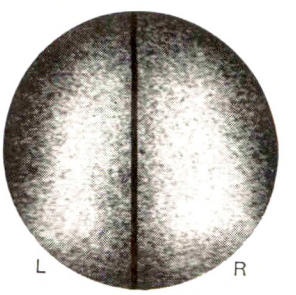
1. PERFUSION (Q)



2. LUNG VOLUME (V_2)

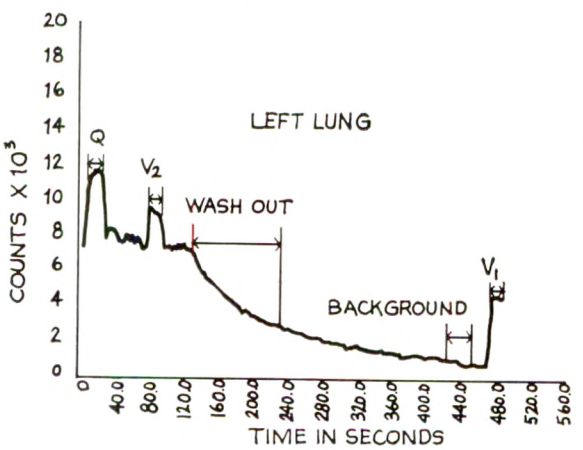


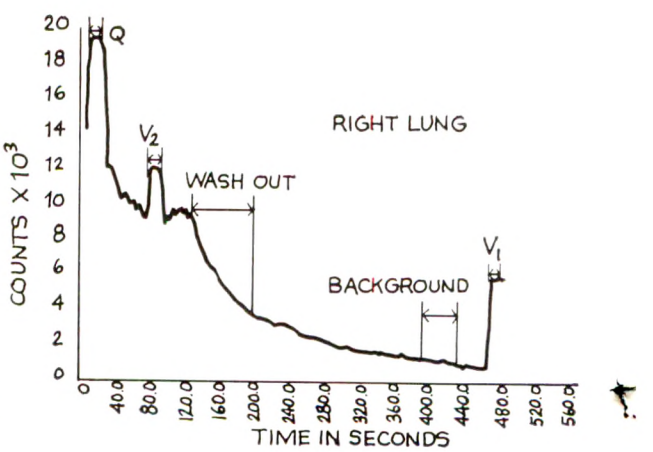
3. WASH-OUT



4. VENTILATION (V_1)

TIME-ACTIVITY HISTOGRAMS.





The Pulmonary Study

Evaluation of Pulmonary Perfusion and Ventilation with the Nuclear-Chicago Pho/Gamma® Scintillation Camera

This technique for dynamic regional pulmonary evaluation uses ^{133}Xenon and the Pho/Gamma Scintillation Camera. The camera is equipped with the diverging hole collimator, allowing the entire lung field to be visualized.

SETTING-UP. The collimator is placed against the posterior thorax. Positioning flexibility of the Pho/Gamma detector permits this study to be performed with the patient in either the upright or supine position, thus accommodating even the patient with limited or no mobility.

ISOTOPE AND DOSE. The clinician administers 30 mC of ^{133}Xe in sterile solution intravenously as a bolus injection to an antecubital vein. The patient exhales the xenon into a spirometer; he also rebreathes from the spirometer, thus providing data for determination of ventilation.

DATA ACCUMULATION. Serial scintiphotos are taken in the divided output mode, each representing approximately 60,000 counts in a 10-15 second period. (Note that Pho/Gamma can be operated on either a preset-time, preset-count, or preset-time/count basis as desired.) Also, as illustrated, time-activity histograms were made using a dual-channel ratemeter/dual-pen chart recorder.

If desired, this data could have been recorded in high-resolution digital form, on the Nuclear-Chicago Data-Store/Playback Accessory, or in digital form on the CDS-4096 Clinical Data System. Both of these system accessories allow stored patient data to be replayed, processed and manipulated in a variety of ways to produce additional qualitative and quantitative data.

EVALUATION. For comparison purposes, the standard chest radiograph on the opposite page shows a patient presented with mild dyspnea. The four serial scintiphotos illustrate the various stages of the pulmonary study. Finally, the recorder plots of each lung constitute time-activity histograms of a complete 8-minute study.

In each histogram, the initial rise indicates passage of xenon into the lungs. Breath-holding at this time permits accumulation of data for determination of regional perfusion (Q, scintiphoto 1). Then, rebreathing from the spirometer to equilibrium is followed by a second deep breath to obtain data on lung volume (V_2, scintiphoto 2). Wash-out follows on expiration (scintiphoto 3), after which a final deep breath is taken to determine regional ventilation of a single breath (V_1, scintiphoto 4).

CONCLUSIONS. The several data presentations shown here demonstrate that in this case blood flow to the left lung is considerably less than to the right lung. The third scintiphoto shows an irregular pattern of delayed wash-out, characteristic of obstructive lung disease.

This Pho/Gamma ^{133}Xenon technique has been routinely performed on a large number of patients with a variety of lung disorders. It is felt that this data, when augmented with other diagnostic information, will assist the physician in making more definitive diagnoses. The results illustrate the severity of the disease states to be evaluated as well as the effectiveness of treatments indicated for the specific disorder.

Nuclear Reviews

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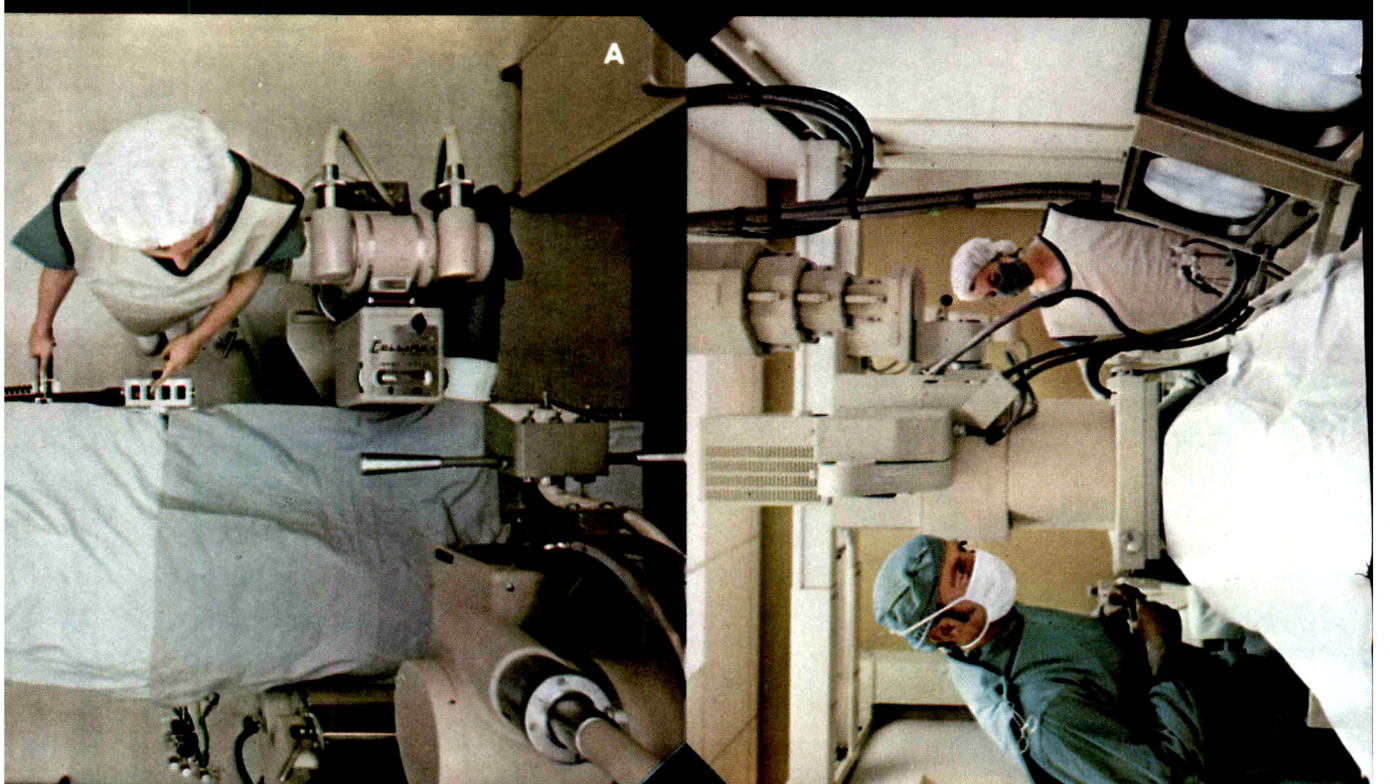
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For complete information, or for a demonstration of the system in use, call or write Westinghouse Medical Products Divisions, Plant: 2519 Wilkins Ave., Baltimore, Md. 21203. Tel. (301) 233-2300; General sales office: 4701 Lydell Rd., Cheverly, Md. 20781. Tel. (301) 322-4200.

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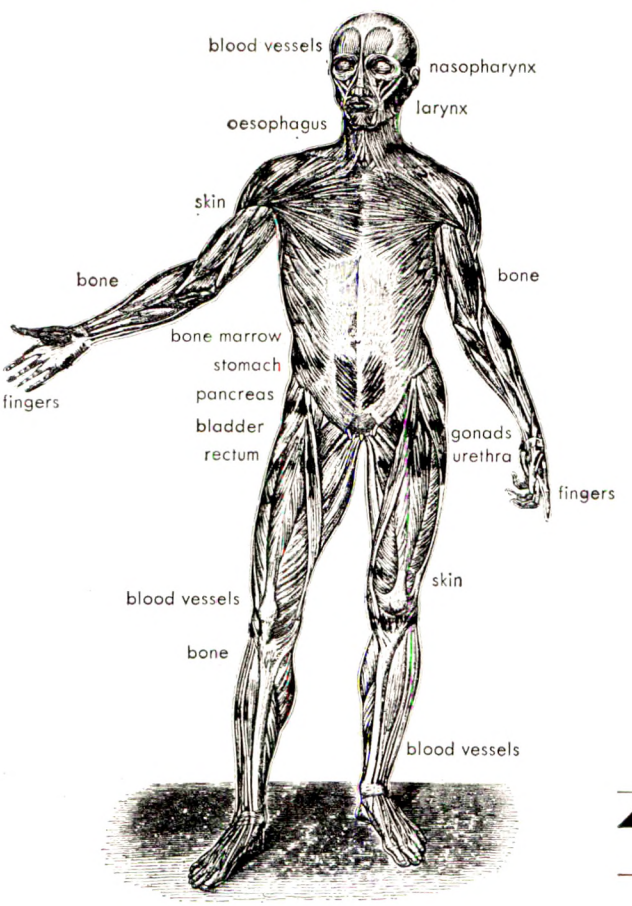
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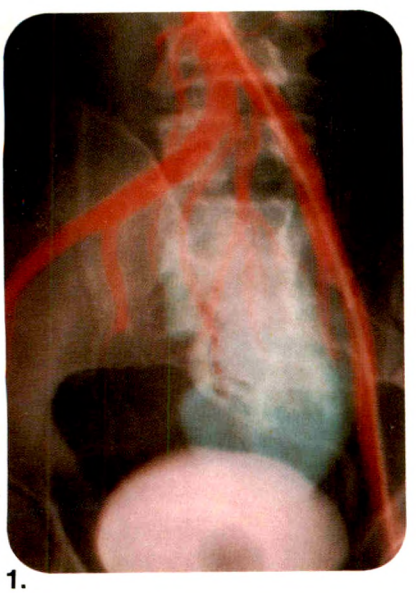
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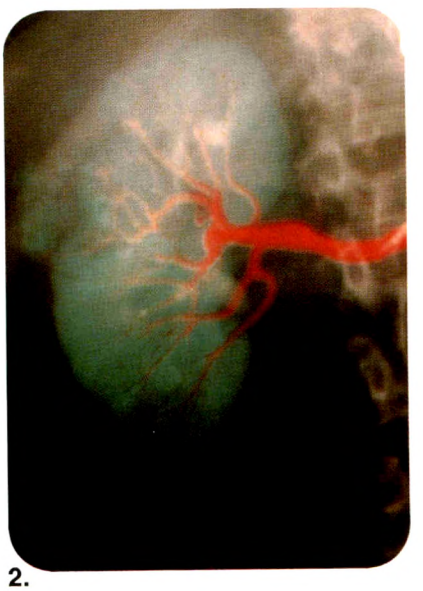
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6. **Selective Renal Angiogram:**
   Renal cyst

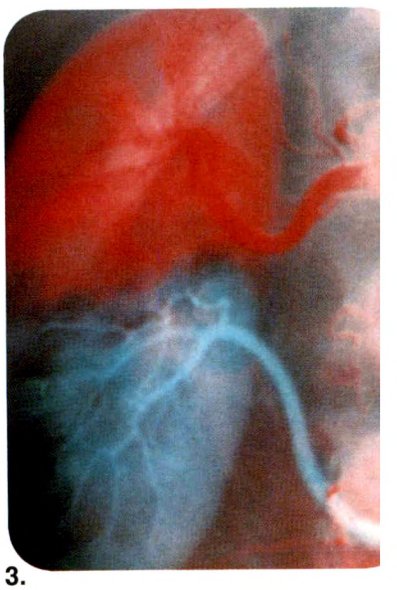
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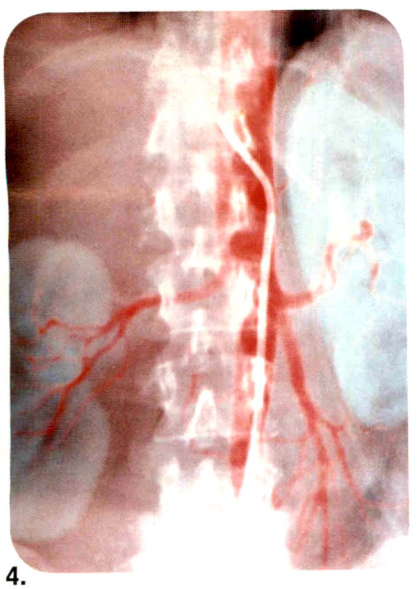
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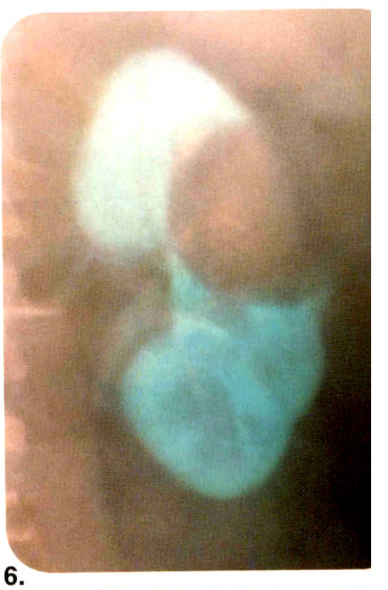
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HYPAQUE® SODIUM (brand of **sodium diatrizoate, USP**) 50% Sterile Aqueous Injection (Weight/Volume)

EXCRETORY UROGRAPHY AND DRIP INFUSION PYELOGRAPHY

Description: Hypaque sodium is sodium 3,5-diacetamido-2,4,6-triiodobenzoate, a radiopaque medium used to delineate internal structures. Hypaque sodium 50 per cent contains 300 mg. iodine per ml. Contains calcium disodium edetate 1:10,000 as stabilizer. Na_2CO_3, NaOH, and HCl added to adjust pH between 6.5 and 7.5. Not for multiple dose use. Should be protected from strong light.

Warning: Do not use Hypaque sodium for myelography. Injection of even a small amount into the subarachnoid space may produce convulsions and result in fatality.

General and Specific Precautions: Caution is advised in patients with a history of bronchial asthma, other allergic manifestations, or of sensitivity to iodine; these conditions represent a special risk, though not an absolute contraindication. To avoid or minimize possible allergic reactions, premedication with antihistamines may be considered. **Benadryl® (brand of diphenhydramine hydrochloride) in the same syringe with Hypaque may result in precipitation.** A test dose of contrast medium is not entirely reliable. Severe reactions, including fatalities, have occurred with a test dose as well as with the full diagnostic dose.

Caution is advised in patients with severe cardiovascular disease. In patients with impaired renal function and cardiovascular disease, drip infusion pyelography imposes a sudden osmotic and sodium load which can precipitate congestive heart failure. Contrast media injected into arteries or veins can promote sickling of red cells in susceptible individuals. In patients with reduced renal function, repeat of excretory or retrograde pyelography should be delayed at least 48 hours to avoid temporary suppression of urine. In preparation for excretory urography of patients with multiple myeloma, partial dehydration is not recommended, since this may predispose to the precipitation of myeloma protein in the kidney tubules. When thyroid function studies (PBI and 24-hour radioiodine uptake levels) are indicated, they should be performed *prior* to radiographic studies or several days afterwards, to avoid inaccurate results.

General and Specific Adverse Reactions: Adverse reactions, usually of a minor nature, have occurred in 10-14 per cent of patients who have received Hypaque intravenously. Reactions due to *faulty technique* include hematomas and ecchymoses, following extravasation from the vein, and pyrogenic reactions. *Hemodynamic reactions* include vasodilatation with flushing, hypotension and, rarely, vein cramp or thrombophlebitis. Serious *cardiovascular reactions* include rare cases of cardiac arrhythmias (e.g., ventricular fibrillation), shock, and cardiac arrest. Transient proteinuria may occur occasionally following the injection of radiopaques and, rarely, oliguria and anuria have been reported secondary to a hypotensive reaction. *Allergic reactions* include asthmatic attacks, nasal and conjunctival symptoms, cutaneous reactions such as urticaria and, rarely, anaphylactic shock, sometimes with fatal outcome. Severe reactions may also be manifested by signs and symptoms relating to the *respiratory system* (dyspnea, cyanosis, pulmonary or laryngeal edema), or to the *nervous system* (restlessness, confusion, or convulsions). *Other reactions* include nausea, vomiting, excessive salivation, anxiety, headache, and dizziness. Infrequently, "iodism" (salivary gland swelling) from organic compounds appears two days after exposure and subsides by the sixth day. Reactions to drip infusion pyelography may not appear until some hours after the examination.

ANGIOGRAPHY (PERIPHERAL ARTERIOGRAPHY AND VENOGRAPHY)

Precautions *(See also General Precautions):* Extreme caution is advised in considering peripheral arteriography in patients suspected of having thromboangiitis obliterans (Buerger's disease). Any procedure (even needling or insertion of a catheter) may induce a severe arterial and/or venous spasm. Caution is also advisable in patients with severe ischemia associated with ascending infection.

Adverse Reactions *(See also General Adverse Reactions):* In addition to those listed under General Adverse Reactions, are those due to arterial trauma during the procedure (i.e., needling, insertion of catheter, subintimal injection, perforation, etc.) as well as to the hypertonicity or effect of the medium; also reported are transient arterial spasm, extravasation, hemorrhage, hematoma with tamponade, injury to nerves proximal to artery, thrombosis (rare in venography, if vein is irrigated following injection), dissecting aneurysm, arteriovenous fistula, (e.g., with accidental perforation of femoral artery and vein during needling), transient leg pain from contraction of calf muscles in femoral arteriography; transient hypotension after intraarterial (brachial) injection, and brachial plexus injury with axillary artery injections.

CEREBRAL ANGIOGRAPHY

Inasmuch as cerebral angiography is a highly specialized procedure requiring the use of special techniques, it is recommended that Hypaque Sodium (brand of sodium diatrizoate) be used for this purpose only by persons skilled and experienced in carrying out the procedure.

Contraindication: Carotid angiography should be avoided during the progressive period of a stroke because of the increased risk of cerebral complications.

Precautions *(See also General Precautions):* Select patients for this procedure with care. Use the 50 per cent solution with caution in extreme senility (but not old age *per se*), advanced arteriosclerosis, severe hypertension, and cardiac decompensation.

The diagnostic value of the procedure, according to many authorities, when employed early enough in locating lesions amenable to surgery, may outweigh the added risk to patients who have recently experienced cerebral embolism or thrombosis (stroke syndrome). A small number of postangiographic fatalities, including progressive thrombosis, have occurred in which the procedure did not appear to play a role. Patients with severe cerebrovascular disease may be examined primarily by indirect methods of angiography.

Care should be exercised to avoid contaminating catheters, syringes, needles, and contrast media with glove powder or cotton fibers.

Angiography is hazardous in subarachnoid hemorrhage. In migraine, the procedure can be hazardous because of ischemic complications, particularly if performed during or soon after an attack.

Adverse Reactions *(See also General Adverse Reactions):* The major sources of complications are faulty technique, occlusive atherosclerotic vascular disease, repeated injections of the contrast media, and higher doses than those recommended.

Untoward reactions are mostly mild and transient, although permanent visual field defects and deaths have been reported.

Vascular reactions: flushing, vessel spasm, thrombophlebitis, and cutaneous petechiae.

Neurologic complications: transient cerebral blindness, neuromuscular disorders, convulsions, coma, hemiparesis, unilateral dysesthesias, visual field defects, aphasia, and respiratory difficulties.

AORTOGRAPHY

Precautions *(See also General Precautions):* Special caution is advised to avoid inadvertent intrathecal injection when using the translumbar technique. The incidence and severity of reactions or complications that may occur are influenced by: the care and experience with which the procedure is performed; the amount and type of medium used; the age and condition of the patient; and the premedication and anesthesia used. Since aortography is not without some danger, it should be performed only by those experienced in the technique. Wherever possible, repeated injections of the solution during a single study should be avoided.

Adverse Reactions *(See also General Adverse Reactions):* Renal damage and shutdown; neurologic complications such as transverse myelitis or paraplegia; cardiovascular complications such as shock, cardiac arrest or failure, coronary occlusion, arterial thrombosis, embolism, and perforation of vessels; extravasation or hemorrhage from the puncture site or retroperitoneal catheter perforation; necrosis of the intestinal wall; acute pancreatitis; deaths; diffuse cutaneous petechiae; subintimal injection and aortic dissection.

INTRAOSSEOUS VENOGRAPHY

See General Precautions and Adverse Reactions. A general anesthetic is sometimes necessary since the method is painful. Occasionally, extravasation of the contrast medium from the needle into the soft tissue may occur.

DIRECT CHOLANGIOGRAPHY

Precaution *(See also General Precautions):* This procedure should be used with caution in the presence of acute pancreatitis, injecting no more than 5 to 10 ml. without undue pressure.

Adverse Reactions *(See also General Adverse Reactions):* These may be attributed to injection pressure or excessive volume of the medium, resulting in overdistention. Such pressure may produce a sensation of epigastric fullness, followed by moderate pain in the back or right upper abdominal quadrant, which will subside when injection is stopped.

Some of the medium may enter the pancreatic duct and cause a transient serum amylase elevation 6 to 18 hours later, without apparent ill effects. Pancholangitis resulting in liver abscess has been reported. Occasionally, nausea, vomiting, fever, and tachycardia have been observed.

HYSTEROSALPINGOGRAPHY

Contraindications: Menstruation, active or imminent; infection in any part of the genital tract, including the external genitalia; pregnancy, known or suspected. Use not advised for six months after end of pregnancy, or 30 days after conization or curettement.

Precaution *(See also General Precautions):* Caution should be exercised in patients with known or suspected carcinoma to avoid possible spread of the lesion by the procedure.

SPLENOPORTOGRAPHY

Precautions *(See also General Precautions):* Procedure should be performed with caution on patients with blood dyscrasias, a tendency to severe bleeding, or a spleen recently become tender and palpable.

Adverse Reactions *(See also General Adverse Reactions):* Persistent hemorrhage with hemoperitoneum has been reported, and, in some instances, hemorrhage has required splenectomy.

How Supplied: Ampuls of 30 ml., boxes of 10 and 25. Boxes of 25—1 ml. ampuls for testing sensitivity are available upon request. Vials of 20 and 30 ml., rubber stoppered, boxes of 10 and 25. Vials of 50 ml., rubber stoppered, boxes of 10. Each vial contains sufficient excess to permit withdrawal of 1 ml. for testing sensitivity.

If the solution is chilled, crystals may form but will readily dissolve if the ampul or vial is placed in moderately hot water before use. Cool to body temperature before injecting.

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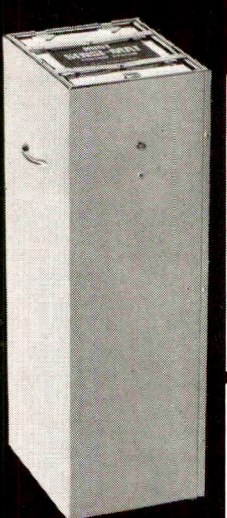
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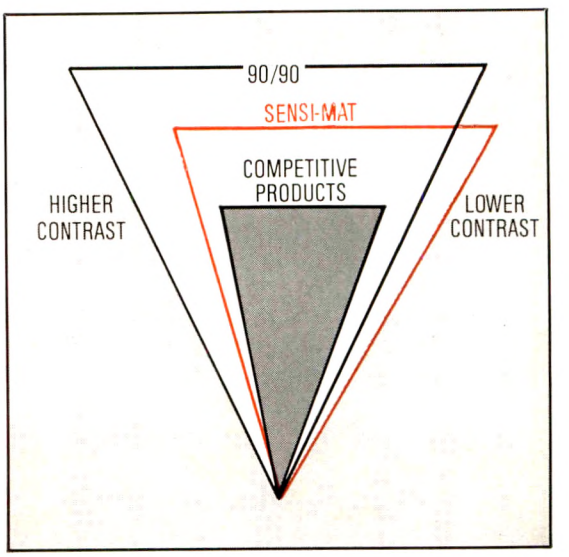




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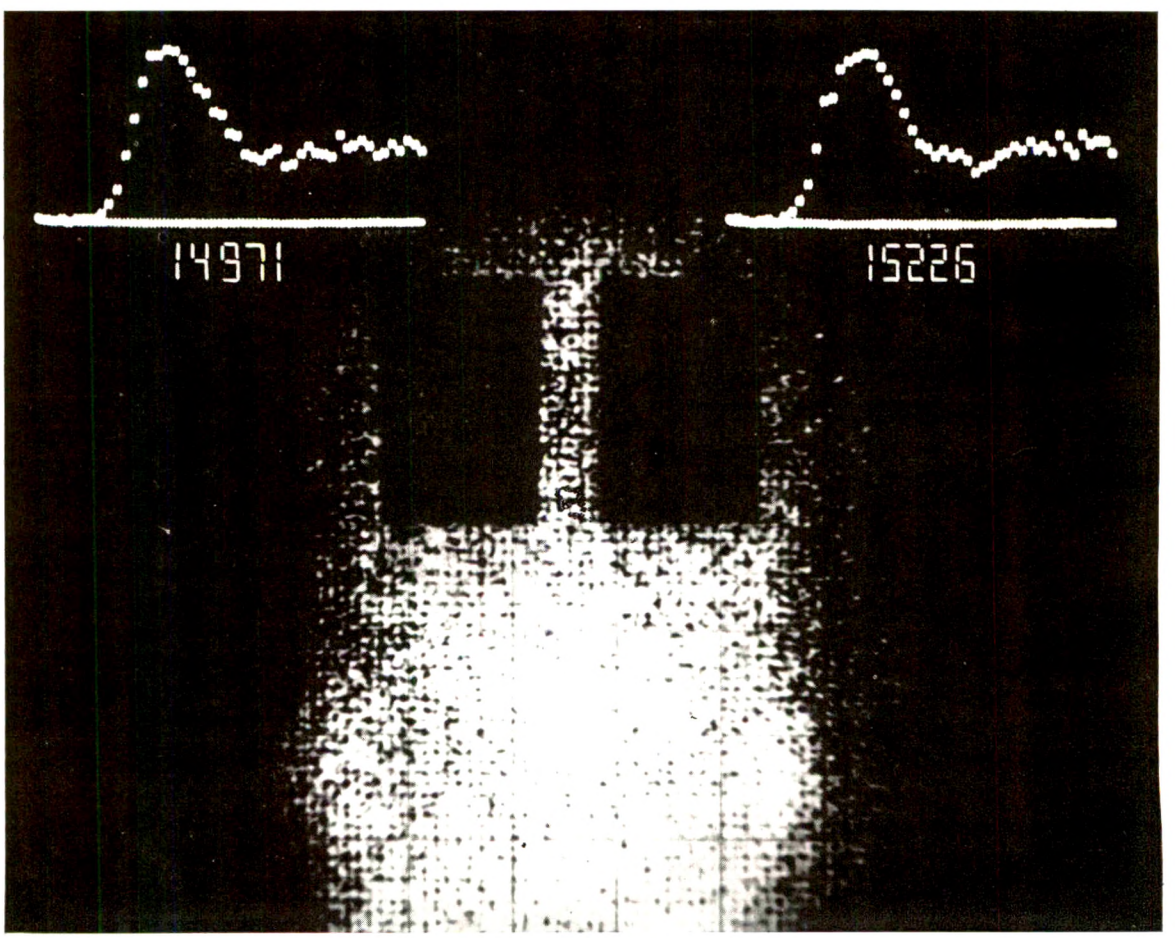
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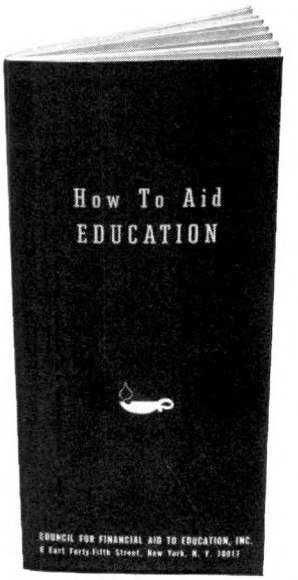
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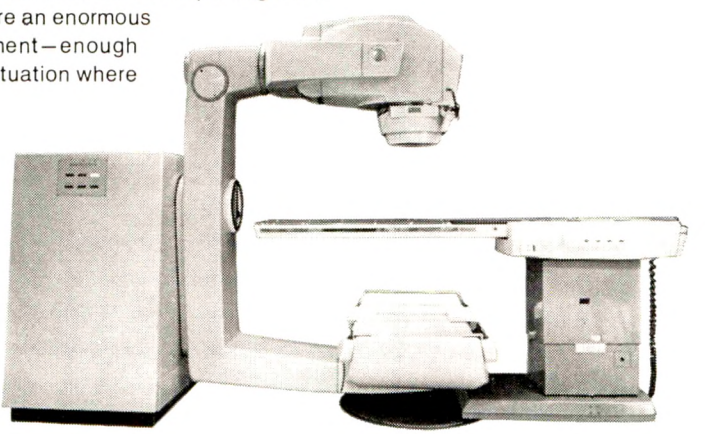
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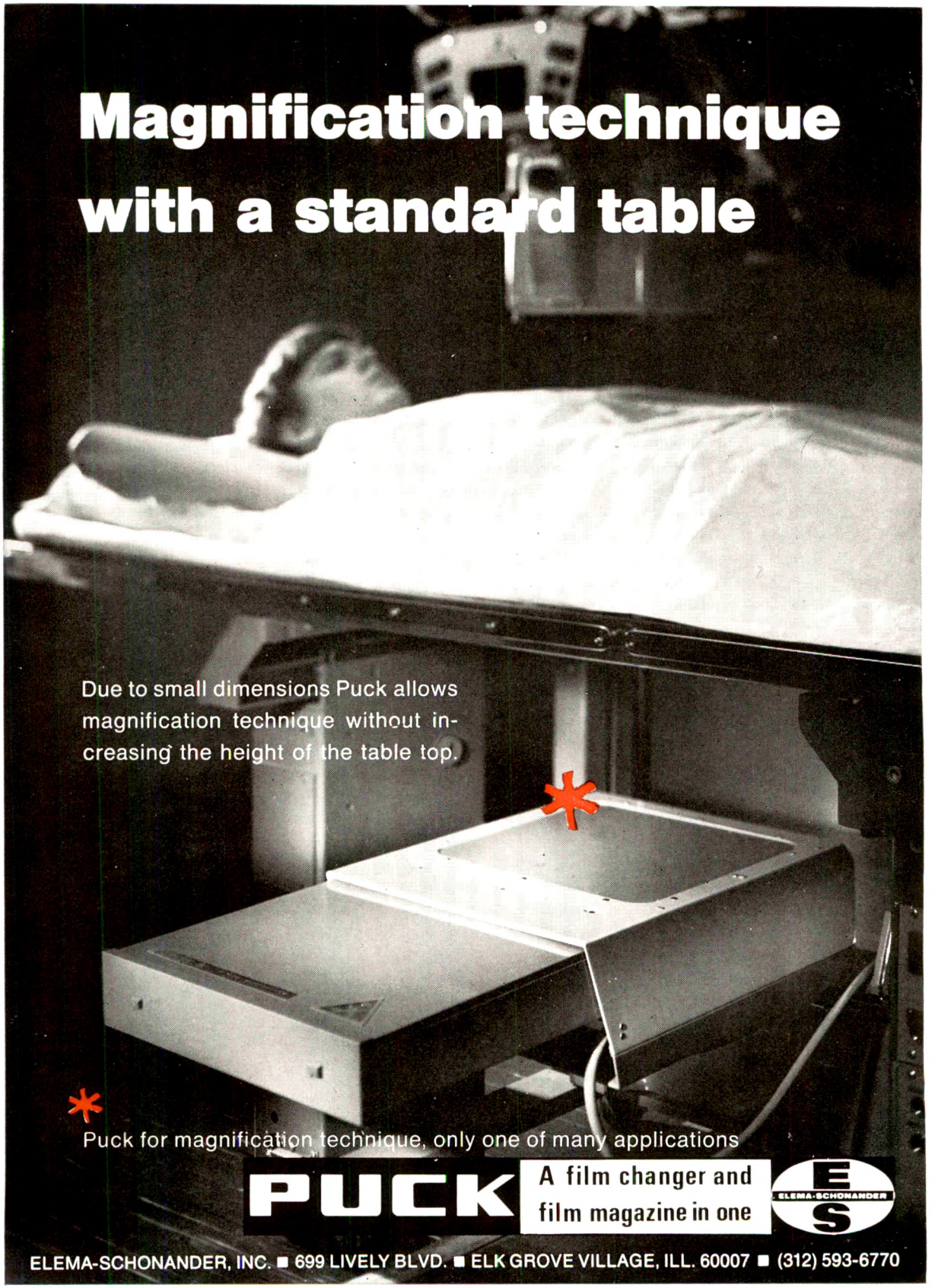
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SABULOGRAPHY*

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MONTREAL, CANADA

THE faintly visualized gallbladder in which the presence or absence of calculi cannot be affirmed is a continuous challenge to the radiologist. By means of a massive reinforcement dosage of telepaque, given over a 2 day period, it is at times possible to opacify fine sand or even sludge-like material which would otherwise remain undetected. These types of amorphous contents have the same clinical significance as well-formed calculi; hence, the importance of demonstrating their eventual presence. It has been emphasized by Staple and McAlister[12] that "one of the principal reasons for false negative cholecystography is the failure to detect gallbladder sand."

Since the specific aim is to bring to evidence small amounts of fine granules which are not discernible in the poorly functioning gallbladder after the routine dosage, we suggest that our 2 day massive telepaque test be called *sabulography* (*sabulum*, Lat., sand). The concept has some similarity with the iodine coating of relatively large stones in the common duct and in the gallbladder which can be obtained with the four day telepaque test devised by Salzman and Warden[6] and later called calculography by Kuster *et al.*[3]—a name which is now widely accepted.

PRINCIPLE

Some degree of opacification of the gallbladder is necessary, however faint it may be, in order to ascertain the patency of the cystic duct. Once all extraneous causes have been ruled out, the poor visualization implies that the gallbladder is diseased. One may then presume that it might contain abnormal material (sluggish bile, calculi). On this basis, we speculated that the administration of a large amount of telepaque, subdivided into a number of intakes over a 2 day period, might force enough contrast material through the cystic duct to permeate the inspissated and stagnant bile in the gallbladder and to reveal lithiasic elements if present. Initially, our system of dosage was meant to rule out the presence of gallstones by means of increased radiopacity. It happened that

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amorphous lithiasic material was surprisingly well visualized and some peculiar features of the abnormal substance demonstrated. This result was explainable by a process of impregnation of the content of the gallbladder by prolonged contact with a richer iodinated fluid. This interpretation is somewhat similar to the mechanism suggested by Salzman *et al.*[5–10,16] to account for the coating of radiolucent stones in the common duct (rim sign).[6]

ADMINISTRATION AND DOSAGE OF TELEPAQUE

For more than 16 years the routine dosage of iopanoic acid for cholecystography at Hôtel-Dieu de Montréal has been 6 tablets of telepaque (3 grams) after the noon meal and 6 tablets again after the evening meal on the day preceding the examination, giving a total of 6 grams. If immediate reading of cholecystograms made the next morning reveals a gallbladder too faint to permit affirmation or negation of lithiasis, the use of our so-called sabulography method may well be recommended. It is especially indicated if the patient has a history of biliary colic, or if previous cholecystography has revealed a poorly opacified gallbladder. This type of reinforcement technique consists of an additional dose of telepaque (18 tablets) begun immediately following the first cholecystographic examination. This additional amount is administered in 3 equal intakes of 6 tablets each; *i.e.*, 6 tablets after lunch, 6 tablets after a 6 P.M. meal, and 6 tablets after a late evening snack. The grand total of iopanoic acid ingested by the patient over a period of 36 hours is thus 30 tablets or 15 grams. It should be noted that fatty foods are forbidden on the second day, whereas no restriction as to quality or quantity of food was made on the first day of preparation. Paregoric may be prescribed if the patient had diarrhea after the "routine" dosage schedule.

The decision to proceed with this method is taken only after the risks of side-effects of such high dosage have been weighed against the importance of knowing if this particular gallbladder contains abnormal material or not. The technique is contraindicated if the patient's renal function is altered and caution should be exercised if hepatic function tests are abnormal.

ROENTGENOGRAPHIC PROCEDURE

The examination should be performed by the radiologist himself, with the use of compression spot cholangiograms and the full range of positions he may select in order to remove all possible sources of error. According to the *modus operandi* set up by Jutras,[2] erect position is essential, but prone, supine, oblique and Trendelenburg positions are also regular requirements.

In order to obtain optimal contrast, the kilovoltage should be set low (in the 60–80 range), because gallbladders submitted to sabulography will remain *a priori* faintly opacified even following the overdose of telepaque. The mas. is modified to adjust the radiation output to the volume of the patient.

CHOLECYSTOGRAPHIC FINDINGS

As a rule, the plain roentgenogram of the abdomen before administration of contrast medium does not reveal any abnormal opaque shadow in the right upper quadrant. The cholecystograms obtained after the "routine" dose of 6 grams of telepaque show a rather capacious and faintly opacified cholecyst of homogeneous appearance, without evident calculus. The examination on the following morning, after ingestion of an additional 9 grams of telepaque, results in little or no improvement in the radiopacity of the viscus. On the cholecystograms taken in the erect position, the gallbladder contains in its lowermost portion a sediment which may appear either as a homogeneous opacity, slightly denser than the surrounding bile, or as a light radiolucent formation punctated with numerous opaque dots.

In our experience, this sediment is composed of sand and mud totally or partially

impregnated with cholepaque material. It may settle in the fundus or be divided into two aggregates, one in the fundus and the other in the lower part of the infundibulum. Its upper border may be linear, or slightly serrated with a horizontal, oblique or sinuous direction, depending on the cohesion of the grains of sand, the viscosity of the supernatant bile, or the presence of interposed mud. On the cholecystograms taken in the Trendelenburg position, some time must be allowed to pass before the contents are dislodged from the fundus into the infundibulum. In one case, a 70° Trendelenburg position was maintained for 5 minutes before the amorphous material could be displaced from the fundus, and it then appeared as a large oval bolus, relatively radiolucent, and constellated with opaque specks, settling in the center of the viscus.

Once the sand and mud have been saturated with cholepaque, the grains retain their opacity for several days, while the poor opacification of the gallbladder will fade within 24 to 48 hours. These facts point to an important biliary stasis. As a consequence, it seems logical to give a high dosage of cholecystographic medium over a short period, in order to permeate the glutinous, stasic bile and to interpenetrate the sticky conglomerate of sand and mud.

SURGICAL AND PATHOLOGIC FINDINGS

At surgery, the gallbladder usually appeared globular and under slight tension. The sand could not be palpated, except in 1 case where the amorphous content was so abundant that it could be felt as a resilient mass slipping between the surgeon's fingers.

The cystic and hepatic ducts were often enlarged. In 2 cases, abnormal contents were found in the common hepatic duct after it was opened. In 1 case, the duct was clogged by a formation of thick mucus and cohering sand which could be extracted as one lump. This patient had suffered an attack of biliary colic 10 days before the operation. In another case the turbid bile was seen coming under pressure from the common hepatic duct. Two tiny calculi (2 and 4 mm. in diameter) were found enclosed in the papilla of Vater. This patient had complained of repeated bouts of biliary colic and 7 days prior to cholecystectomy a mild transient jaundice had been noted.

Upon opening the gallbladder specimens, a thick and viscid bile of a dark green or black color escaped. No sizeable calculus was seen, except in 1 case where 6 calculi 3 to 4 mm. in diameter were admixed in the sand. Two similar calculi had been found in the papilla of Vater at operation. The sand was composed of grains of variable size and color. The general color of the sand was light brown, rusty, or black.

When the contents of the gallbladder were poured into a test tube, the sand collected slowly at the bottom and adhered to the wall. After a few days of rest, the coarser grains occupied the lowest portion while the finer ones layered above. A more or less thick coat of mud and haze formed in the supernatant liquid. A few black, yellow, and red grains were intermixed in the generally brown sand. The roentgenogram of the test tube may reveal a rather homogeneous opacity if nearly all the grains have been impregnated by cholepaque medium. In other cases, when only some of the grains have been impregnated, a radiolucent column constellated with opaque specks may be seen.

REPORT OF CASES

Case 1. A.A., a 60 year old woman, had a history of repeated attacks of angina pectoris following a cardiac infarction in 1961. Cholecystography had been performed in 1960 for flatulent dyspepsia. The gallbladder was then moderately opacified and slightly globular; it contained no demonstrable calculus (Fig. 1*A*).

In January 1966, she was hospitalized with a history of retrosternal pain of 1 week's duration. Four days previously, she had had an episode of vomiting and diarrhea, with epigastric pain radiating to the right arm and shoulder and accompanied by numbness of the right arm. The evacuation roentgenogram after a barium enema examination carried out before cholecystography showed no calcific density in the gallbladder area (Fig. 1*B*).

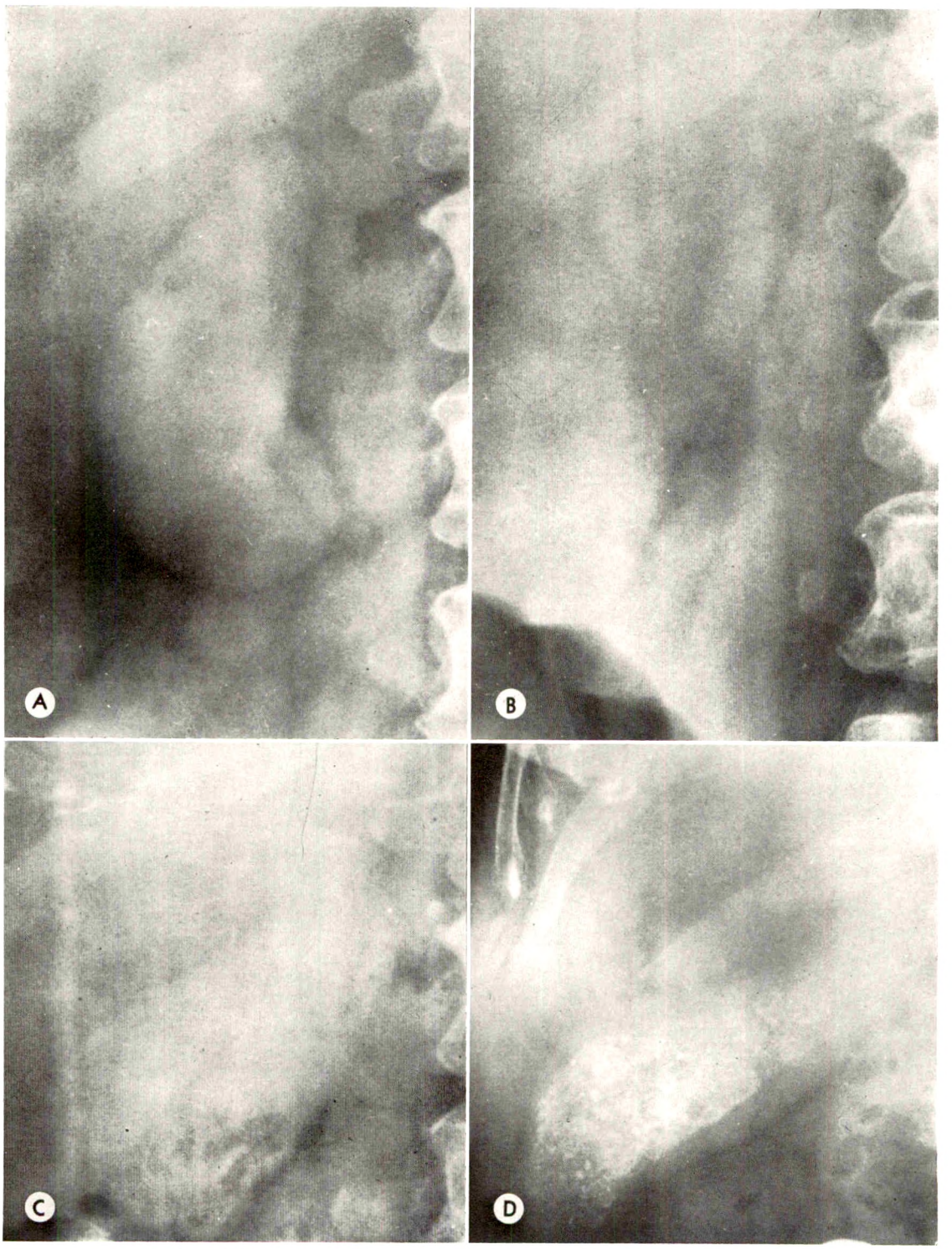


FIG. 1. Case 1. (*A*) The gallbladder is moderately opacified and globular; it contains no demonstrable calculus. (*B*) The evacuation roentgenogram from a barium enema examination obtained before cholecystography shows no calcific density in the gallbladder area. (*C*) Cholecystography performed after "routine" intake of 6 gm. iopanoic acid reveals a faintly opacified viscus, but without evidence of lithiasis. (*D*) After sabulog-

The patient had a white blood cell count of 9,900 with 76 per cent neutrophils and 10 per cent lymphocytes. The hematocrit was 38 per cent and the corrected sedimentation rate 28 mm. Direct and total bilirubin tests were normal. SGOT was 15 and SGPT was 3. Cholecystography performed after the "routine" intake of 6 grams of iopanoic acid revealed a faintly opacified viscus, less dense and smaller than in 1960, but without evidence of lithiasis (Fig. 1*C*).

After sabulography (9 grams of iopanoic acid taken following the routine dosage of 6 grams), the gallbladder remained faintly visualized, but within it a large, heterogeneously opaque formation was demonstrated, grossly reproducing the outline of the gallbladder. This corresponded to a conglomeration of sand and mud, the grains being of different size and displaying a varied affinity for impregnation by the cholepaque medium (Fig. 1*D*).

At operation, the viscus appeared voluminous, but without adhesions. The diameter of the cystic duct was 3 mm. The hepatic duct was slightly enlarged, and upon its opening an elongated plug of very thick mucus embedded with lithiasic debris and biliary pigments was easily extracted. After washing of the hepatic duct, no residual debris were noted. The gallbladder specimen contained roughly 20 cc. of dark green bile admixed with mud and sand (Fig. 2, *A–E*).

Case II. A.B., a 65 year old male, was admitted to the emergency department for an acute epigastric pain which had started after his lunch, 1 hour before. The pain radiated across the abdomen and to the back. It was not relieved by nitrogylycerin and was not accompanied by nausea and vomiting. He had sustained many similar attacks during the previous 6 years. Five years ago, he had been hospitalized in another institution for a cardiac infarction, and had since suffered from coronary insufficiency. He had a history of alcoholism. His white blood cell count was 18,200, with 80 per cent neutrophils and 20 per cent lymphocytes. SGOT was 235 and SGPT 209. Sedimentation rate was at 25 mm. Thymol test was 13.7 units (normal 3 to 4 units). Thymol flocculation was +++. Cholecystography, performed after the usual intake of 6 grams of iopanoic acid, revealed an enlarged liver and a low-lying gallbladder, faintly opacified, elongated and homogeneous in appearance (Fig. 3*A*).

The faint concentration of the gallbladder was attributed clinically to the hepatic insufficiency, but the biliary colic remained unexplained. After this first cholecystography, it was decided to resort to sabulography the same day in order to rule out amorphous lithiasis. An additional dose of 9 grams of iopanoic acid was administered to the patient in 3 parts, at 6 hour intervals. The cholecystograms obtained the following morning showed a slightly more voluminous gallbladder, with little improvement in concentration. The procumbent cholecystogram revealed in the fundus an elongated, opaque, crescent-shaped, homogeneous shadow, slightly denser than the surrounding iodinated bile and corresponding to the complete impregnation of the sand contained in the viscus (Fig. 3*B*).

On the compression spot cholangiograms taken in the erect position 1½ hours after the fatty meal a slight reduction in the size of the gallbladder was noted, but no improvement in the density of the viscus. The sand was deposited in the lower part of the viscus and its upper margin was slightly sinuous (Fig. 3, *C* and *D*).

The liver was found enlarged at operation, and showed micronodular changes due to nutritional cirrhosis. Fibrous adhesions were seen around the whole gallbladder, the cystic duct, the adjacent transverse mesocolon, and the great omentum. The hepatic duct measured 7 to 8 mm. and the cystic duct 4 mm. in diameter. Fine granules in the gallbladder could be palpated by the surgeon. After opening of the specimen, 50 cc. of bile escaped, and a mass of brownish sand containing no sizeable calculus was seen. Hemorrhagic adhesions were noted

raphy (9 gm. iopanoic acid following the routine dosage of 6 gm. iopanoic acid) the gallbladder remains faintly visualized, but within it a large heterogeneous opaque formation is demonstrated grossly reproducing the outline of the gallbladder. This corresponds to a conglomeration of sand and mud, the grains being of different size and displaying a varied affinity for impregnation of the cholepaque medium.

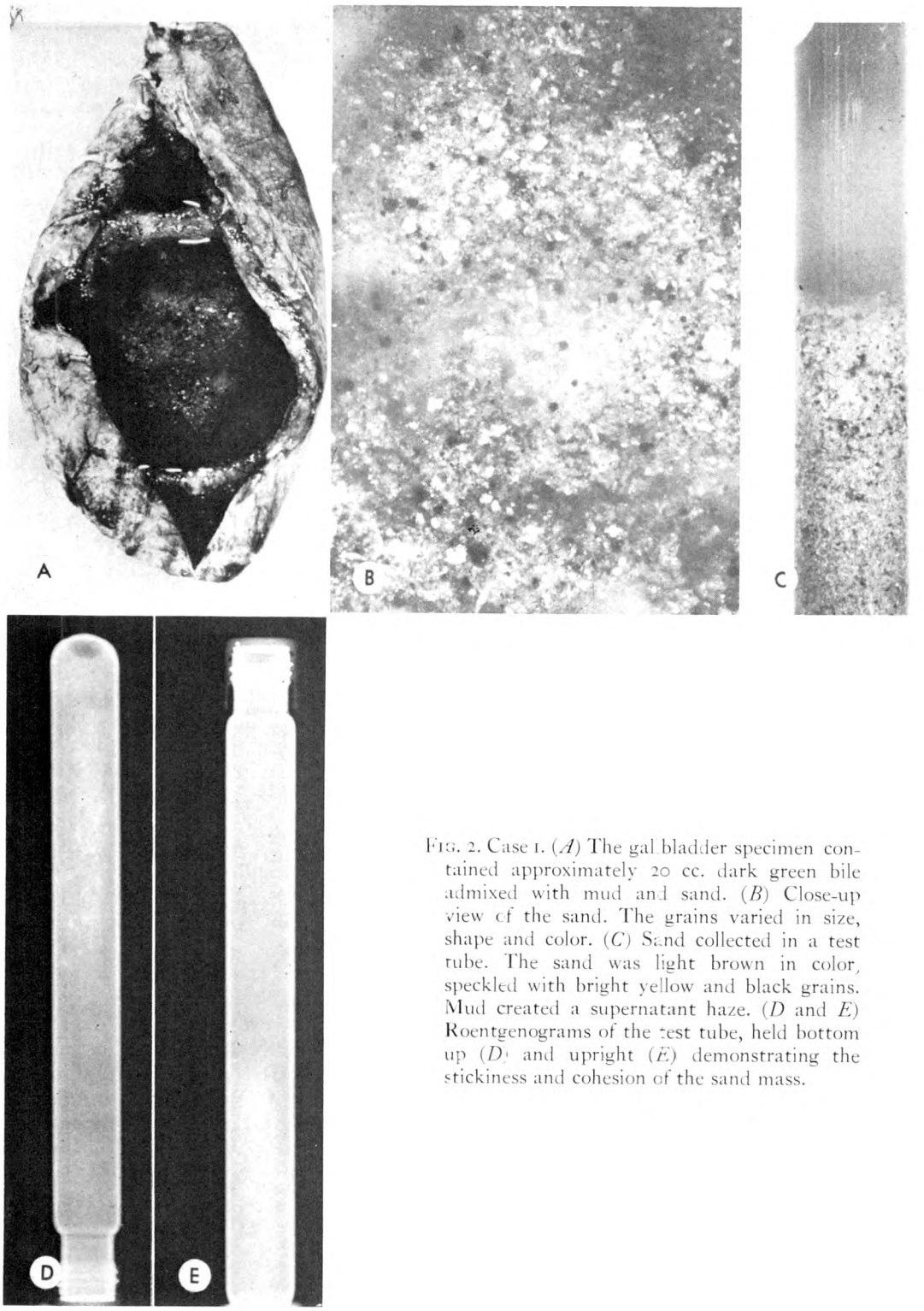


FIG. 2. Case 1. (*A*) The gal bladder specimen contained approximately 20 cc. dark green bile admixed with mud and sand. (*B*) Close-up view of the sand. The grains varied in size, shape and color. (*C*) Sand collected in a test tube. The sand was light brown in color, speckled with bright yellow and black grains. Mud created a supernatant haze. (*D* and *E*) Roentgenograms of the test tube, held bottom up (*D*) and upright (*E*) demonstrating the stickiness and cohesion of the sand mass.

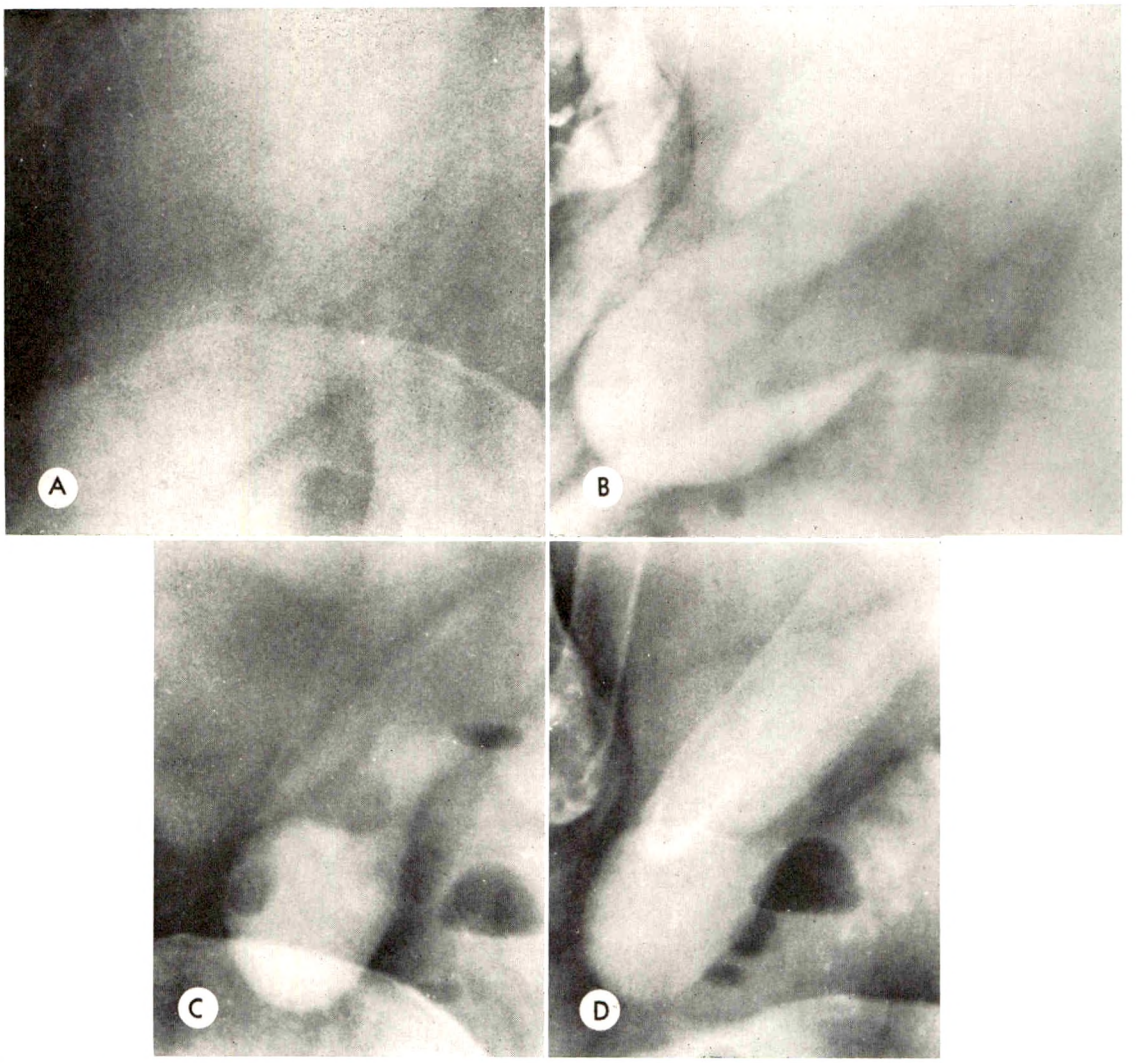


Fig. 3. Case II. (*A*) Cholecystogram, obtained after the usual intake of 6 gm. iopanoic acid, reveals an enlarged liver and a low-lying gallbladder, faintly opacified, elongated, and homogeneous in appearance. (*B*) The morning after sabulography (9 gm. iopanoic acid being administered in 3 parts at 6 hour intervals on the same day), the procumbent cholangiogram reveals in the fundus an elongated, opaque, crescent-shaped, homogeneous shadow, slightly denser than the surrounding iodinated bile, corresponding to the impregnated sand contained in the viscus. (*C* and *D*) Compression spot cholangiograms in the erect position 1½ hours after the fatty meal show reduction in size of the gallbladder and descent of the sand into the lower part of the viscus.

on the external surface of the gallbladder. The lamina propria was invaded by an inflammatory infiltration in which lymphocytes and plasma cells were predominant. Few thrombosed vessels were observed. Rokitansky-Aschoff sinuses were present. The pathologist gave a diagnosis of chronic cholecystitis secondary to biliary sand. Biopsy of the liver produced a report of portal fibrosis with lymphocytic infiltration. In the test tube, there was an excellent stratification of the sand with a supernatant coat of mud (Fig. 4*A*). Roentgenograms of the test tube, inclined at 45° and upright, revealed that the greater part of the grains had adsorbed the opaque medium. The demarcation line between the sand and the adjacent bile was caused by the cohesion of the grains to undulate slightly (Fig. 4, *B* and *C*).

Case III. W.F., a 74 year old male, was

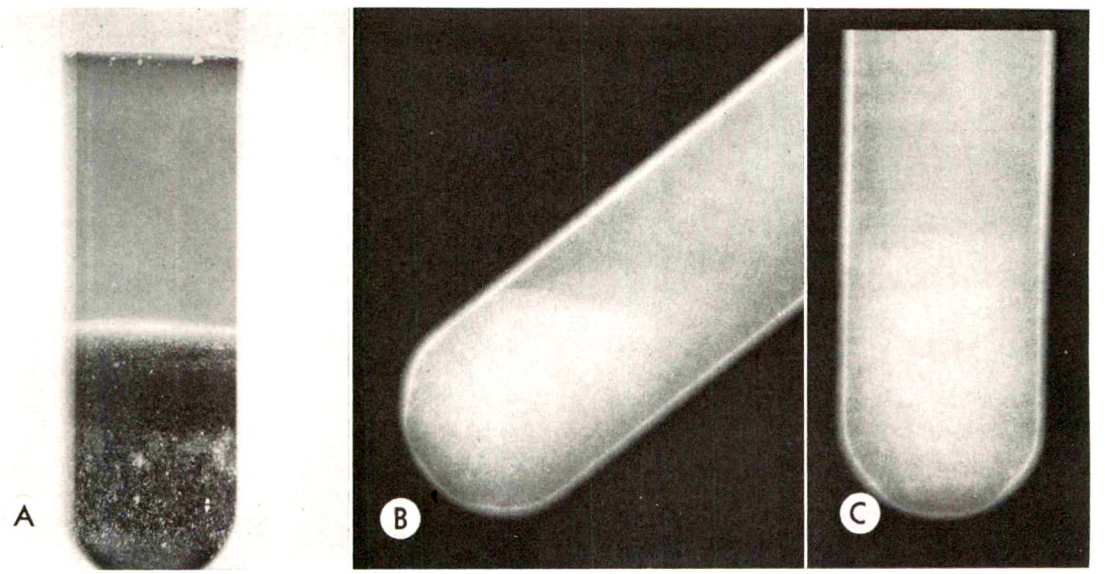


FIG. 4. Case II. (*A*) At operation 50 cc. of bile was aspirated and a mass of brownish sand containing no sizeable calculus was obtained. The test tube shows an excellent illustration of the stratification of the sand with a supernatant coat of mud. (*B* and *C*) Roentgenograms of the test tube, inclined at 45° (*B*) and upright (*C*) reveal that the greater part of the grains adsorbed the opaque medium. The slight undulation of the demarcation line between the sand and the adjacent bile is caused by the cohesion of the grains.

admitted to hospital for constrictive epigastric pain, recurring for the past 2 years and radiating to the back. He had been admitted to another hospital for the same complaint. The last attack had occurred 1 month previously. The pain now radiated to the left axilla and the internal side of the left arm, and was only a little relieved by nitroglycerin. This epigastric pain was mostly provoked by a recumbent position and was improved by antacids. In March 1966, cholecystography after the "routine" administration of 6 grams of iopanoic acid yielded a moderately opacified gallbladder, fairly large, without calculus. The fundus was curved medially over the body of the viscus. The hepatic flexure showed a slight adynamic dilatation (Fig. 5*A*, procumbent cholangiogram; Fig. 5*B*, compression spot cholangiogram in standing position).

In view of the history of biliary colic and of the relatively weak opacity of the gallbladder, the patient was submitted to sabulography. Erect compression spot cholangiogram showed coarse granules of sand settling in the fundus of the gallbladder, which remained, however, faintly opacified (Fig. 5*C*).

The patient was then kept in Trendelenburg position (75 °) for more than 5 minutes, and a compression spot cholangiogram taken; it showed the inert mass of sand dislodged from the fundus to occupy the center of the gall bladder. The sand appeared unevenly opaque and surrounded by a radiolucent rim. The hepatic duct and its branches were slightly dilated (Fig. 5*D*).

Two days later, a spot film was exposed to check the degree of evacuation of the gallbladder which had not emptied after the fatty meal administered following sabulography. Barium was given to opacify the duodenal loop. The gallbladder was still opacified and the sand remained well impregnated. Half the sand occupied the fundus and half had collected in the infundibular portion (Fig. 5*E*).

The decision to operate was postponed until December 1967 when the patient was once more hospitalized. He had been awakened 2 days before by a sharp pain in the right hypochondrium, and had subsequently experienced 5 or 6 similar attacks, each lasting 10 to 15 minutes and with increasing severity. The patient felt weaker on the second day of hospitalization and presented a transient icterus which lasted 2 days. The urine was brownish red. On the third day, he had another attack of pain in the right hypochondrium, rather typical of biliary colic. A mass was palpated at that time and was attributed to an enlarged gallbladder or a pericholecystic reaction. Subicterus persisted. Total bilirubin was 1.32 mg. per

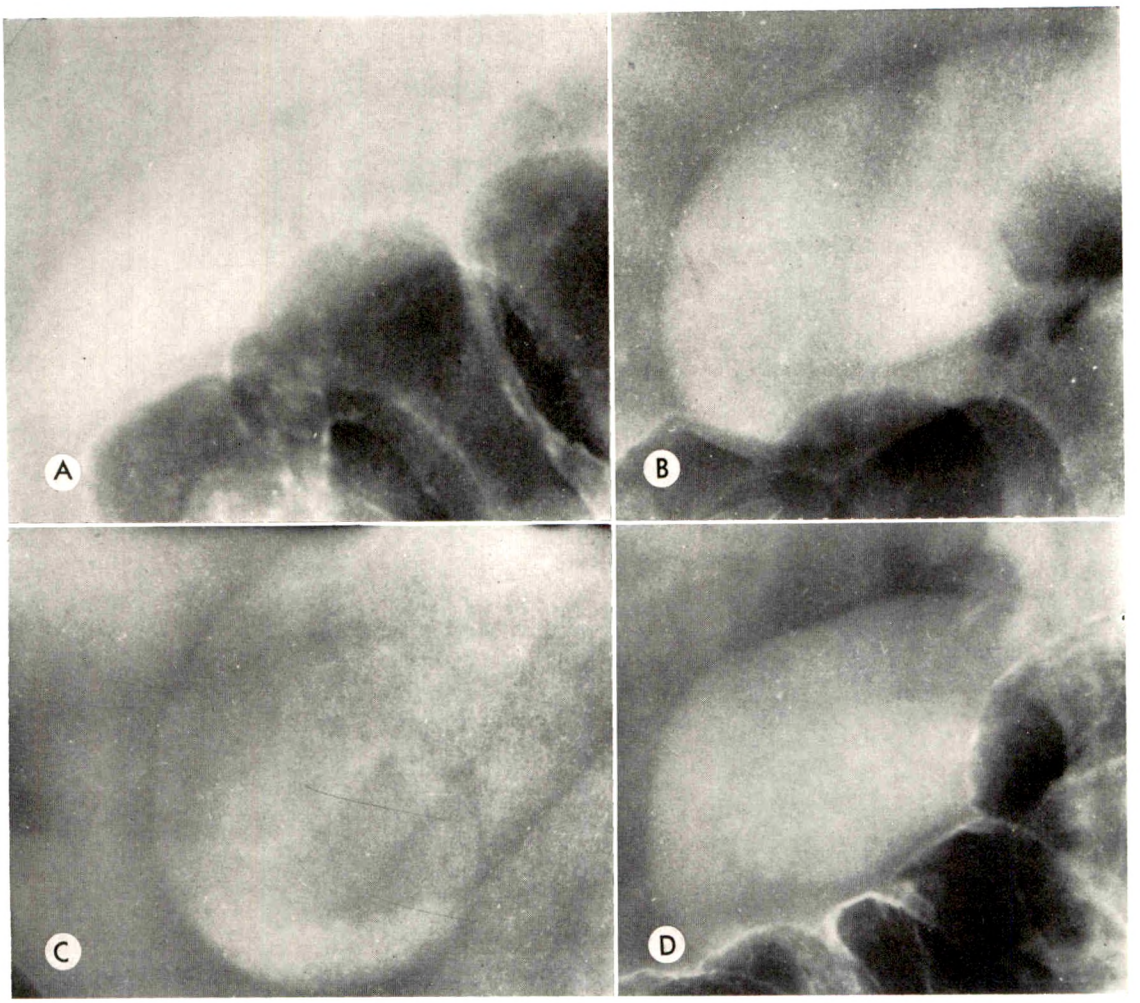


Fig. 5. Case III. (*A*) Procumbent roentgenogram and (*B*) compression spot roentgenogram of routine cholecystography show a fairly large moderately opacified gallbladder, without calculus. The fundus is curved medially over the body of the viscus. The hepatic flexure shows a slightly adynamic dilatation. (*C*) Erect compression spot cholangiogram after sabulography shows coarse granules and sand settling in the fundus of the gallbladder, which remained faintly opacified. (*D*) Compression spot cholangiogram, after the patient was kept in 75° Trendelenburg position reveals an inert mass of sand dislodged from the fundus to occupy the center of the gallbladder. The sand appears unevenly opaque and is surrounded by a radiolucent rim. The hepatic duct and its branches are slightly dilated.

cent (normal 0.2 to 1.2 mg. per cent). The patient was operated 5 days later. The gallbladder was voluminous, elongated, hyperemic but without adhesions. Its content could not be palpated. The hepatic duct measured 1 cm. in diameter. After a choledochotomy, a clear bile escaped under pressure, followed by turbid, mucopurulent fluid. Two tiny calculi, 2 and 4 mm. in diameter, were mixed with the turbid fluid. The gallbladder was opened by the pathologist who gave its mensuration as 10.5 ×2.7 cm. It contained a large amount of mud and sand, and 6 tiny calculi varying from 0.3 to 0.5 cm., some yellow, some brown, of the facetted or mixed type. The amorphous content was poured into a test tube (Fig. 5*F*).

Case IV. R.M., a 58 year old female, was known to have been hypertensive for the past 4 years. For the last 2 years she had been complaining of palpitations and nocturnal precordial pain with dyspnea. Three weeks before hospitalization she experienced an acute pain in the right hypochondrium after her dinner. This pain extended across the abdomen and radiated to the retrosternal and interscapular

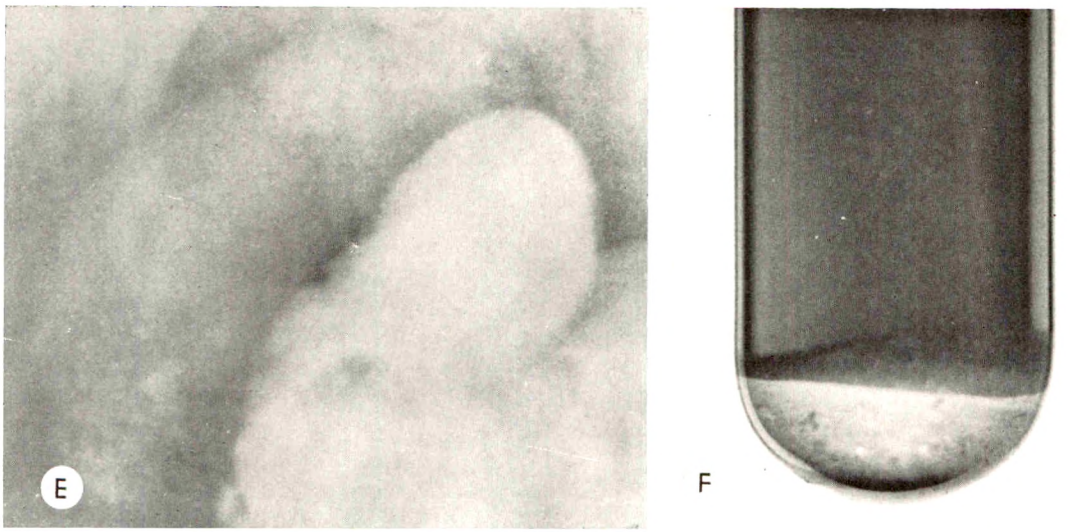


FIG. 5. (*E*) Spot roentgenogram 2 days later (the gallbladder did not empty after the fatty meal), following administration of barium to opacify the duodenal loop, showed that the gallbladder is still opacified and the sand remains well impregnated. One-half of the sand is in the fundus and the other one-half in the infundibular portion. (*F*) Amorphous content of the excised gallbladder in the test tube.

areas and to the right shoulder, and was accompanied by nausea and dyspnea. The attack lasted 2 hours and was not relieved by nitroglycerin. She subsequently had 5 or 6 similar attacks, after a meal or during the night, the most recent occurring 24 hours before admission. Plain roentgenogram of the abdomen revealed no abnormal calcifications in the gallbladder area (Fig. 6*A*).

Cholecystograms obtained after the "routine" intake of 6 grams of telepaque showed a fair-sized gallbladder, faintly opacified, of homogeneous appearance and without recognizable calculi (Fig. 6*B*).

Following the additional dosage of 18 tablets of telepaque for sabulography, the cholecystograms showed a more voluminous gallbladder, but still with low concentration. In its lowermost portion a mound of irregular annular opacities, intermingled with opaque specks, was visible, corresponding to an admixture of sand and muddy pseudo concretions (Fig. 6*C*).

Two hours after a fatty meal the viscus was appreciably reduced in size and the abnormal content differently arranged, but the cholecystic density remained unchanged (Fig. 6*D*).

Three days later, the patient suffered another acute attack in the middle of the night which could not be eased by nitroglycerin, but was later relieved by spasmalgin. Transaminases were normal. Cholecystectomy took place 4 days later. At operation, the gallbladder was voluminous, under tension, and its amorphous content could not be palpated. The cystic duct measured over 5 mm. in diameter. The hepatic canal was dilated, measuring 1 cm. in diameter, and upon its opening dark bile escaped, but no calculi were found. The gallbladder specimen contained biliary mud of paste-like consistency that could be easily spread out with a finger. The pathologist reported edema of the mucosal folds, fibrosis of the perimuscular layer and some intramural diverticula, all of which would correspond to a chronic cholecystitis.

DISCUSSION

Theander[13–15] reported a case of "precipitate of telepaque or of some iodic part or product of this substance" surrounding 2 adhering radiolucent calculi in the gallbladder. The preliminary roentgenograms of the abdomen did not reveal any opaque calculi. After intake of 3 grams of telepaque the gallbladder was not visualized. The repeat examination on the following day, after the same dose, did not bring visualization of the gallbladder, but showed an oval opacity (about 7×12 mm.), presumably in the gallbladder. Cholecystography with biligrafin was carried out 6

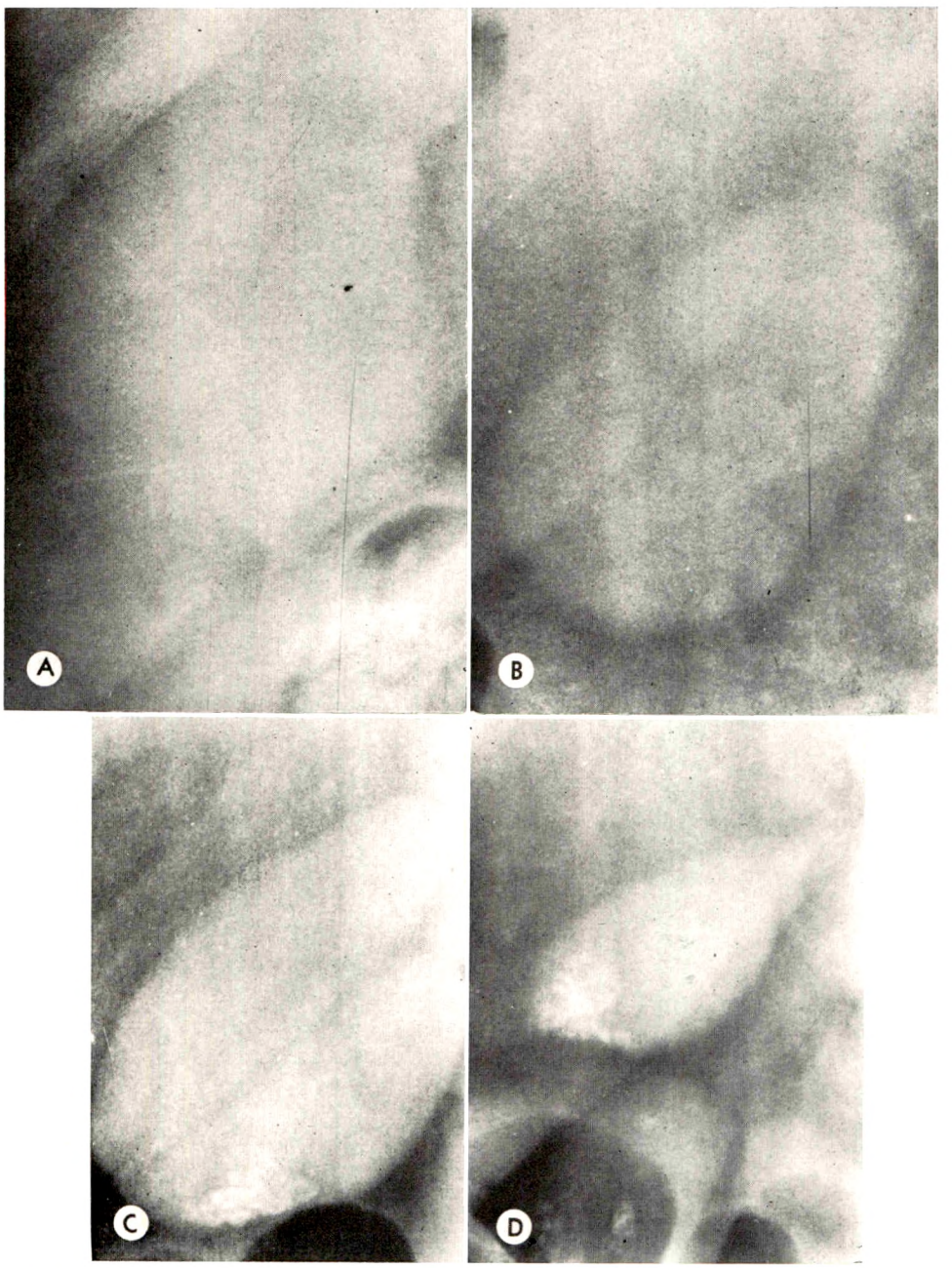


Fig. 6. Case IV. (*A*) Plain roentgenogram of the abdomen reveals no abnormal calcifications in the gallbladder area. (*B*) Routine cholecystogram shows a faintly opacified gallbladder, of homogeneous appearance, without recognizable calculi. (*C*) Following sabulography there is a collection of irregular annular opacities in the lowermost portion of the gallbladder, intermingled with opaque specks, corresponding to an admixture of sand and muddy pseudoconcretions. (*D*) Two hours after the fatty meal, the viscus was reduced in size and the abnormal content differently arranged, but the cholecystic density remained unchanged.

days later. The preliminary cholecystography showed the persistence of an opaque coating around what appeared to be 2 adjacent radiolucent calculi. When the biligrafin entered the gallbladder it was definitely demonstrated that the opaque formation lay there, and that the gallbladder contained several other rounded calculi corresponding in size to the 2 radiolucent parts of the opaque formation. The patient refused operation. One month later, plain roentgenograms of the abdomen no longer revealed the opaque coated object, neither did the gallbladder show filling by cholecystography. Theander theorized that contrast media might be less soluble in bile of pathologic composition, and might therefore be precipitated.

Salzman *et al.*[5–10] proposed the term "rim sign" to designate the telepaque deposit around biliary calculi. Patients were given a 3 to 4 day regimen of 3 grams of telepaque daily, 1 gram after each meal. A relatively fat free diet was followed during this period. Opaque impregnation on the surface of the stones in the hepatic duct was noted in 3 out of 34 cases examined by this method. Later, Watkins and Salzman[16] reported that they had obtained the rim sign in 13 out of 120 patients. Salzman named his technique the "four-day telepaque test."

Kuster *et al.*,[3] investigating Salzman's technique, called it "calculography," which is a distinctive and convenient term. This is why we decided to call our own 2 day massive telepaque test "sabulography."

Santagostini[11] devised the impregnation of radiolucent calculi with a more prolonged underdosage. A single tablet (one-tenth of the usual *per os* dosage) is given each day over a period of 8 days.

The various techniques described above are aimed at "opacifying" bile duct calculi, especially in cases with slight or moderate jaundice, in which the bile ducts are not usually visualized by intravenous cholangiography. Such demonstration of calculi in the gallbladder is exceptional and the purpose is not the opacification of the viscus itself. Moreover, the daily dosage of the contrast agent is low and given over a prolonged period. By contrast, our technique is to give a high dosage over a short period so as to increase the amount of contrast laden bile in a gallbladder that is already known to be poorly functioning, and to be able to diagnose the presence of sand. This sand can be impregnated, either partially or totally, by a cholepaque agent. A reinforcement dosage over a short period of time seems necesssary to bring enough contrast material through the cystic duct to permeate the viscous gallbladder bile, interpenetrate the eventual sand and impregnate part or all of the granules. All our operated cases showed significant stasis of the bile in the gallbladder, and sludge was frequently found in a dilated common duct. Cholecysto-cholestasis has been reported as a factor of poor or nonvisualization of the gallbladder.[17]

The rationale for the successful impregnation of gallbladder sand with a high dosage of cholepaque over a short period might be related to the concept of Berk and Lasser.[1,4] They speculated that re-absorption of opaque material through the damaged gallbladder wall could be too rapid to allow satisfactory opacification. This was corroborated by radiographic and radioisotopic studies on animals.

The sand demonstrated by sabulography was opacified throughout in some cases, and only partly in others. In some instances, chunks of cohesive sludge showed only peripheral coating. The fundamental reason for the adsorption of telepaque by in-dwelling grit or slime in the gallbladder remains undetermined. According to Salzman's work, the "opacifying" bile duct stones were always heavily pigmented or covered with biliverdin.

CONCLUSION

Sabulography is a new cholecystographic technique for the demonstration of sand and/or mud in the gallbladder by a reinforced dosage of cholepaque medium over a short period of time. It will further improve

the diagnostic accuracy of cholecystography by shedding light upon a hitherto neglected cause of poor opacification of the gallbladder. The alert radiologist, confronted by a faintly visualized gallbladder, will advise administration on the same day of an additional and increased dosage of telepaque, in order to prove or rule out the presence of sand. This is particularly indicated if the patient has a history of biliary colic, provided the renal function tests are normal. Caution should be exercised if the hepatic function is altered.

It is important to disclose the presence of this sand and/or mud in the gallbladder so that a cholecystectomy may be performed before some of it passes into and collects in the hepatic duct and causes obstruction, in which case it would be more difficult to arrive at a precise diagnosis. Moreover, a subsequent re-intervention for residual sand might thus be avoided.

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CAN IPODATE CALCIUM SAVE THE PATIENT ONE DAY IN HOSPITALIZATION?*

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THE value and accuracy of oral cholecystography is well established. Over the years various opaque media have been introduced and proven highly successful for the visualization of the gallbladder. Despite all these positive attributes one still encounters certain problems in the daily evaluation of the oral cholecystogram. One of our major problems is the poorly visualized and the nonvisualized gallbladder following the oral administration of opaque medium. The diagnosis of gallstones in the presence of a poorly visualized gallbladder can frequently not be established. A nonvisualized gallbladder is highly suggestive of pathology, particularly if it is a repeat study; however, about 10 per cent of these cases show a normal gallbladder with a repetition of the examination. It is customary to ask for a repeat oral cholecystogram in these patients which often necessitates an additional day of hospitalization for the patient.

The purpose of this paper is to present our experience with the use of ipodate calcium (oragrafin granules*) in the case of the poorly and nonvisualized gallbladder; the success or failure with this method in achieving a reduction of time required for a diagnostic oral cholecystogram. The general subject of cholecystography and the use of the various opaque media have been extensively discussed in the literature and will not be repeated here.

MATERIAL AND METHOD

For a routine oral cholecystogram the patient receives 6 tablets or 3 gm. of iopanoic acid (telepaque) at 6:00 P.M. the night before the examination in conjunction with a low fat supper. The tablets are given over a period of 10 to 15 minutes. The following morning a posteroanterior roentgenogram of the gallbladder in the left anterior oblique position is taken and also a right lateral decubitus view. All the films are checked by the radiologist who determines the further action. A fatty meal may be given if indicated. The results are classified as diagnostic or nondiagnostic. Determination of the degree of contrast medium concentration in the gallbladder has not been attempted. The nondiagnostic group comprises the poorly visualized gallbladder, in which stones cannot be excluded, and the nonvisualized gallbladder.

In the case of a nondiagnostic study, a supplementary dose of 6 gm. or 2 packs of ipodate calcium is given, circumstances permitting. The examination is repeated in the same original views after 3 hours, and, if necessary, after 6 hours. Since maximum absorption of ipodate calcium seems to take place after about 4 to 5 hours, we now take roentgenograms 4 hours after the supplementary dose of the opaque medium. Furthermore, with the absorption of ipodate calcium taking place in the upper small bowel, we encourage the patient to drink large amounts of water following the oral administration of the drug to quickly move the contrast medium out of the stomach. No specific attempts were made to demonstrate the common bile duct.

From the review of the literature it appears that all the cholecystographic agents have some degree of renal toxicity. We consider patients with known renal and liver disease as not suitable for this type of examination and do not give a supplementary dose of ipodate calcium.

RESULTS

It has been of the utmost interest to us to establish how much improvement can be

* Oragrafin granules, E. R. Squibb and Sons, Inc.

* From the Department of Radiology, Baroness Erlanger Hospital, Chattanooga, Tennessee.

expected from a supplementary dose of ipodate calcium. In the process of reviewing the results of our studies it soon became very obvious that the patients of the nondiagnostic group could be divided into 2 categories: patients with a faintly visualized gallbladder and patients with a nonvisualized gallbladder. The results of a supplementary dose of ipodate calcium in these 2 categories are remarkably different, and they are tabulated and classified accordingly. Concentration and density of the opaque medium, side effects, visualization of the common duct or other salient features of cholecystography have not been listed, since these have been reported in the literature. Our objective has been: can a nondiagnostic oral cholecystogram be converted into a diagnostic study the same day?

The nondiagnostic group comprised a total of 271 patients. Of these, 179 had a faintly visualized gallbladder shadow, 92 did not show any gallbladder shadow at all on the original studies. All of these patients received a supplementary dose of ipodate calcium (Table 1).

The category of the originally faintly visualized gallbladder comprised 179 examinations, out of which 144 could be converted into a diagnostic study the same day. Nineteen of these demonstrated gallstones. Thirty-five studies did not improve sufficiently after ipodate calcium to be called diagnostic. This represents an 80 per cent success rate (Fig. 1, *A* and *B*; and 2, *A* and *B*).

TABLE I

RESULTS FOLLOWING A SUPPLEMENTARY DOSE OF IPODATE CALCIUM

	Total	Diagnostic Study	Non-diagnostic Study
First Group	179	144	35
Second Group	92	12	80

First Group: originally faintly visualized gallbladder.
Second Group: originally nonvisualized gallbladder.

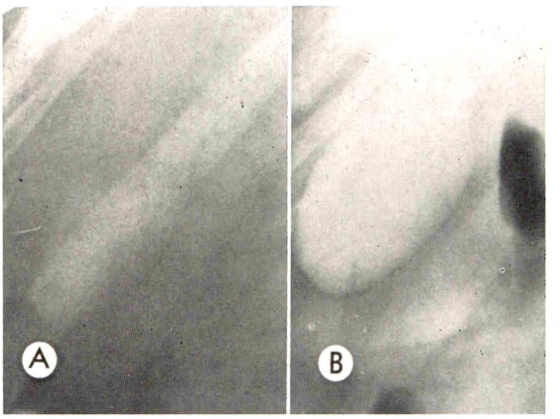


FIG. 1. (*A*) Original oral cholecystogram. Faintly visualized gallbladder. (*B*) Three hours after a supplementary dose of ipodate calcium there is a well visualized normal gallbladder.

The category of the originally nonvisualized gallbladder comprised 92 examinations. Twelve could be converted into a diagnostic study the same day. Four of these demonstrated gallstones, while 80 did not show a gallbladder shadow after a supplementary dose of ipodate calcium or improved sufficiently to be called a diagnostic study. In 7 out of 80 patients the common bile duct was visualized as being highly diagnostic of gallbladder disease. This represents a successful conversion rate of only 13 per cent (Fig. 3, *A* and *B*).

The above figures clearly indicate that ipodate calcium can very well save the patient 1 day of hospitalization if the original cholecystogram shows a faintly visualized but nondiagnostic gallbladder. However, in the case of a nonvisualized gallbladder, the use of ipodate calcium in all probability would fail to save the patient the one day in the hospital.

It is of specific interest that a number of the originally faintly visualized gallbladder studies had been done the second time with the same result. Most of these repeat studies had been made within 24 hours of the first examination. Yet in all of these cases we succeeded in completing the examination the same day through the use of a supplementary dose of ipodate calcium.

Any undue side reactions in all these

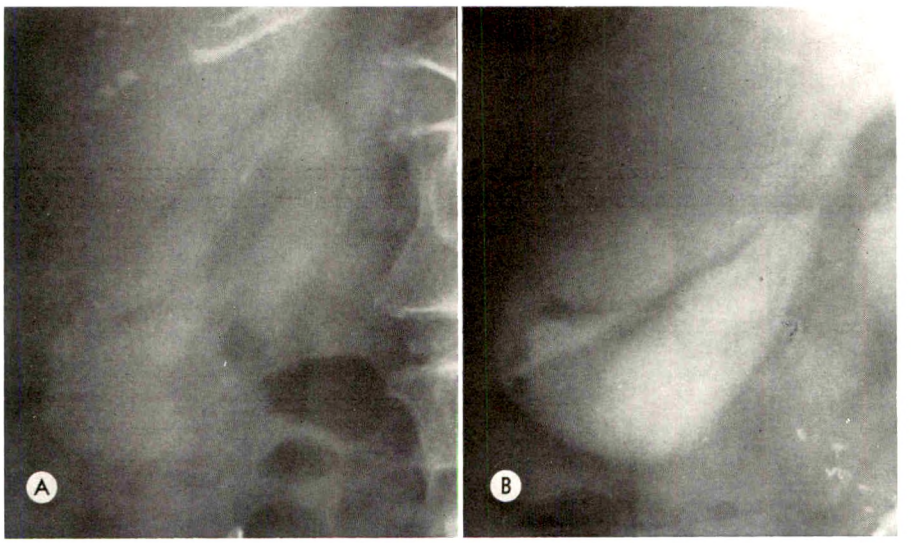


Fig. 2. (*A*) Original roentgenogram shows a faintly visualized gallbladder. Stones are suspected. (*B*) Three hours after a supplementary dose of ipodate calcium, the diagnosis of cholelithiasis is unequivocally established.

cases have not come to our attention. However, we do try to eliminate patients with known renal and liver disease, as stated above. The number of cases in which the common duct has been demonstrated is rather small. A normal common bile duct in the presence of a nonvisualized gallbladder is of very important significance and almost invariably implies gallbladder disease (Fig. 3, *A* and *B*).

DISCUSSION

For some time we have been facing the frustrating problem of the faintly visualized and nonvisualized gallbladder. Past experience has shown that in these cases

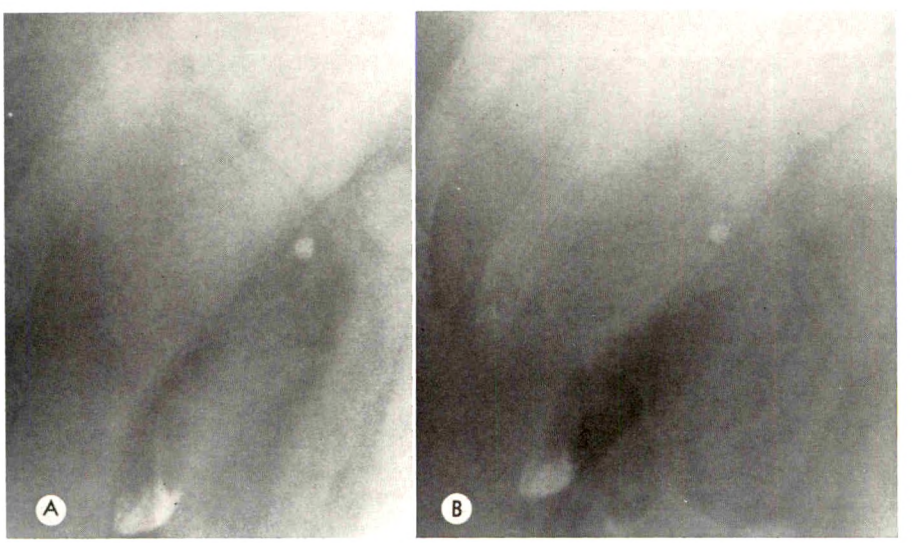


Fig. 3. (*A*) Oral choiecystogram with a nonvisualized gallbladder. Notice radiopaque calculus. (*B*) Three hours after a supplementary dose of ipodate calcium, the common duct is shown but not the gallbladder. This is diagnostic of gallbladder disease. The patient was operated upon and the calcific density shown on the roentgenogram represented a stone in the cystic duct.

gallstones are frequently overlooked or the presence of a pathologic gallbladder is greatly enhanced. We never have been able to establish exactly the cause for the poorly visualized gallbladder. In order to obtain a better diagnosis we have routinely asked for a repeat examination in these cases, requiring frequently an additional day of hospitalization for the patient. Tight surgical schedules and the patient's prolonged stay in the hospital have been in common conflict with this custom. Thus, the search for alleviating this problem has led us to the use of ipodate calcium.

The use of ipodate sodium and calcium has been discussed repeatedly in the literature.[1–8] The circumstances under which ipodate calcium can be most helpful in the daily practice of radiology have received very little attention. A supplementary dose of ipodate calcium in the patient with a faintly visualized gallbladder has proven highly successful and has an 80 per cent chance to complete the study the same day, thus saving everyone concerned some time. Apparently, the nonvisualized gallbladder is not suitable for a supplementary dose of ipodate calcium. Normally about 10 per cent of these patients show a diagnostic gallbladder shadow with a repeat study. The use of ipodate calcium increases this chance to about 13 per cent, being very little above the expected 10 per cent of success with a repeat study.

We find that ipodate calcium is a valuable adjunct in oral cholecystography and has helped us in many cases to change a poor study into a diagnostic examination on the same day.

SUMMARY

Ipodate calcium can save the patient 1 day of hospitalization if the original cholecystogram shows a faintly visualized gallbladder shadow. The use of a supplementary dose of ipodate calcium has an 80 per cent chance to turn the examination into a diagnostic study the same day.

If the original roentgenograms did not show any gallbladder shadow at all, then ipodate calcium has only a 13 per cent chance to convert the study into a diagnostic one on the same day.

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Part of the ipodate calcium (oragrafin granules) was kindly supplied by E. R. Squibb and Sons, Inc.

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THE CONSECUTIVE DOSE PHENOMENON IN ORAL CHOLECYSTOGRAPHY*

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IN 1964 Lasser and the author became interested in the clinical situation in which the second dose cholecystogram results in excellent visualization of apparently normal gallbladders when the first dose examination shows nonvisualization or faint visualization[1] (Fig. 1, *A* and *B*). The amount of contrast material in the gallbladder on the second day as judged by the roentgenographic density of the organ is more than twice the amount present on the first examination. Some mechanism exists whereby a higher proportion of the second dose is retained in the gallbladder in comparison to the first. This situation is termed the consecutive dose phenomenon.

In order to determine the relevancy of several pharmacodynamic and technical mechanisms which could explain this phenomenon, a series of patients with this clinical situation were studied to ascertain the incidence and the reproducibility of the phenomenon. The results of this investigation and their implications are reported here.

METHOD

From 1964 to 1967, 2,382 patients had cholecystography at the North Hills Passavant Hospital in Pittsburgh, Pennsylvania. Patients routinely were given 3.0 gm. of telepaque* the evening before the examination after a low fat supper. Afterwards, nothing was allowed by mouth until the completion of the roentgenographic examination the following morning. Three hundred and ninety-six patients (17 per cent) had faint or no visualization of the gallbladder on the initial cholecystogram. A repeat examination on the following day with a second dose of 3.0 gm. of telepaque was required for a definite diagnosis. Faint visualization was defined as concentration insufficient to permit fluoroscopic and spot roentgenographic identification of the gallbladder. The cholecystograms in these patients were reviewed to determine which patients demonstrated the consecutive dose phenomenon. The degree of gallbladder opacification on the initial and the second examination was classified as nonvisualization, faint, moderate and dense visualization. Those patients whose gallbladders changed from nonvisualization to moderate or dense visualization or from faint opacification to dense opacification without cholelithiasis were considered to have the consecutive dose phenomenon. Concentration on the second cholecystogram appeared to be more than double that of the first examination.

All patients with the consecutive dose phenomenon were contacted by mail and asked to return for follow-up cholecystography. The interval between the first cholecystogram and the follow-up investigation varied from 1 to 4 years. The follow-up cholecystograms were obtained using the same technique as the initial examinations.

RESULTS

Of the 396 patients who had faint or nonvisualization of the gallbladder on the initial cholecystogram, 198 (50 per cent) showed no improvement in concentration on the second study; 79 (20 per cent) showed moderate improvement; and 119 (30 per cent) showed marked improvement (Fig. 2). Hence, 30 per cent of the initial nonvisualizers or faint visualizers demonstrated the consecutive dose phenomenon. Considering those 160 patients whose gallbladders faintly visualized on the initial

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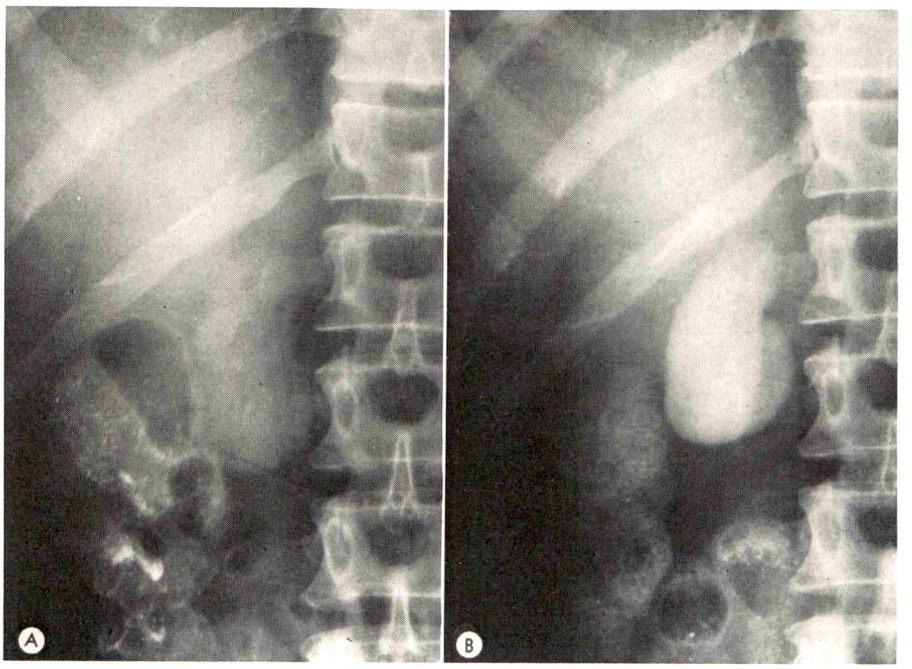


Fig. 1. The consecutive dose phenomenon. (*A*) Faint visualization of the gallbladder after a single dose of telepaque. (*B*) Marked improvement in visualization of the gallbladder on the following day after a second dose of telepaque. The opacification appears to be more than double that of the first examination.

cholecystogram, 32 (20 per cent) showed no improvement on the second examination; 48 (30 per cent) showed moderate improvement; and 80 (50 per cent) showed marked improvement (Fig. 3). Of those with nonvisualization in the initial cholecystogram, 177 (75 per cent) showed no increase in opacification; 24 (10 per cent) showed moderate increase; and 35 (15 per cent) showed marked increase (Fig. 4). Hence, the consecutive dose phenomenon is more frequent in patients with initial faint visualization (50 per cent) than in those with initial nonvisualization (15 per cent).

Of the 119 patients who demonstrated the consecutive dose phenomenon, 38 agreed to return for follow-up evaluation. The follow-up cholecystogram in these patients showed good initial opacification of the gallbladder in 33 cases (86 per cent) (Fig. 5). Two of these had cholelithiasis which was not present earlier. A second dose of telepaque in the other 5 patients again resulted in the consecutive dose phenomenon. Hence, the phenomenon is not reproducible in the majority of cases.

DISCUSSION

A series of physiochemical and biochemical interactions must proceed normally in order for telepaque to accumulate in gallbladder in sufficient concentration to be visible roentgenographically. Because telepaque is lipid soluble, it is capable of dissolving in the lipid boundary of the intestinal epithelium and is absorbed from the intestine by the process of diffusion through the cellular membrane.[6] It is then temporarily stored in widespread body depots which include fat, plasma albumen, liver and muscle.[3] Enzymes are present in the liver which permit lipid soluble drugs and other materials in food to be eliminated from the body by converting them to water soluble compounds. These enzymes are located in the endoplasmic reticulum of the liver cells which are disrupted by homogenization to form microsomes.[4] Telepaque is converted to the water soluble compound, telepaque glucuronide, by esterification by the microsomal enzymes.[8] The transport mechanisms for the transfer of telepaque from the blood

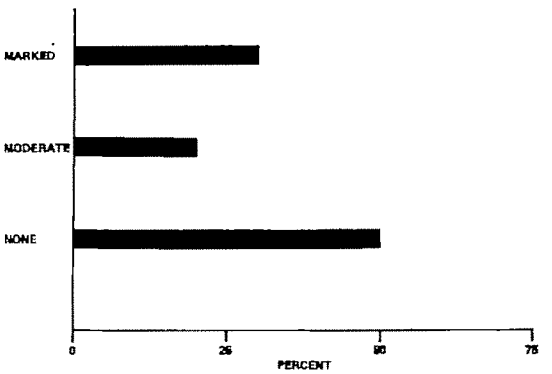


FIG. 2. Improvement of second dose visualization. First dose nonvisualization or faint visualization (396 patients).

to the cytoplasm of the hepatic cells and from the cytoplasm across the cell membrane to the bile canaliculus are unknown. Because telepaque glucuronide is water soluble, it is not reabsorbed when it reaches the gallbladder, but remains there to be concentrated with the bile as water is reabsorbed. Final elimination from the body occurs when humoral and neural stimuli cause the gallbladder to contract and the telepaque glucuronide is emptied into the intestine. Being water soluble, reabsorption of a significant amount of telepaque glucuronide does not occur.

In an attempt to explain the pharmacodynamics of the consecutive dose phenomenon, Lasser and the author postulated that reabsorption of the contrast material through a marginally inflamed gallbladder wall prevents dense opacification on the initial examination.[1] This reabsorption is diminished during the second examination because binding sites in the blood have been saturated by the first dose of telepaque. Consequently, more contrast material is retained in the gallbladder and visualization after the second dose is more than twice as dense as after the first. Reabsorption of telepaque from inflamed gallbladders was, in fact, demonstrated *in vivo* in dogs, and in humans *in vitro*.[1] Unless the postulated gallbladder inflammation is transient or intermittent, the consecutive dose phenomenon would occur each time the patient had a cholecystogram. At least some of these patients could be expected to develop overt gallbladder disease subsequently. In the series of patients described here with follow-up examinations done 1 to 4 years later, the gallbladder remained normal in 36 of 38 patients. Thirty-three of the 38 patients did not have repetition of the consecutive dose phenomenon on the follow-up cholecystogram.

Other pharmacodynamic explanations can be postulated. Hereditary excess of binding sites in fat, muscle, liver and blood could limit the amount of free contrast material available to the liver for excretion.[3] A higher proportion of the second dose of telepaque would reach the bile because the binding sites are saturated by the first dose. This, too, would result in a reproducible situation. Other possibilities concern the microsomal enzymes of the liver. Certain drugs, when taken together with the first dose of telepaque, could compete with the contrast material for the enzymes, thus limiting the rate of excretion of the contrast material.[5] If the drug were not taken along with the second dose, there would be no interference and the telepaque would be excreted in greater proportion on the second examination. This is unlikely to be reproducible on follow-up examination.

Drug induction of enzymatic capability may play a role.[9] The microsomal enzymes could be stimulated by the first dose of

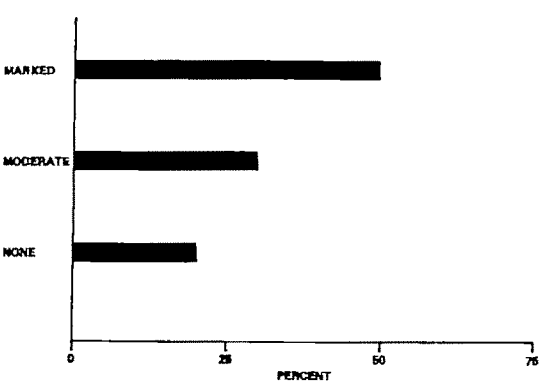


FIG. 3. Improvement of second dose visualization. First dose faint visualization (160 patients).

telepaque, so that the second is metabolized more efficiently and excreted in higher concentration in the bile. While this would be compatible with the fact that there is usually no repetition of the consecutive dose phenomenon, drug induction usually requires high doses over long periods of time.[9] It is unlikely that it could occur after a single dose of telepaque.

Loss of some of the first dose of telepaque from vomiting and diarrhea or failure to ingest a part of the first dose could explain impaired initial visualization of the gallbladder. This might not be reproducible on follow-up examination. However, these factors would very likely involve the second dose in the same proportion. There is no clinical evidence in this series that these factors occur with greater frequency in patients with the consecutive dose phenomenon.

Technical factors involving the roentgenographic exposure could produce an apparent variation in density stimulating a difference in gallbladder concentration. However, the magnitude of the difference on the two examinations appears to be more than can be explained on a technical basis alone.

Another consideration is the possibility of delayed absorption of the first dose of telepaque from the small intestine. Roentgenograms of the abdomen made during the initial examination in this series show no excess of the contrast material in the intestine and no explanation for such delay in absorption is apparent. Reabsorption of conjugated telepaque from the intestine could supplement the second dose of telepaque and account for the consecutive dose phenomenon. However, conjugated telepaque is water soluble and only poorly absorbed from the intestine. Furthermore this does not account for the poor visualization initially. Hydrolysis of the conjugated telepaque with subsequent reabsorption of the lipid soluble form is a theoretic possibility, but again this does not explain the failure to visualize the gallbladder properly on the first examination.

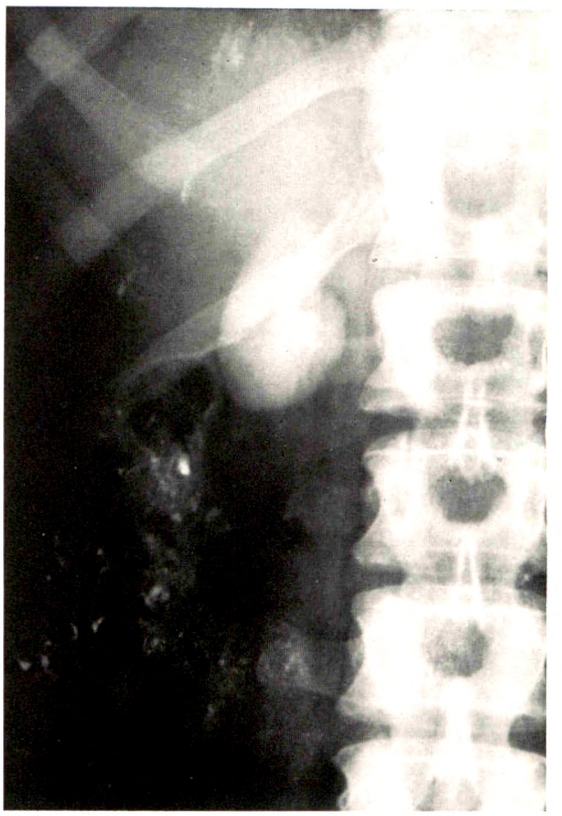


Fig. 5. Follow-up cholecystogram done 3 years later on the same patient as in Figure 1. There is excellent opacification of the gallbladder with only a single dose of telepaque.

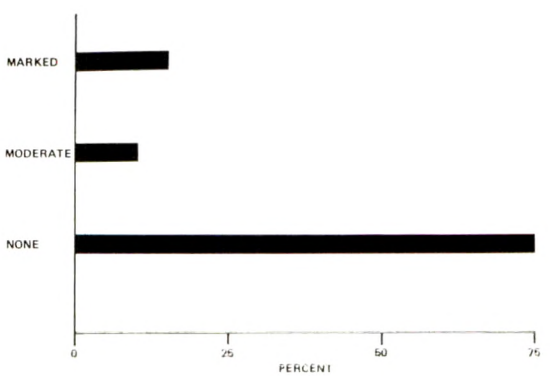


Fig. 4. Improvement of second dose visualization. First dose nonvisualization (236 patients).

The relationship of the two cholecystograms of the consecutive dose phenomenon to each other in producing the phenomenon is unclear. Since the situation is usually not reproducible, it is possible that the second

dose produces the same degree of opacification it would if it were an isolated examination not preceded by the first cholecystography. The phenomenon would then be caused solely by failure of the first dose to reach the gallbladder in normal concentration. If the gallbladder were flaccid and distended with bile due to physiologic stasis prior to the first examination, telepaque could not accumulate in the gallbladder in normal concentration and poor first dose visualization would result.[2] Telepaque is a relatively potent cholinesterase inhibitor and produces tonic contraction of the gallbladder.[7] The contraction could empty the gallbladder enough to relieve the physiologic stasis. The second dose of telepaque would be able to enter the gallbladder properly and normal visualization would result. No high fat meal was given prior to any of the gallbladder examinations. However, no decrease in size of the gallbladder was visible roentgenographically on the consecutive cholecystograms in these cases.

CONCLUSION

The consecutive dose phenomenon in oral cholecystography is that situation in which a second dose of telepaque results in excellent visualization of apparently normal gallbladders when the first dose examination shows nonvisualization or poor visualization. This occurs in 30 per cent of patients who have nonvisualization or faint visualization on the initial examination. It is more common when there is faint visualization of the gallbladder initially than when there is nonvisualization on the first examination.

Thirty-eight patients with this phenomenon were studied by follow-up cholecystograms 1 to 4 years after the initial examination. The phenomenon was not reproducible in the majority of patients. Thirty-three of 38 patients opacified their gallbladders normally with a single dose of telepaque on the follow-up study.

Failure to reproduce the situation and other factors make several postulated pharmacodynamic and technical explanations unlikely. The actual mechanism of the phenomenon remains uncertain.

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INTRAVENOUS CHOLANGIOGRAPHY WITH INTACT GALLBLADDER*

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IN RECENT papers[2–4,6–8] the usefulness of the intravenous cholangiogram has been indicated in the diagnosis of acute cholecystitis and in further evaluation of the gallbladder after it appears to be nonfunctioning as depicted in an oral cholecystogram. These indications for the use of intravenous cholangiography make it imperative to have a working knowledge of factors influencing opacification of the gallbladder during this examination.

It is the purpose of this study to determine the frequency and significance of opacification or nonopacification of the gallbladder during intravenous cholangiography and to evaluate the usefulness of the procedure in detecting pathologic states of the gallbladder, as well as to evaluate the accuracy of detection of pathologic states of the extrahepatic biliary ducts when the gallbladder is present.

MATERIAL AND METHOD

This study is based on records of 133 patients who had not undergone cholecystectomy and for whom at least one intravenous cholangiogram had been made. Included in the study were all patients undergoing intravenous cholangiography between 1956 and 1961, as well as random patients, up to 1967, whose condition met the criteria we set up. The group included 60 females and 73 males, with an average age of 56 years (range 10 to 90 years). Abdominal operations subsequently were performed in 57 of the 133 cases.

The cholangiograms were obtained after the intravenous administration of 20 ml. of a 26 per cent solution of iodipamide (cholografin) over a 5 to 10 minute period. All patients received premedication in the form of 10 mg. of chlorpheniramine maleate (chlor-trimeton) injected intramuscularly. The film sequence included: (1) preliminary scout roentgenogram; (2) anteroposterior roentgenograms and tomograms for all patients, 25 minutes after completion of injection; and (3) delayed anteroposterior roentgenograms and tomograms for most patients. The period of observation was terminated at 25 minutes in 44 cases, at 50 minutes in 37 cases, at 90 minutes in 41 cases, and after 90 minutes in 11 cases. The longest examination period was 2 hours and 25 minutes.

RESULTS

Three overlapping arrangements of the group were studied: (1) the entire group; (2) those who underwent abdominal operations; and (3) those for whom cholecystograms had been made previously, after the oral administration of contrast medium.

I. ENTIRE GROUP

The gallbladder was visualized during intravenous cholangiography in 88 of the 133 cases (66 per cent). Initial visualization of the gallbladder occurred within 25 minutes in 70 of these 88 cases (80 per cent). The longest interval before visualization was 135 minutes. The over-all average time for initial visualization was 33 minutes; 44 of the 133 examinations were terminated at 25 minutes. The time of maximal opacification of the gallbladder could not be evaluated in this study because the maximal opacification observed occurred on the final roentgenograms in all but 8 cases.

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The biliary ducts were visualized in 121 cases (91 per cent), the initial visualization occurring at 25 minutes in 115. The longest interval before initial visualization of the ducts was 50 minutes. No attempt was made to study the time-density relationship for the biliary ducts.

Since the flow of fluid in any system is always in the direction of least resistance, the relative "resistance" of the cystic duct compared to that of the sphincter of Oddi undoubtedly determines whether or not there will be filling of the gallbladder, assuming that the term "resistance" includes the factor of pressure within the gallbladder and duodenum. One might expect to see less opacification of the gallbladder when the contrast medium flows copiously into the duodenum and *vice versa*. However, in many cases there was a considerable amount of contrast medium in the duodenum and satisfactory opacification of the gallbladder, and in many there was little contrast medium in the intestine and poor filling of the gallbladder (Table 1).

Iodipamide is excreted through the biliary system and, to a lesser extent, the urinary system. Because the right kidney is included in the area of the abdomen that is filmed, the presence of contrast medium in the calyceal system might signal poor visualization of the biliary ducts; yet, no evidence was found during the study to indicate that the quality of biliary tract visualization was in any way related to the presence or absence of contrast medium in the kidney.

Table I

Intravenous Cholangiography: Relationship of Amount of Contrast Medium in Duodenum to Opacification of Gallbladder

Contrast Medium in Duodenum, Relative Amount, Grade	Opacification			No. of Cases, Total
	Poor	Fair	Good	
0	1	2	10	13
1	5	7	11	23
2	7	2	13	22
3	3	5	3	11
4	4	4	11	19
Total	20	20	48	88

2. SURGICAL GROUP

The 57 patients for whom abdominal operations were performed were classified into 3 separate subgroups according to the structures that were visualized in the intravenous cholangiogram: (a) gallbladder and biliary ducts, 31 cases; (b) biliary ducts, 16 cases; and (c) no visualization of ducts or gallbladder, 10 cases.

(a) The 31 patients in whom both the gallbladder and biliary ducts were visualized were further subdivided according to the degree of opacification of the gallbladder; the amount of time elapsed seemed to have very little relationship to the density of the shadow of the gallbladder (Table II). Delay in visualization of the gallbladder seemed to have no relationship to the presence of stones in that structure. Among 17 patients who had cholelithiasis, the gallbladder opacified within 25 minutes in 11 (65 per cent), within 45 minutes in 2, 50 minutes in 3, and 60 minutes in 1. On the other hand, when no stones were found in the gallbladder or common bile duct at operation, opacification had ensued in 6 of 11 patients within 25 minutes (55 per cent) and in 1 each within 45, 60, 75, 120 and 135 minutes. In this group there were 17 instances of surgically proved cholelithiasis, in 6 of which the diagnosis was made correctly by intravenous cholangiography. Among the 11 other cases, opacification of

Table II

Intravenous Cholangiography: Opacification of Gallbladder According to Duration of Examination

Opacification	No. of Cases	Examination, Maximal Time (min.)	Mean Time of Last Roentgenogram (min.)
Poor	12	120	66
Fair	8	135	59
Good	11	75	53

the gallbladder was poor in 8. Of the 12 cases of choledocholithiasis, in all of which visualization of the ducts was satisfactory, the condition was correctly diagnosed by intravenous cholangiography in 6.

(b) In the 16 cases of surgical exploration in which only the biliary ducts had been visualized roentgenographically, complete mechanical obstruction of the cystic duct was found in 5; in these 5, impacted stones were noted in 4 and acute cholecystitis in the other. In the remaining 11 cases in which obstruction of the cystic duct was not found at the time of operation, the gallbladder was completely normal in 2, there was some evidence of subsiding cholecystitis in 1, and in 8 stones were found in the gallbladder without impaction in the cystic duct. Thus, of 16 patients in whom the gallbladder did not opacify at cholangiography, 14 had definite disease in the gallbladder, although in only 5 was a clear mechanical explanation found at the time of operation for nonfilling of the gallbladder.[5–8]

(c) Of the 10 surgical patients in whom neither the biliary ducts nor the gallbladders were visualized, results of tests of hepatic function were abnormal in all, which probably accounts for nonvisualization.[1] The average value for bilirubin was 2.02 mg. per 100 ml. of serum, direct, and 0.71 mg., indirect, and the average value for alkaline phosphatase was 45.1 units per liter. At operation 1 patient was found to have stones in both the gallbladder and biliary duct, 4 had stones in the gallbladder only and 5 had no stones.

3. GROUP IN WHICH ORAL CHOLECYSTOGRAM HAD BEEN MADE PREVIOUSLY

Oral cholecystograms had been made for 109 of the 133 patients; in 90 this examination had been performed less than a month prior to the making of the cholangiogram by the intravenous procedure used in this study. Members of this group were divided according to the result in the report of the previous oral cholecystogram (Table III).

Twenty-two patients had surgically proved cholelithiasis in the course of which both oral and intravenous cholecystography had been done. The gallbladder opacified in the oral cholecystogram in 9 of these and in the intravenous cholangiogram in 6. Of the 6 who had gallbladders which opacified during intravenous cholangiography, 4 were found to have stones. In 2 of these 4 oral cholecystography gave normal results; in 1 there was nonvisualization; and in 1 the cholecystogram depicted a functioning gallbladder with stones. The oral cholecystograms of 5 of 22 patients yielded normal results.

DISCUSSION

The filling of the gallbladder with intravenously administered contrast medium during cholangiography is a complex phenomenon. The patency of the cystic duct and the resistance to flow into the gallbladder relative to the resistance to the flow of contrast medium into the duodenum should play the major part in control of opacification of the gallbladder. The variability of the relative "resistance" of the cystic duct was demonstrated by: absence of filling of the gallbladder in 16 of the 73 cases (22 per cent) in which previous studies with the oral technique had demonstrated a functioning gallbladder and hence a patent cystic duct; and variations of the initial time of visualization of the gallbladder in the intravenous cholangiogram between 25 and 135 minutes. Delay in visualization of the gallbladder beyond 25 minutes but within 135 minutes did not indicate a greater likelihood of the presence of stones in the gallbladder.

Among the 57 surgical patients 10 exhibited no evidence of physical obstruction of the cystic duct at the time of surgical and pathologic examination despite the fact that the gallbladder could not be visualized at the time of intravenous cholangiography. Nonfilling of the gallbladder in these cases must be attributed either to functional resistance to flow through the cystic duct or to intermittent obstruction

TABLE III

COMPARATIVE RESULTS: ORAL *vs.* INTRAVENOUS CHOLANGIOGRAPHY

Recent Oral Cholecystogram Depicted	Comparison*			Visualization in Intravenous Cholangiogram (over-all per cent)	Mean Time of Last Roentgenogram (min.)
	Surgical	Nonsurgical	Over-all		
Normal	9/12	39/49	48/61	79	50
Functioning with stones	5/7	4/5	9/12	75	42
Nonfunctioning	9/21	7/15	16/36	44	66

* Comparison shown as: Number in which gallbladder was visualized/Number in which oral cholecystography was done. in intravenous cholangiogram

caused by stones. The data indicate that in only a minority of instances of nonfilling of the gallbladder during intravenous cholangiography is there an impacted stone or edema obstructing the cystic duct, but that in an overwhelming majority the gallbladder is diseased, and that cholelithiasis usually is present (88 per cent).

The presence of free stones in the gallbladder should have little effect on opacification of the gallbladder during intravenous cholangiography because the concentrating function of the gallbladder is not involved in this process. This assumption appears to be supported by our data because, among the patients in whom a functioning gallbladder was depicted during oral cholecystography, the presence or absence of stones visualized in the study done with orally administered contrast medium did not influence the incidence of opacification of the gallbladder during intravenous cholangiography. Still, stones found to be free at cholecystectomy could, at the time of roentgen examination, have been causing obstruction, and the number of cases involved was small.

Cholelithiasis was diagnosed on the basis of the intravenous cholangiogram in only 35 per cent of the cases in which the gallbladder had been visualized and stones subsequently had been demonstrated surgically. The proportion increases to 67 per cent if instances of poor visualization of the gallbladder are excluded. Yet, even in the face of this relatively low rate of accuracy, the intravenous cholangiogram may be helpful in demonstrating stones in the gallbladder in some cases, despite the fact that oral cholecystography done previously may have given normal results.

Stones were detected in the biliary duct in 9 of 19 cases (47 per cent) in which they were proved to be present. The percentage was disappointing, especially in the face of the most meticulous attention to technique, enhancement of opacification and the use of serial tomograms through the level of the ducts.[9,10]

SUMMARY

Results of intravenous cholangiography as carried out for 133 patients who had not undergone cholecystectomy were studied. Opacity of the gallbladder was obtained in 88 cases.

Opacification of the bile duct without visualization of the gallbladder was found to be caused by complete mechanical obstruction of the cystic duct by stone or edema in about a third of the patients for whom surgical exploration subsequently was done. Of the remaining patients, most were found to have free gallstones at operation, but in about an eighth of the cases there was no anatomic reason for nonfilling of the gallbladder.

Delay in opacification of the gallbladder beyond 25 minutes, but within 135 minutes, did not seem to be influenced by the presence of stones in the gallbladder. Delay occurred in 35 per cent of those patients who had stones and in 45 per cent of those who did not.

Cholelithiasis later proved surgically was detected by intravenous cholangiography in 6 of 11 cases (55 per cent) in which the gallbladder was visualized. Choledocholithiasis was detected in 9 of 19 cases (47 per cent) in which the condition subsequently was proved at operation. In some cases, however, gallstones not demonstrated by oral cholecystography were disclosed by the intravenous examination.

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CHOLECYSTOKININ CHOLECYSTOGRAPHY*

A FOUR YEAR EVALUATION

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A PROVOCATIVE subject in medicine has been whether or not a gallbladder, normal on routine oral cholecystography, can be pathologic and symptomatic. Occasional emergency cases admitted with acute upper abdominal pain and fever but with normal preoperative oral cholecystography reveal gross and microscopic evidence of acute cholecystitis. Recent experience with 2 such cases, both in the same week, confirms the fact that the pathologic gallbladder can appear roentgenographically normal at times.

This is also true, however, in the absence of acute onset and in the absence of fever. Many of these patients, who remain "miserable" with chronic recurring postprandial pain and distress, are often labelled "neurotic," or operated mistakenly for "hiatus hernia" or for "duodenal ulcer" without relief.

When the routine oral cholecystogram is normal, the surgeon is loath to perform cholecystectomy because of the high rate of persistence or recurrence of symptoms. Even at abdominal exploration, the pathologic gallbladder may appear perfectly normal, and the surgeon often closes the abdomen without a diagnosis; the neurotic label has been "confirmed!" Or do such patients become neurotic because of their symptoms and their inability to obtain relief or even a diagnosis?

The difficulty lies, first, in demonstrating that an acalculous gallbladder is pathologic and second, in differentiating other conditions which may simulate biliary pathology. Cholecystokinin cholecystography (CCK-GB) has provided a means of selecting those patients with biliary pathology, so that proper treatment may be instituted.

The results of a 3 year study, presented December, 1968, to the Radiological Society of North America, indicated that all except 1 of 37 patients were improved by cholecystectomy in a 3 to 36 month follow-up.[12] These results were very encouraging.

One of the discoverers of the cholecystokinin (CCK), Doctor A. C. Ivy, approached by one of his students with a successful experiment on 50 rats is quoted as having said, "Come back and discuss this with me after you have investigated 50 more rats." This is one reason for this paper: to obtain confirmation.

MATERIAL

Following Doctor Ivy's admonition, 92 more controls and 158 additional patients were studied by CCK-GB which was administered intravenously in place of the fatty meal during oral cholecystography. Only 39 patients had cholecystectomy and these, along with the 103 patients from the initial study[12] were subjected to a complete analysis, making a total of 141 patients and 142 normal controls (Table IV) included in the present evaluation.

The 142 normal controls were chosen in a fashion meant to eliminate those with possible gallbladder pathology. All were males between 21 and 35 years of age with no history of gastrointestinal complaints. No women, Indians, or Mexicans were permitted in the study because of the high incidence of gallbladder pathology in these groups. Thirty-five of the normal controls were doctors.

Although our findings and conclusions were essentially the same as those in the

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previous article,[12] some important modifications in utilization of the findings are necessary for correct interpretation.

METHOD

Patients accepted for investigation were required to have a history strongly suggestive of gallbladder pathology and a recent normal oral cholecystogram. The patient is prepared with a double dose of opaque medium* preceded earlier in the day by a fatty lunch. Roentgenograms of the gallbladder 12 hours later are made with the patient in the right prone oblique position rotated about 15 to 35° with sharp coning. Very large or very obese patients are roentgenographed in the prone position with the right side elevated 15 to 35° and with a sponge placed for right upper quadrant compression. Each patient receives 75 Ivy dog units of cholecystokinin† intravenously in approximately 15 to 30 seconds. The cholecystograms are made at 45 seconds (when almost all gallbladders are beginning to contract) and then at 3, 5, 10 and 15 minutes after the injection. It is particularly important that *no change in the patient's position* be allowed, since this will affect the gallbladder configuration and size.

The patient is encouraged to describe all symptoms experienced after the CCK injection. At the same time, he should not be misled by anyone, including the referring physician, into "experiencing" certain symptoms. If the patient is told that he may develop right upper quadrant pain, it is possible for him to imagine this.

RESULTS

The gallbladder contracts and the sphincter of Oddi relaxes when cholecystokinin, a hormone in the walls of the duodenum and proximal jejunum, is stimulated by certain food substances introduced into the duodenum.

After intravenous injections of 75 Ivy dog units, the gallbladder contraction usually takes place much more quickly and vigorously than after the fatty meal and information, usually not obtained by the fatty meal, becomes available.

Tables I and II summarize the results of the CCK-GB examination obtained from the control group and from the patient group. The most significant findings will be discussed.

SYMPTOMS INDUCED BY CHOLECYSTOKININ

The most common symptom caused by intravenous cholecystokinin injected within 15 to 30 seconds is cramping in the lower and mid abdomen due to bowel contractions. Nausea is not infrequent, usually is mild, occurs within 30 to 60 seconds, and is usually transient lasting only 1 to 3 minutes.

Only 3 of the 107 non-doctor controls had mild (more than transitory) to moderate epigastric cramping while 6 of the doctor controls had similar cramping.

Right upper quadrant or epigastric pain is rare in normals. Following the intravenous injection of 75 Ivy dog units of cholecystokinin, right upper quadrant or epigastric pain was found in none of the 107 non-doctor controls (Table II). Of the 35 doctor controls 1 had epigastric and 1 had right upper quadrant pain. There was no correlation of this pain or cramping with the speed of gallbladder evacuation.

The disproportionate number of doctors who developed symptoms suggests that their response to the CCK may have been affected by their medical knowledge. When utilizing this information for the evaluation of biliary tract pathology, the results of the doctor group probably should be discarded except when the patient is a doctor or anyone who "knows" what symptoms to expect from CCK-GB.

Right upper quadrant pain and epigastric pain have been found to be extremely important as signs of gallbladder pathology

* Twelve cholegrafin capsules. The cholegrafin was donated by E. R. Squibb and Sons, New York.

† Refined cholecystokinin (CCK), now being produced by Jorpes and Mutt[7] in Sweden, is under control of the Federal Drug Administration and is still available only for investigative purposes, although there have been no serious reactions reported in over one-quarter million intravenous injections.

TABLE I

ROENTGENOGRAPHIC FINDINGS AFTER INTRAVENOUS CHOLECYSTOKININ

Spasm Visualization	Roentgenographic findings					
	Controls				Patients	
	Doctors (35)		Non-Doctors (107)		(141)	
	(No.)	(Per Cent)	(No.)	(Per Cent)	(No.)	(Per Cent)
Fundus	1	3.0	0	0.0	15	11
Body	0	0.0	0	0.0	4	3.0
Neck and Infundibulum*	1	3.0	2	2.0	77	55
Cystic Duct	0	0.0	1	1.0	33	23
Cystic Duct	28	80	94	88	118	84
Common Bile Duct	23	70	85	79	114	80
Hepatic Duct Reflux: One Plus	1	3.0	0	0.0	3	2.0

* Persisting on 5 minute cholangiogram or beyond.

after intravenous CCK (Table II). There were only 2 controls (1.4 per cent) who complained of right upper quadrant and/or epigastric pain which, to reiterate, occurred only in the 35 doctor controls and not in the 107 non-doctor controls. This contrasts sharply with the 65 (46 per cent) patients who developed right upper quadrant and/or epigastric pain. On the other hand, epigastric and right upper quadrant *cramping* is considered important only when this reduplicates the patient's symptoms, since 11 (8 per cent) of the controls complained of this symptom as compared to 16 (11 per cent) of the patients.

Seven patients appeared normal roentgenographically but developed epigastric or right upper quadrant pain and/or reduplicated symptoms after cholecystokinin; chronic cholecystitis was found at surgery. Nine patients experienced no significant symptoms after the CCK injection and of these 6 had positive and 3 had negative roentgenographic findings. All proved to have chronic cholecystitis. There was another case which developed no symptoms and was roentgenographically normal, but several months later became roentgenographically positive and reduplicated his symptoms, which suggests progression.

TABLE II

SYMPTOM RESPONSE TO INTRAVENOUS CHOLECYSTOKININ

Symptoms	Controls				Patients	
	Doctors (35)		Non-Doctors (107)		(141)	
	(No.)	(Per Cent)	(No.)	(Per Cent)	(No.)	(Per Cent)
Nausea	8	23	6	6.0	13	9.0
Right Upper Quadrant and Epigastric Cramps	6	17	5	5.0	16	11
Right Upper Quadrant and Epigastric Pain	3	9	0	0.0	63	46
Reproduction of Symptoms	—	—	—	—	25*	66

* Of 38 analyzed.

ROENTGENOGRAPHIC FINDING

Absent or minimal gallbladder evacuation and complete or marked evacuation in 10 to 15 minutes are not significant. The speed of contraction neither influenced the common bile duct size nor the hepatic duct reflux in the normal controls.

Of the 111 controls in whom the common bile duct was visualized (approximately 80 per cent of the total) there were only 6 who showed a maximum common duct size of 6 mm. or larger and there were only 2 of these who exhibited the maximum size of 7 mm. (Table III). Mild reflux of opaque contrast medium into the common hepatic duct was found in several controls but there was only one who showed a moderate degree of reflux (Table 1). None had pancreatic duct reflux.

Gallbladder "spasm" is a term used in this study to describe a disproportionately greater contraction in a localized area (fundus, body, infundibulum, and neck). Spasms varied from absent to marked and are classified from 0 to +++.

Positive roentgenographic findings include spasm at the neck of the gallbladder, at the infundibulum, at the body or at the fundus. Spasm of the cystic duct is another positive finding but this is more difficult to demonstrate since there is usually associated neck spasm which prevents the filling of the cystic duct. The most common site of spasm is at the neck of the gallbladder where the greatest accumulation of muscle in the biliary tract is found. When 75 Ivy units of CCK are injected intravenously in less than 30 seconds, spasm of the neck and infundibulum can occur normally,[15] but in our experience it usually does not last more than 3 or 4 minutes. There was 1 control who had typical neck spasm; 2 others had moderate spasm of the neck and infundibulum lasting 5 minutes (Table 1). By comparison, 77 of 141 patients (54.6 per cent) had spasm of the neck and/or infundibulum.

One of the 3 controls (less than 1 per cent) had mild spasm of the tip of the fundus and another had spasm of the cystic duct, while 15 (11 per cent) patients had fundus spasm and 33 (23 per cent) of the patients had cystic duct spasm. None of the controls had spasm of the body; this was found in 4 (3 per cent) of the patients.

From these results it can be seen that spasm is a sign of a pathologic gallbladder. Spasms of the neck and infundibulum of the gallbladder are rare 5 minutes or more after the intravenous injection of CCK in normals and, if they persist through the 5 minute cholangiogram, are a reliable sign of gallbladder abnormality.

Other positive roentgenographic signs are dilatation of the common bile duct, persistence of the opaque medium in the duct, and marked hepatic duct reflux; these are signs of stenosis at the distal common duct. Mild reflux is not unusual even on routine oral cholecystography after the fatty meal or after CCK. On the other hand, moderate hepatic duct reflux occurred in only 1 of the 142 controls and there were none with marked hepatic duct reflux.

Among the 142 controls, there were no common bile ducts above 7 mm. in diameter (Table III) and there were only 2 that were larger than 6 mm. By contrast, 27 patients had common bile ducts more than 6 mm. in diameter; 18 of these were 7 mm. in diameter and 4 were 8 mm. in diameter. Any common bile duct larger than 6 mm. in diameter should be regarded with *suspicion* for *possible* stenosis at the Vaterian segment. This suspicion holds also when oral cholecystagogues are used, but probably not with intravenous cholangiography. Perhaps more important than the common bile duct size in determining duct stenosis is retention of the opacified bile in the common bile duct after the gallbladder has completed its maximum evacuation.

The common bile duct size reached a maximum of 9 mm. in 1 patient and 7 mm. in three other patients; another had a common bile duct that measured 6.5 cm. in diameter (Table III). The patient, who showed a 9 mm. common bile duct with a markedly scarred, small gallbladder, at surgery 18 months after the roentgen examination had revealed a very large gallbladder. Unfortunately, the surgeon did not recall the roentgenologic report of prob-

TABLE III

COMMON BILE DUCT DIAMETERS IN CHOLECYSTOKININ CHOLECYSTOGRAPHY

Size (mm.)	Controls (108)	Patients (114)
2	21	8
3	50	13
4	21	34
5	12	21
6	2	10
7	2	18
8	0	4
9	0	2
11	0	1
12	0	1
16	0	1

able stenosis at the sphincter of Oddi, and the common bile duct was neither probed nor examined by cholangiography. Three months after surgery the patient is still experiencing the same marked symptoms he had prior to surgery and it is likely that a sphincterotomy will have to be performed. Of the remaining patients all but one was considered to have stenosis by the surgeon; in 3 of these patients a No. 4 Bakes dilator could be passed but not a No. 5; after sphincterotomy these patients did well except the one who had concomitant pancreatitis. Recently 2 patients, not included in this study, had common bile ducts measured at 8 to 9 mm. in diameter and there was no stenosis found at surgery.

SERUM AMYLASE

Serum amylase studies were performed immediately before and approximately 1/2 to 1 hour after the CCK injection in 33 volunteers. No appreciable change in amylase level occurred. One volunteer only (J. C.) had slightly elevated amylase before the cholecystokinin and this remained unchanged after the injection; he is the volunteer who exhibited neck spasm with mild fundus spasm. Serum amylase studies before or after the CCK injection in the patient group were, unfortunately, not practical.

FOLLOW-UP SURGICAL RESULTS (TABLE IV)

In the first study made 1 year ago, 49 patients underwent cholecystectomy. Twelve

TABLE IV

PATIENTS STUDIED BY CHOLECYSTOKININ CHOLECYSTOGRAPHY (CCK-GB)

Total Patients Studied by CCK-GB	275
Patients with+CCK-GB who had surgery	79
Proved+by microscopy	77
Proved negative, 1 by microscopy and 1 by surgical inspection (false+)	2
Patients with negative CCK-GB who had surgery	8
Proved negative, 1 by microscopy and 1 by surgical inspection	2
Proved+by microscopy (false negative; explored despite negative CCK-GB)	6

of these had other upper abdominal surgery at the same time, such as hiatus hernia repair, vagotomy and pyloroplasty, which could have relieved their symptoms or caused symptoms in the follow-up period; therefore, these patients were dropped from the follow-up study. The follow-up period in the remaining 37 patients now ranges from 15 months to 4 years. Only 1 of these 37 patients had poor results and was sorry she had undergone surgery. All the remaining patients were improved and were glad they had the operation. Twenty were enthusiastic about the results. No appreciable change in this subjective improvement has occurred since the results were reported 1 year ago.

In the present study a total of 39 patients had cholecystectomy of whom 6 are eliminated because of concomitant upper abdominal surgery that might interfere with clinical evaluation. There were 3 false negatives but no false positives in the second study as compared to 3 false negatives and 2 false positives in the initial study.

These 33 cases have been followed for a period ranging from 3 months to 1 year after surgery. All but 2 of the patients had good results and the large majority stated that they felt fine and had no problems (Table v). The latter were classified as +++ results. There were 27 of these patients who fell in this category. Classified as ++ were 4 who were markedly improved with complete relief of postprandial pain but with occasional mild symptoms of gas

Table V

POSTOPERATIVE RESULTS OF CHOLECYSTECTOMY OVER A FOLLOW-UP PERIOD OF 3 TO 48 MONTHS

Patients	Patients Interviewed	+++ Results	++ Results	+ Results	o Results
70	70	55	11	1	3
70	70	Total 66 (94 per cent)		Total 4 (6 per cent)	

+++ Patients free of symptoms.
++ Patients markedly improved; some mild complaints.
+ Patients with slight improvement.
o Patients with no improvement.

and indigestion. One of these patients had complete relief of severe nausea after food, while upper abdominal burning, which occurred even after water, has now been relieved except for an occasional episode. There were 2 who had no relief of symptoms and they were classified o. No patient was classified as +, slightly improved.

One of the 2 patients classified o had concomitant pancreatitis. The other patient, mentioned earlier, is believed to have stenosis at the Vaterian segment but his bile duct was not explored at the time of cholecystectomy.

In summary, the 2 studies totaled 70 patients who have been followed 3 months to 4 years after cholecystectomy (Table v). Three patients were unimproved and 1 patient showed only slight improvement. Over 94 per cent were completely relieved of symptoms or greatly improved with complete relief of the most serious symptoms, particularly postprandial pain.

By way of comparison, Glen and Mannix[6] revealed results of cholecystectomy in 135 patients who had no evidence of calculi or of acute cholecystitis. There were 3 deaths. Twenty-five per cent of the 121 patients followed had no relief from their original symptoms. Their statistics explain the surgeons' reluctance to perform cholecystectomy in the presence of a normal routine oral cholecystogram or even when he finds a normal appearing gallbladder at exploratory laparotomy.

PATHOLOGY IN THE RECENT PATIENT GROUP (39)

The surgical description indicates that of the 39 cases operated, 29 had a gross appearance consistent with chronic cholecystitis and a few of these had acute cholecystitis superimposed. Signs of chronic cholecystitis on gross inspection included fibrosis about the gallbladder and portions of the cystic and common ducts, scarring of the gallbladder and sometimes of the cystic ducts, thickening of the wall of the gallbladder, and paraductal lymphadenopathy occasionally. Fibrosis about the gallbladder may, however, sometimes be caused by an inflammatory process in the adjacent organs. Cholesterolosis of the gallbladder was sometimes associated, and gallstones were found in 3 patients, 2 of whom had unsuspected stones which were not demonstrable roentgenographically even in retrospect; cholecystectomy was performed in these latter 2 cases because of positive CCK-GB examinations.

The microscopic appearance of chronic cholecystitis in 38 of these cases was considered definite, although a few were called "mild" or "minimal." The pathologist's description included some or all of the following: thickening of the gallbladder wall, numerous foci of inflammatory cells, areas of fibrosis, Rokitansky-Ashoff sinuses, thickening and fusion of mucosal folds, edema, congestion and telangiectasia. Cholesterolosis is occasionally associated.

All of the 29 gallbladders that were grossly abnormal had unequivocal evidence of microscopic cholecystitis, most of which was chronic; there were 4 classified as subacute and 2 classified as acute cholecystitis superimposed on a chronic cholecystitis. Of

the 10 that were grossly normal at surgery, there were 9 cases which had signs consistent with chronic cholecystitis. In another case called "mild chronic cholecystitis," the pathologist's description of the microscopic findings was quite meager and the slide is not available for review. It is not possible, therefore, to conclude that this case had chronic cholecystitis; at the same time, the history is that of typical gallbladder colic which has been completely relieved during the 6 month postoperative follow-up.

ILLUSTRATIVE CASES

Most of the cases have had very similar histories. The patients complained of food intolerance for 6 months to 3 years. Usually this began with indigestion and right upper quadrant or epigastric discomfort or pain soon after ingestion of certain foods including, particularly, spicy foods, cucumbers, and fried or greasy foods. These symptoms became progressively worse to the point where any food or even water could set off an attack. Oral cholecystographies performed in these cases, some times as many as 3 or 4 times, were considered normal. Several patients had undergone surgery for hiatal hernia and occasionally for what was thought to be duodenal ulcer without relief of symptoms.

The various roentgenographic findings which occur after 75 Ivy dog units of CCK given intravenously in 20 to 30 seconds are demonstrated in Figures 1–7. All of these cases were proved to have chronic cholecystitis by microscopy. All had complete relief of symptoms after cholecystectomy.

DISCUSSION

Although the majority of the patients with gallbladder disease have gallbladder calculi, there are a large number of patients with acalculous cholecystitis. In the United States the incidence of cholecystitis in all gallbladder diseases has been given as 5 to 10 per cent,[1,9,13] but our experience at Baptist Hospital of Scottsdale suggests that it is 38 per cent, consistent with the European figures of 12 to 47 per cent.[2,3]

Most of these patients suffer discomfort for years; some are labeled "neurotic" and some undergo surgical procedures for "hiatus hernia" or "peptic ulcer" when these diseases are not significant or only apparent. Exploratory surgery is frequently performed, but with no abnormalities found. In more than 25 per cent of our cases the gallbladder appeared normal to the surgeon, and it was removed only on the basis of the CCK-GB with complete relief of symptoms in most cases.

The results of cholecystectomy in these cases, normal on routine oral cholecystography but positive on CCK-GB, compare very favorably with the best results of cholecystectomy done for cholelithiasis and are far better than the results obtained in patients submitted to cholecystectomy when the routine oral cholecystograms were only questionably abnormal.[10,12] The latter result suggests that CCK-GB permits a better selection of patients for cholecystectomy.

The diagnosis of a positive CCK-GB rests on 2 main factors: first of all, the dynamic changes which occur in the gallbladder and in the ducts following intravenous cholecystokinin; and, secondly, the symptomatic response of the patient during the examination.

The roentgenographic findings and the symptomatic response of the patient to the CCK are of about equal importance in determining the presence or absence of a pathologic acalculous gallbladder. Either the positive roentgenographic findings alone or a positive symptomatic response alone is reliable in making a positive diagnosis.

In placing reliance on the symptomatic response, however, caution should be used not to suggest positive symptoms to the patient in any way. It is also important to evaluate the patient's response objectively. Sometimes positive symptoms stimulated by the CCK in a patient with obvious "psychogenic overlay" were ignored in the presence of normal roentgenographic findings. In 2 of these cases relief of the patients' symptoms, which, incidentally, could conceivably have caused the "psychogenic

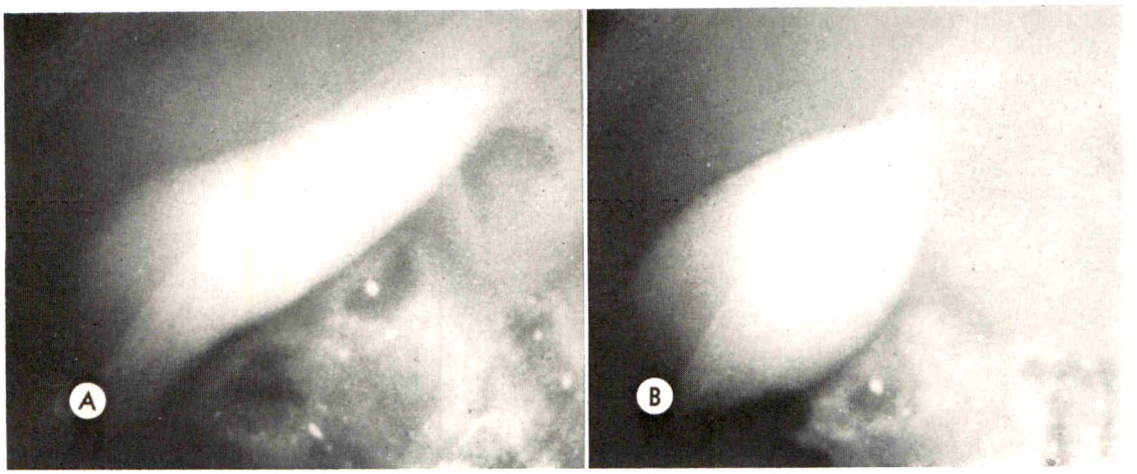


Fig. 1. (*A* and *B*) Marked spasm of the gallbladder neck. The marked change to an oval configuration and failure of the gallbladder to empty are typical findings in chronic cholecystitis.

overlay," was finally obtained by cholecystectomy several months after the patients' positive symptomatic response to CCK had been considered unreliable. In 1 of these a superimposed acute cholecystitis required emergency surgery. In the other there was progression of symptoms.

Reduplication of the patient's symptoms, particularly right upper quadrant pain, is considered a reliable indication of a pathologic gallbladder and/or stenosis at the distal end of the common duct.

Positive roentgenographic findings include spasm of the neck, infundibulum, body or fundus of the gallbladder or spasm of the cystic duct. One must be cautious in interpreting spasm of the neck of the gallbladder, particularly when it is mild or if it disappears at 5 minutes.

In normals the gallbladder contracts in

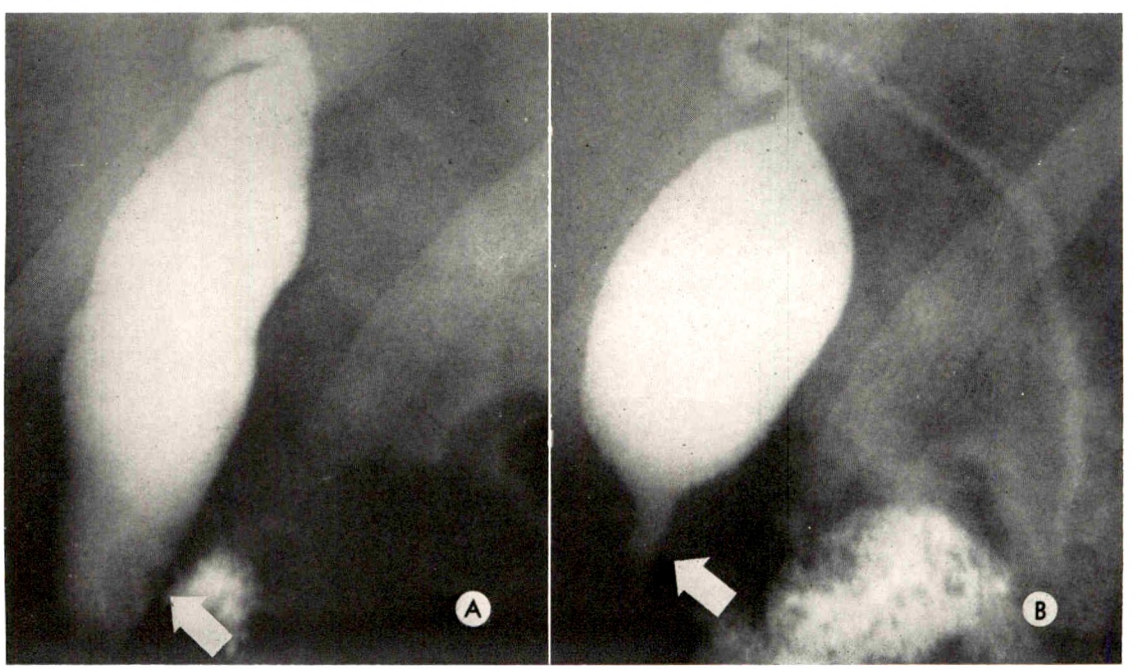


Fig. 2. (*A* and *B*) Marked fundus spasm. Neck spasm in the gallbladder is associated with change to an oval configuration in the body which suggests strong contractions of the gallbladder.

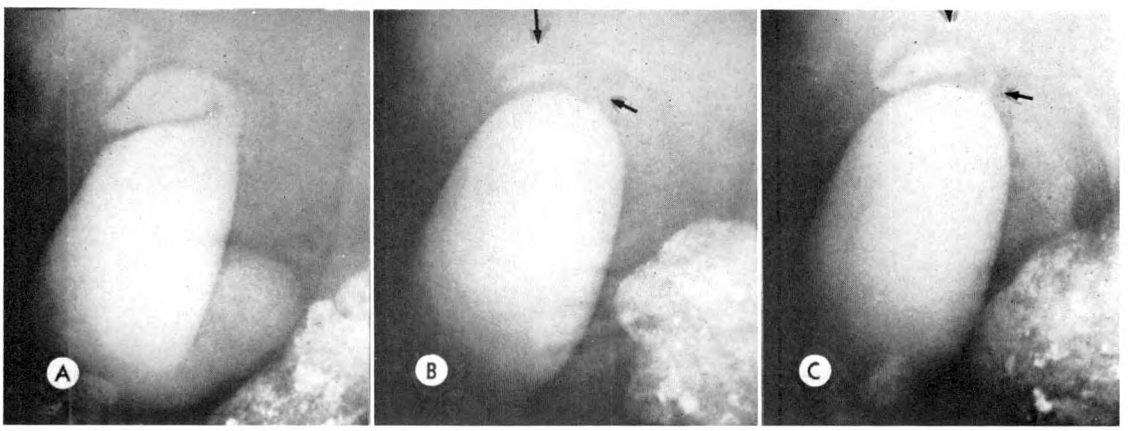


FIG. 3. (*A–C*) Cystic duct spasm is well demonstrated along with neck spasm and spasm of the fundus.

a generalized fashion. The amount and speed of contraction alone are not significant. Abnormal acalculous gallbladders exhibit localized areas of disproprotionate contraction termed "spasm." Spasms may occur anywhere in the gallbladder or in the cystic duct, and several areas may be involved simultaneously.

Spasm can sometimes occur in normals and this is particularly true at the gallbladder neck where the muscle is the thickest. To be significant it should persist on the 5 minute cholangiogram or should begin later after the 3 or after the 5 minute cholangiogram.

Stenosis at the distal end of the common bile duct is suspected when the duct measures more than 6 cm. in diameter and when the opaque material persists in the duct after cessation of gallbladder contraction. Moderate to marked hepatic duct reflux occasionally is an associated sign.

Of course, the presence of a positive symptomatic response and of a positive roentgenographic appearance together would seem to provide a more reliable basis for a positive diagnosis than either alone. Whenever the diagnosis of a pathologic gallbladder or stenosis of the distal end of the common duct by CCK-GB is uncertain, however, the examination can be repeated at a later date, possibly in 12 to 18 months, if the symptoms persist or progress.

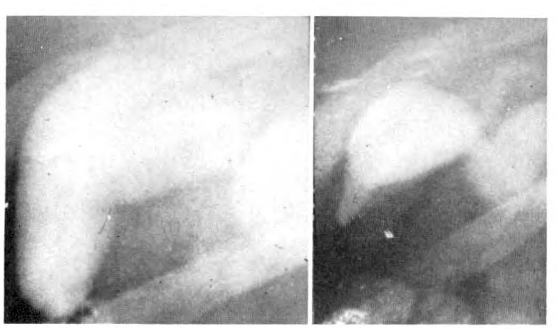


FIG. 4. Fundus spasm.

When the gross appearance of the gallbladder is normal, the microscopic findings are often minimal, and it is in these cases that some difference of opinion among pathologists may exist. The difficulty arises from uncertainty as to the appearance of a normal gallbladder, since rapid postmortem changes and changes which some have ascribed to the aging process make it extremely difficult to obtain a normal gallbladder in its pure normal state for microscopic study.

There are some who feel that an economic factor also will affect the pathologist's interpretation of the microscopic appearance. In an effort to eliminate the latter factor, all microscopic slides of the 39 more recently operated patients were reviewed by an independent pathologist (R.C.) at Maricopa County General Hospital with the understanding that he had to be critical and conservative in his analysis. Despite his position of immunity from economic pressures, he reached the same conclusions regarding the gross and micro-

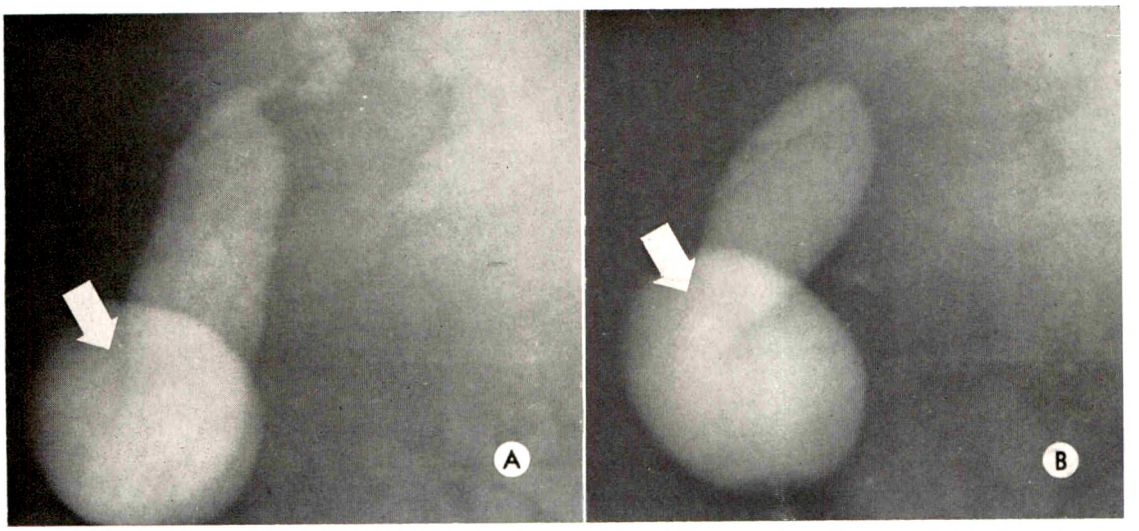


Fig. 5. (*A* and *B*) Spasm of the body associated with neck spasm and strong contractions of the gallbladder.

scopic diagnosis as did the pathologists in the various other hospitals where the cholecystectomies were performed.

The normal gallbladder has the following microscopic appearances: (1) the mucosa shows delicate connective tissue stalks covered by tall columnar cells; (2) there is a rather loose fibroconnective tissue beneath the epithelium; and (3) smooth muscle fibers are arranged longitudinally and then obliquely or circularly. The external layer is the peritoneum.

In minimal cholecystitis, the epithelium is usually intact, with coarse mucosal folds

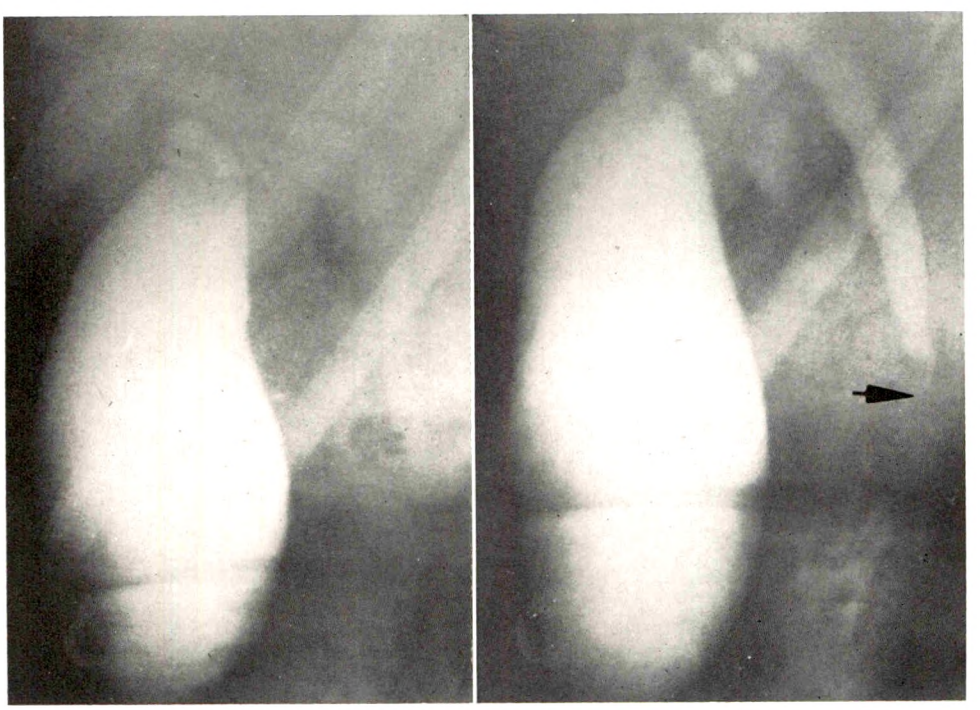


Fig. 6. Stenosis of the Vaterian segment of the common bile duct (arrow). There is also moderate neck spasm.

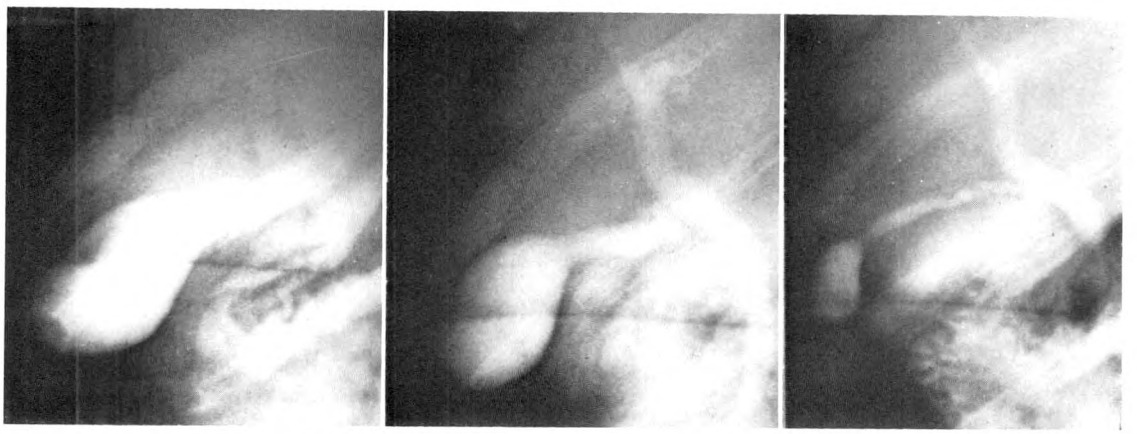


FIG. 7. Marked common hepatic duct reflux and dilatation of the common bile duct due to spasm at the Vaterian segment.

and infiltration of the lamina propria and muscularis by inflammatory cells including lymphocytes, plasma cells and large mononuclear cells. Fibrosis of various degrees is a usual component of the picture. Enlargement and occasionally fusion of the mucosal folds may be found.

Munster and Brown[11] consider the following as evidence of mild chronic cholecystitis: scattered foci of lymphocytic infiltration; and fibrosis and some plasma cells in the muscular coat or lamina propria. In a personal communication Brown, reflecting the thinking of the senior staff in the Department of Pathology at Peter Bent Brigham Hospital, has stated, "It is unclear what the normal gallbladder has for a cell population—it would appear that the normal gallbladder may contain a few scattered lymphocytes and histiocytes in its muscularis or lamina propria, but no plasma cells."

On the basis of our experience thus far, chronic cholecystitis is believed to be the common denominator in essentially all of these cases and the same process may be the cause of Vaterian stenosis. Dyskinesia at the sphincter of Oddi, on the other hand, has been suspected in only 1 of the cases who underwent surgery while Vaterian stenosis is not uncommon and was considered to be present in 12 cases (14 per cent).

Failure to diagnose stenosis, either in the presence of chronic cholecystitis or in the presence of cholelithiasis, is likely to result in persistence of the symptoms or recurrence of symptoms later. Preoperative evaluation of the common bile duct, preferably by the CCK-GB or by intravenous cholangiography, would be advantageous and might make exploration of the duct unnecessary. When stenosis of the Vaterian segment is suspected, a new form of operative cholangiography using manometry[4] probably should be done since it would give much more information concerning stenosis; if this technique is not available, evaluation for stenosis by passage of a surgical probe through the common duct may be necessary.

Should a patient with a definitely positive CCK-GB be subjected to surgery? The answer to this is the same as for most elective surgery: if the patient's symptoms can be controlled by diet or medication, or if the patient is not disturbed excessively by his symptoms, it is advised that no surgery be performed. Progression of disease may, however, intervene and make cholecystectomy necessary.

Progression of pathology was observed in several of our cases including the 2 instances of acute cholecystitis superimposed on chronic cholecystitis. In 2 other cases marked decrease in the size of the gallbladder from the time of the CCK-GB, when the gallbladders were both observed to be quite large, until the time of the surgery (6 months later in 1 and 18 months

later in the other), when the gallbladder in each case was markedly scarred and quite small, also indicates progression. There were 3 other cases of progression in which tiny radiolucent calculi developed, as shown by repeat CCK-GB, 6 to 18 months after the initial examination (this does not include 2 cases in which the gallstones could not be demonstrated roentgenographically).

The true evaluation of this new method of gallbladder examination rests on the results of surgical therapy in the final analysis. These results were excellent and symptomatic relief was achieved in 94 per cent of these patients.

CONCLUSIONS

1. Cholecystokinin cholecystography is a safe, reliable examination which provides information usually not obtainable by routine oral cholecystography.

2. The diagnosis of the pathologic gallbladder by cholecystokinin cholecystography (CCK-GB) relies either on symptomatic response to the intravenous CCK or on the dynamic roentgenographic findings, but either alone can provide a correct diagnosis.

3. Chronic cholecystitis is found apparently in all cases of acalculous gallbladder which, although appearing normal on routine oral cholecystography, are abnormal on the CCK-GB as determined by the dynamic roentgenographic changes or by the symptomatic response.

4. Progression of chronic cholecystitis may occur in these cases and acute cholecystitis may be superimposed.

5. Analysis for possible stenosis at the distal end of the common duct by means of the CCK-GB is important if postcholecystectomy recurrence or persistence of symptoms is to be avoided not only in chronic cholecystitis but also in cholelithiasis.

6. Marked to complete alleviation of symptoms is obtainable in 94 per cent of cases exhibiting positive cholecystokinin cholecystography.

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ABSENT GALLBLADDER AND CYSTIC DUCT

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ABSENCE of the gallbladder and cystic duct, exclusive of surgical removal, may be congenital in origin or may be the end result of inflammatory disease. Cholecystic agenesis is uncommon with only about 150 reported cases,[9] and usually the cystic duct is also absent.[7] Acquired absence may be difficult or impossible to differentiate from congenital cases. Present preoperative roentgen methods cannot differentiate lack of a gallbladder from a nonvisualized viscus, but operative cholangiography is of value in this regard.

Three illustrative cases of absence of the gallbladder and cystic duct are reported.

REPORT OF CASES

Case I. A 64 year old white man was admitted to Methodist Hospital on June 16, 1966 because of increasingly severe pain in the upper abdomen of 1 day's duration, followed by vomiting. The patient was an alcoholic. Since the age of 9, he had frequent attacks of generalized abdominal pain lasting a few days and associated with constipation. There was no previous abdominal surgery. Serum bilirubin was 3.0 mg. per cent on admission which then decreased to normal levels by June 22. Cholecystographic examination failed to disclose a gallbladder shadow. A gastrointestinal series was normal, except that there were 2 chicken bones in the small intestine; the patient, however, had no recollection of having swallowed these bones.

At laparotomy on June 24, there were extensive adhesions which had effaced the normal anatomy. The small and large intestines were enveloped in adhesions of omentum from the right to the left lumbar gutter, the omentum was adherent to the anterior abdominal wall and the entire anterior surface of the liver was coated with dense adhesions. Two chicken leg bones were removed from the bowel. Despite a diligent search, no gallbladder or cystic duct was found. The major biliary ducts were easily located. There was no cystic duct nubbin or cholecystic remnant. The common duct was opened and the distal and proximal portions were carefully palpated. Dilators were passed into the duodenum easily and into the proximal duct system without obstruction. A "T" tube was inserted and a cholangiogram was obtained, which showed a small filling defect in the lower aspect of the common bile duct. Otherwise, the common bile duct, common hepatic duct and its branches appeared normal.

Two weeks later, "T" tube cholangiography demonstrated a filling defect at the lower aspect of the common bile duct with no sign of a gallbladder or cystic duct. The ducts otherwise seemed normal. On July 15, the patient went to surgery again. The common duct was reopened and the "T" tube removed. After probing the common duct, sludge was removed, there was a gush of bile and patency was established. A new "T" tube was inserted and the cholangiogram now appeared completely normal. Once more, there was no evidence of a gallbladder or cystic duct. On August 12, the patient was discharged in good condition.

A "T" tube cholangiogram on September 19 displayed normal biliary passages. Again, this examination failed to disclose the presence of a gallbladder or cystic duct. His general condition was good and the "T" tube was removed (Fig. 1).

Comment. Could inflammatory obliteration in the past have been so completely selective that no gallbladder remnant or cystic duct vestige remained while leaving intact all major biliary channels? This cannot be excluded and therefore the lack of a gallbladder and cystic duct, whether congenital or acquired, remains indeterminate.

Case II. A 64 year old white woman entered Methodist Hospital on November 16, 1967 with a history of vomiting and generalized abdominal pain, which was most marked in the epigastric region and was of 4 days' duration.

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The patient had diabetes for which she had been taking tolbutamide, but during the present illness she stopped the medication. On admission, the blood sugar was recorded as 540 mg. per cent and urinalysis showed 4+ sugar and a moderate amount of acetone; insulin was given and the next day the urine was negative for sugar and acetone. The patient was alert. The abdomen was distended but soft, and there was no tenderness. During the following 2 days, she vomited several times, the abdominal pain continued and the abdomen remained distended. There was no jaundice. Roentgenograms of the abdomen displayed typical findings of gallstone ileus.

An exploratory laparotomy on November 18 revealed extensive inflammation and adhesions in the right upper quadrant distorting the normal anatomy in this area. The patient was in such poor condition that no attempt was made to separate and identify the structures of the upper abdomen. The distal ileum was obstructed by a gallstone. An ovoid, brownish, yellow calculus measuring 3.5×2.5×2.0 cm. was recovered from the ileum, which was reported by the pathologist to be a mixed cholesterol stone. The patient died 12 hours after the completion of surgery.

Postmortem examination showed bilateral bronchopneumonia, hepatoduodenal fistula with hepatic abscess and a large opening through the postbulbar region of the duodenum, which was adherent to the abscess cavity. The cavity contained yellowish, necrotic material with a yellow-green, purulent margin surrounded on all sides by a congested zone of liver parenchyma measuring 3 cm. in depth. No structures were recognizable within the abscess. The distal portion of the common bile duct and the vaterian region appeared normal, but the remainder of the extrahepatic radicals were not identifiable. A gallbladder was not seen and no cholecystic remnant or cystic duct was found either on thorough gross inspection or microscopic study. The intrahepatic ducts were normal. There was no intrahepatic gallbladder.

Comment. Here the lack of a gallbladder and cystic duct, one would have to assume, was due to severe inflammatory disease which destroyed and eliminated all traces of the 2 structures.

Case III. A 34 year old Puerto Rican woman was admitted to Methodist Hospital on November 3, 1969, with a 1 year history of intermittent right upper abdominal quadrant pain, which radiated to the back. This, on occasion, was associated with nausea and vomiting. There was no history of jaundice. Two years previously, a vaginal hysterectomy was performed for uterine polyps, cystocele and rectocele. There was no visible gallbladder by oral cholecystography on July 25, 1968. On November 4, 1969, intravenous cholangiography demonstrated normal common hepatic and common bile ducts, with nonvisualization of the gallbladder. On the following day, laparotomy was performed. The gallbladder and cystic duct were absent. The major biliary radicals appeared normal and there was no other abdominal abnormality. The patient was discharged in good condition 10 days later.

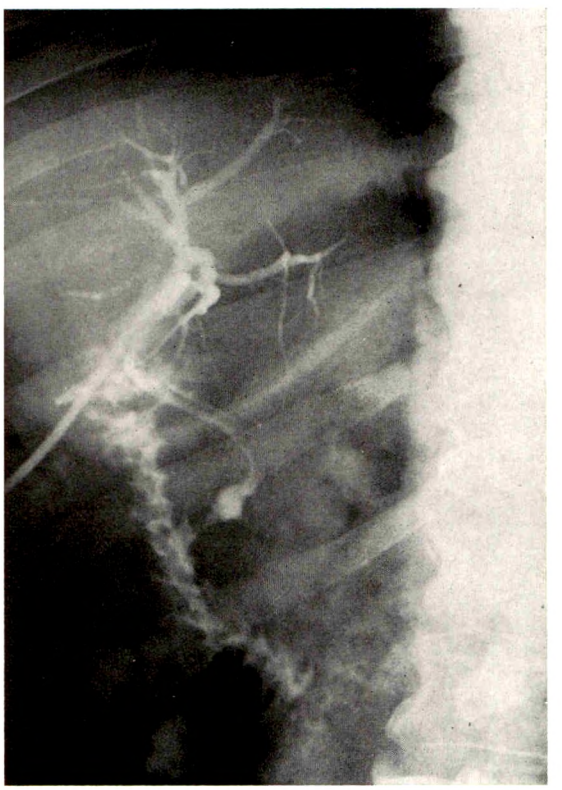


Fig. 1. Case 1. "T" tube cholangiogram. Normal bile radicals, absent gallbladder and cystic duct.

Comment. This must be considered a true agenesis of the gallbladder and cystic duct since there is no indication whatever of any previous, additional intraabdominal abnormality which might have fibrosed or otherwise destroyed these 2 structures.

DISCUSSION

A definitive preoperative diagnosis of absence of the gallbladder has not and probably cannot be made.[1,3,4] As might be expected, the preoperative impression consistently is cholecystic disease, or common duct obstruction.

Despite good reviews by several authors,[1,2,5–7,10] it is debatable whether or not cholecystic agenesis accentuates any inclination to functional or structural disturbance of the biliary channels. To the proponents, the idea of a causal relationship is based on the assumption that biliary disease occurs relatively more often in people born without a gallbladder.[2,3] One cannot, however, offer any statistical support for this assumption. Also, the mechanism of such action is enigmatic.

There are a number of reasons to support the adverse viewpoint which holds that congenital lack of a gallbladder does not increase the tendency to biliary tract disease,[8] and that when they do co-exist, the agenesis is simply an incidental finding. In some species of animals this viscus is normally absent.[2] In adult cases of agenesis first found at postmortem examination, there is little evidence that in life they suffered from biliary tract disease.[7] Furthermore, the sex distribution and the age incidence of the clinical cases compare with the usual run of biliary tract disease in the general population. While the autopsy cases indicate an equal sex distribution, the surgical cases show a ratio of 3 females to 1 male.[6] In the necropsy cases, although all age groups from the newborn to old age are recorded, in those that come to surgery the age spread is from the twenties to the seventies, with most patients falling within the 40 to 50 year bracket.[3] Finally, most cholecystectomized patients do well and there is no apparent essential difference in function between congenital absence and surgical removal of the gallbladder.

As indicated above, some people born without a gallbladder remain asymptomatic throughout life, the agenesis being discovered at autopsy. Others are found incidentally at laparotomy performed for unrelated conditions. Still others may present with complaints suggesting biliary tract disease, and such symptomatic cases have been recorded with completely normal biliary radicals as in our third case, with normal caliber ducts containing stones, and with choledochal dilatation with and without stones.[1] This indicates, as is well known, that stones can be formed in biliary channels and that the gallbladder is not the only breeding ground for gallstone formation.

Lack of a gallbladder must be differentiated from one that is unusually placed and also from one that may be concealed or unrecognized. Operative cholangiography is valuable. This will aid not only in establishing a diagnosis of absent gallbladder, but will help in determining whether or not the biliary tree is diseased.

An intrahepatic gallbladder may be suspected preoperatively by cholecystectography in which this structure will appear in a rather high position as if located within the liver shadow. It may also be demonstrated by operative cholangiography. In another abnormally located position to be considered, the organ is bound to the inferior aspect of the left hepatic lobe, the so-called left-sided gallbladder. Also, very rarely, the gallbladder may be located in the abdominal wall, in the falciform ligament and even retroperitoneally.[3]

In situations of disease involving the right upper abdominal quadrant with gross distortion of the normal anatomy, the surgeon may find it very troublesome to identify the gallbladder and cystic duct. These structures may go undetected if encompassed by dense adhesions or buried in an inflammatory mass, or, as suggested in our second and possibly in our first case, they may be completely eliminated by inflammatory destruction. Operative cholangiography should be of help, but distinguishing these cases from agenesis may prove impossible.

A rudimentary gallbladder, represented by a small nubbin attached to the cystic duct might be due to hypoplasia or be the end result of repeated attacks of cholecystitis—roentgenographic methods cannot be expected to distinguish such cases from congenital absence of the gallbladder.

SUMMARY

Three patients are reported with absence of the gallbladder and cystic duct.

The first case, whether congenital or acquired is indeterminate. In the second case, the gallbladder and common duct were probably destroyed by acute inflammatory disease. The last case most likely represents a true agenesis.

A preoperative diagnosis probably cannot be made.

Operative cholangiography is of value.

Acquired absence of the gallbladder and cystic duct may be difficult or impossible to differentiate from agenesis.

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A METHOD FOR THE EXPULSION OF STONES RETAINED IN THE COMMON BILE DUCT*

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NOT infrequently retained gallstones are detected in the common bile duct following cholecystectomy or choledocolithotomy. The exact incidence of retained calculi varies from 8 to 9 per cent in patients following choledocolithotomy[9] to a low of 1.4 per cent in a series of patients in whom operative cholangiographies have been routinely performed.[8]

Many patients with retained calculi will continue to experience difficulties related to the biliary tract until all of the stones have been surgically removed.[5, 9] Because of this, re-exploration of the common duct is generally indicated in these patients.[9]

It seems desirable that if retained calculi can be successfully removed or expelled from the common duct without the necessity for further surgery, then this should be undertaken. A recently described technique[4] for the expulsion of common duct stones was successfully utilized, with minor modifications from the original description, by us in a patient in whom 2 calculi were revealed in the common bile duct on a postoperative T-tube cholangiogram.

REPORT OF A CASE

A 60 year old white male was admitted to The University of Chicago Hospitals during an acute attack of pain in the right upper quadrant of the abdomen. The patient was jaundiced and on examination there was marked tenderness in the right upper quadrant of the abdomen. The temperature was 39° C. On the fifth hospital day he underwent cholecystectomy and exploration of the common bile duct. The gallbladder was found to be small and contracted and adhered to the surrounding omentum, but no calculi were detected within it. The common bile duct was markedly distended and 7 calculi, measuring 0.8 to 0.5 cm. in diameter, were removed from it. There was no evidence of stenosis of the sphincter of Oddi, and Bakes dilators up to No. 6 passed readily through into the duodenum. An operative cholangiogram following removal of the stones revealed no additional calculi and the contrast material flowed freely into the duodenum. The patient's postoperative course was unremarkable and the jaundice rapidly cleared.

A T-tube cholangiogram on the seventh postoperative day revealed 2 radiolucent calculi within the common bile duct. A repeat examination on the following day confirmed the presence of 2 stones. The T-tube was clamped and the patient was discharged and then re-admitted 5 weeks later for re-evaluation. Another T-tube cholangiogram was obtained and this

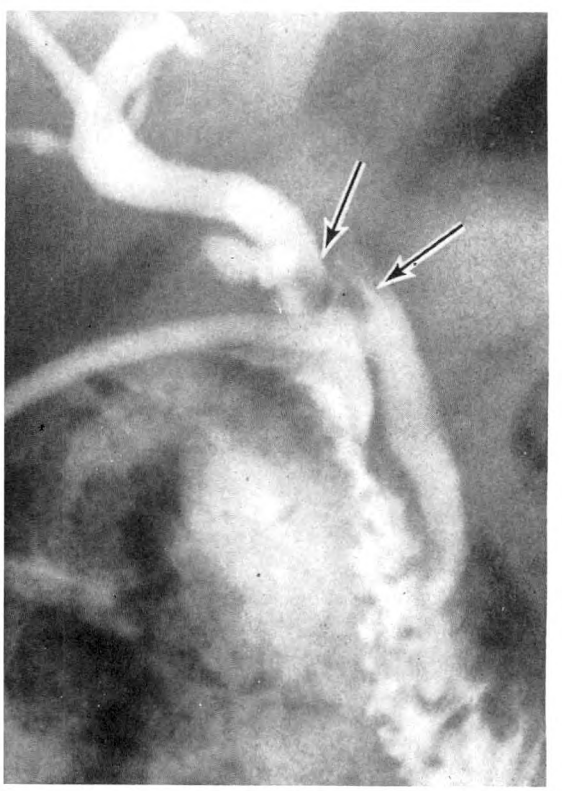


FIG. 1. Postoperative T-tube cholangiogram demonstrating 2 calculi (arrows) within the common bile duct.

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again demonstrated the presence of 2 stones within the common bile duct (Fig. 1). The T-tube was removed and a soft rubber urinary catheter No. 14 Fr. was inserted through the tract into the common bile duct. Contrast material was injected through the catheter re-demonstrating the calculi (Fig. 2). The duct was then irrigated with warm saline and the catheter was advanced through the sphincter of Oddi into the duodenum (Fig. 3). Minimal resistance was encountered at the sphincter but the patient experienced no discomfort at any time. The catheter was then withdrawn into the common duct and contrast material was again injected. This revealed that only 1 of the calculi remained in the duct. On the next day the procedure was repeated. Again a single calculus was demonstrated and by tilting the fluoroscopic table into a semi-upright position and by manipulating the catheter, the remaining calculus was pushed into the distal common duct adjacent to the sphincter of Oddi (Fig. 4). By gently advancing the catheter with the table in a semi-upright position it was possible to maintain the calculus in the distal common duct. The calculus was then easily pushed through the sphincter of Oddi into the duodenum. Contrast material was again injected into the common bile duct and no further calculi were seen. In order to confirm removal of all stones a further contrast medium injection was performed on the next day (Fig. 5) and despite a careful search no stones were seen. The catheter was then withdrawn. The tract healed readily and the patient has remained asymptomatic since that time.

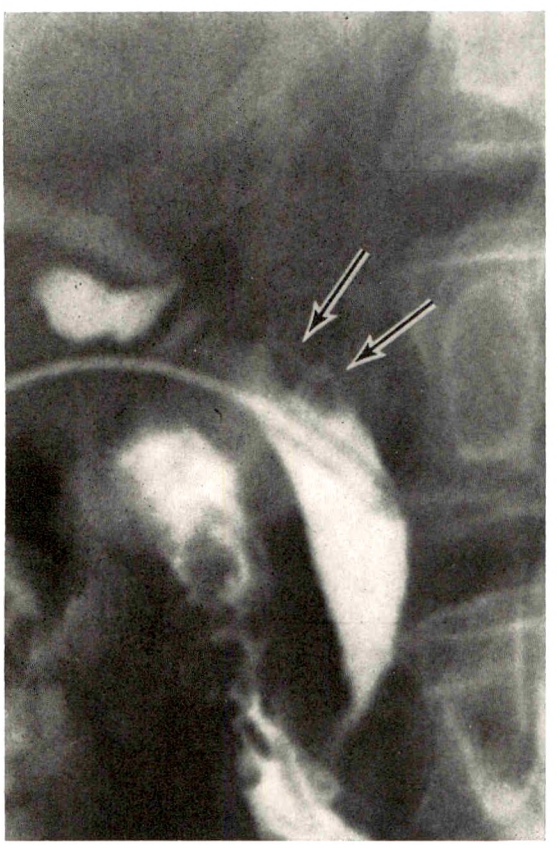


Fig. 2. The T-tube has been removed and a soft rubber urinary tract catheter has been inserted through the T-tube tract. Injection of contrast material again demonstrates 2 calculi (arrows) within the common bile duct.

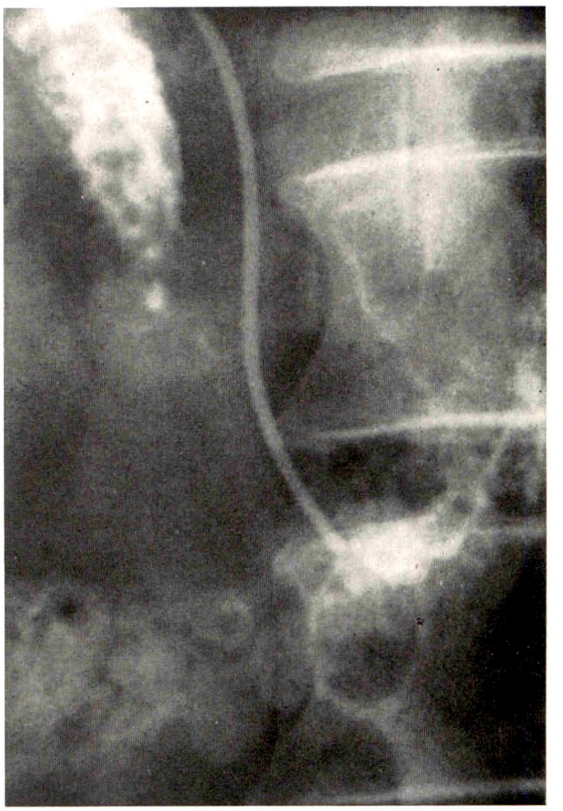


Fig. 3. Following irrigation of the common bile duct with warm saline, the catheter containing a small amount of contrast material is advanced through the sphincter of Oddi into the duodenum.

DISCUSSION

Many techniques have been advocated for the nonoperative removal of retained biliary tract calculi. Solvents such as

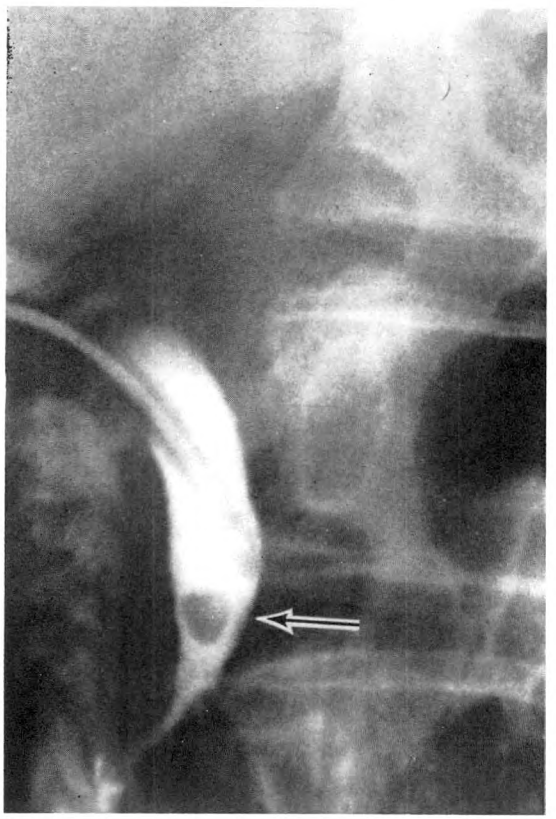


FIG. 4. The catheter has been drawn back into the common bile duct and a repeat injection of contrast material at this time demonstrates only 1 stone in position. By manipulation of the catheter, with the patient in a semi-upright position, the remaining stone is advanced to the region of the sphincter of Oddi (arrow).

ether[6] or chloroform[2] have been instilled through T-tubes but their use is no longer recommended.[10] Irrigation with oil[1] or bile[7] has also been employed; however, Thorbjarnarson and Glenn have suggested that only warm water or saline should be used for this purpose.[10] On reviewing descriptions of the utilization of some of the more esoteric solutions, it seems quite probable that the successes reported with these techniques might equally well be attributed to the irrigation alone rather than to the chemicals utilized.

The technique which we have described is, in essence, that published previously by Lamis *et al.*,[4] who reported the successful expulsion of retained calculi from the bile ducts in 2 patients. In a third case, however, the attempt was unsuccessful.

It is possible that calculi in the intrahepatic ducts might not be dislodged by this technique. We feel that a modified Fogarty catheter inserted through a coude-catheter with an end rather than a side hole and guided to the appropriate duct by means of a device such as a Mueller guide might be successful in these cases. We have not yet had an opportunity to attempt this technique clinically, but the use of Fogarty catheters for the removal of bile duct stones has been described.[3]

SUMMARY

A simple atraumatic method for the expulsion of retained common duct calculi



FIG. 5. The final cholangiogram obtained by injection of contrast medium through the catheter demonstrates that the biliary duct system is now free of calculi.

is described. While experience with one case cannot be construed as confirmation of the value of the technique, we feel that the simplicity of the procedure, both for the operator and the patient, warrants bringing it to the attention of radiologists.

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THE INFERIOR ESOPHAGEAL SPHINCTER—ANATOMIC, ROENTGENOLOGIC AND MANOMETRIC CORRELATION, CONTRADICTIONS, AND TERMINOLOGY*

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IN 1950, Lerche[22] demonstrated detailed dissections of the esophagogastric region and attempted to draw functional conclusions from anatomic findings—with no supporting evidence. In the majority of 80 specimens, Lerche found a localized ring of thickened circular muscle in the muscularis propria at the site of the "lower physiological constriction" described in 1883 by Laimer. Laimer had described this as a discrete circumferential narrowing of the esophagus located about 2 cm. above the hiatus. The presence of a muscular ring at this site has been confirmed by others,[12] although its demonstration depends on the method of preparing the specimen. Lerche introduced the term "inferior esophageal sphincter" for this muscle band. He suggested that it marked the distal end of the esophagus and that the segment below it, between the esophagus and stomach, should be considered a separate anatomic as well as functional unit. He coined the term "vestibule" for this segment and defined it as being bounded by the "inferior esophageal sphincter" above and by "constrictor cardiae" fibers below. It therefore consists of supradiaphragmatic and infradiaphragmatic portions. On the basis of specimens presenting "states of contraction that showed markings characteristic of regurgitation," he concluded that regurgitated material temporarily collected in the vestibule between two closing mechanisms. Lerche was impressed by potential ballooning of the vestibule as a unit rather than by independent contractile activity of this segment. In Lerche's specimens, the relationship of the vestibule to the hiatus was not clearly described and an unknown number of small hiatal hernias were no doubt included. He did not appreciate that, *in vivo*, a tubular segment at least 2 cm. in length is present within and below the hiatus (Fig. 1, *A* and *B*). This segment has been referred to by various names—abdominal gullet, abdominal esophagus, gastroesophagus, epicardia, empty or submerged segment. The term "submerged segment"[39] is appropriate to indicate its intraabdominal location and is noncommittal as to its nature. When the submerged segment is displaced above the hiatus as a result of herniation (Fig. 2), it loses its tubular configuration and cannot be identified as such. A "submerged segment" of similar appearance, however, may be formed by a portion of herniated stomach enclosed by the hiatus or created at operation for hiatal hernia repair. This term therefore at times must be modified as a "submerged segment of stomach" or "an artificially created submerged segment."

Lerche also used the term "ampulla of the esophagus" or sometimes phrenic ampulla, apparently not to slight previous authors. In order to distinguish ampulla from vestibule, he had to assume that there was a second functionally distinct distensible segment above the vestibule of about the same size. Lerche's ampulla, as indicated in his specimens, is quite artificial and he attributed no special function to it. The ampulla of Lerche does not exist and must not be equated with the phrenic ampulla introduced into the radiologic literature by Templeton[33] (Fig. 2). The roentgenologic phrenic ampulla refers to a more distensible region of the esophagus immediately above the hiatus. This region assumes an inverted

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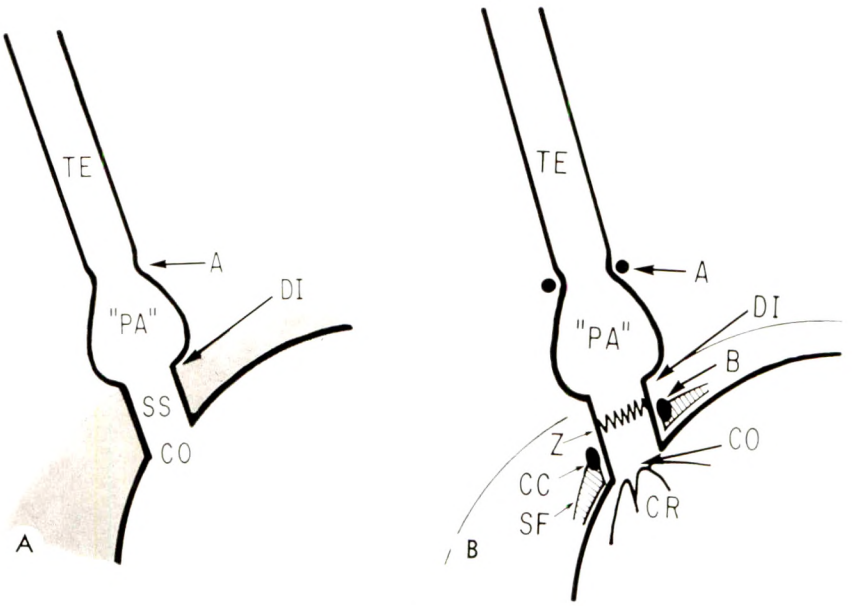


Fig. 1. Diagrammatic representation of the normal esophagogastric region.

(*A*) Appearance of lumen when filled with fluid barium. The end of the tubular esophagus (TE) is marked by a shallow circumferential indentation which indicates the A level or residual A ring activity. The segment between this level and the diaphragmatic indentation (DI) is the "phrenic ampulla" ("PA"). The tubular segment between the diaphragmatic indentation and the cardiac orifice (CO) is the submerged segment (SS). The normal esophagogastric *region* consists of the phrenic ampulla and the submerged segment, *i.e.*, it extends from the A level to the cardiac orifice. Since there is no accepted definition of the esophagogastric *junction*, it is advisable to avoid this term, although, in the configuration shown, it is often applied to the cardiac orifice. (*B*) Anatomic features added to luminal configuration. At the A level, there is potentially a circumferential discrete muscular band, "the inferior esophageal sphincter" of Lerche. The squamo-columnar mucosal junction or Z line is usually located in the upper portion of the submerged segment. Constrictor cardiae (CC) and sling fibers (SF) surround the distal portion of the submerged segment. The cardiac rosette (CR) is located at the cardiac orifice (CO). The B level corresponds to the location of the Z line or the adjacent constrictor cardiae fibers which are sufficiently close to each other to permit the designation of a single level. This level has also been called the esophagogastric *junction*. The "vestibule" extends from A to B.

pear-shaped configuration when the esophagus proximal to it empties (Fig. 3). It is also well seen during the course of a maintained deep inspiration, that is, a pinchcock maneuver, in which barium is trapped above the hiatus. The supradiaphragmatic portion of the vestibule is included within the phrenic ampulla and its infradiaphragmatic portion within the submerged segment (Fig. 2). Strictly speaking, the pointed cap of the phrenic ampulla, created by the peristaltic wave as it reaches the ampulla, is located on top of the vestibule. This may be evident during emptying of the phrenic ampulla when the cap configuration is converted into the flat top of the vestibule (Fig. 3). In practice, no confusion is created if the term phrenic ampulla is used for any of the *normal* ampullary dilatations above the hiatus.[39] However, an "ampulla" clearly larger than normal must include a herniated portion of the submerged segment and should not be referred to as the phrenic ampulla. Since the upper limit of normal size of the phrenic ampulla is difficult to determine, border-line cases will occur in which the term ampulla must be used in a nonspecific or descriptive way and then suitably qualified. Moreover, some unavoidable confusion arises because of up and down movement of the esophagogastric region in relation to the hiatus during fill-

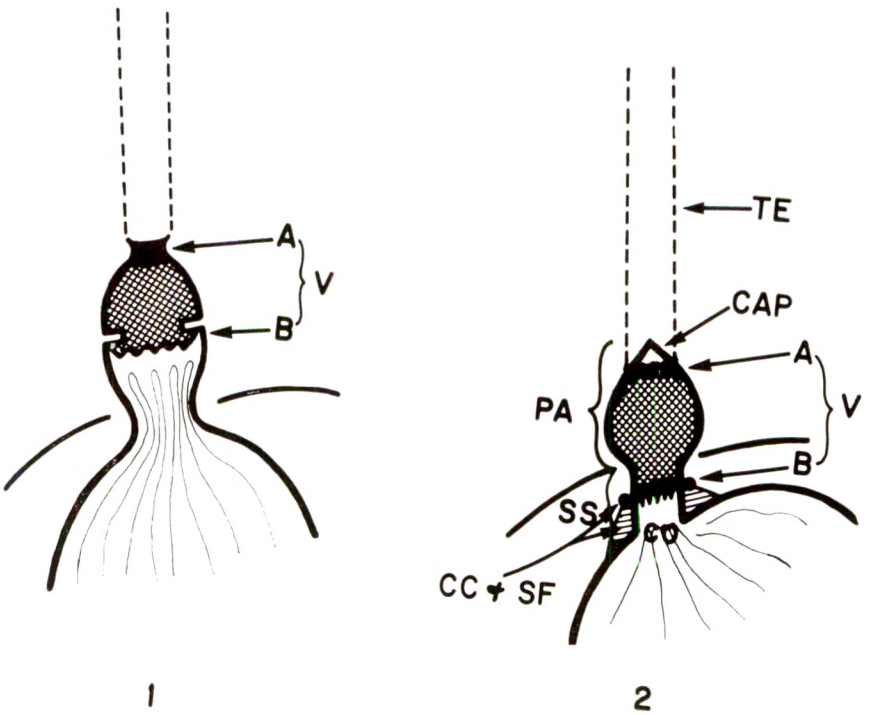


Fig. 2. Diagrammatic representation of the "vestibule" and phrenic ampulla.

Panel *1* indicates a prototype small sliding hiatal hernia with identifiable A and B rings. The vestibule (V, cross-hatched) is the segment between the two rings. No "cardiac orifice" is evident. Panel *2* indicates the normal esophagogastric region; A and B rings are indicated but are not identifiable as such. The vestibule (V, cross-hatched) includes a large supradiaphragmatic portion and a short infradiaphragmatic portion. Strictly speaking, it does not include the apical cap (CAP) of the phrenic ampulla or the sling fiber (SF) portion of the submerged segment. No landmark for the distal end of the vestibule exists normally since neither the Z line nor the constrictor cardiae fibers (CC) have any roentgen manifestation. Phrenic ampulla (PA), the submerged segment (SS), the tubular esophagus (TE), and the cardiac orifice (CO) are also labeled.

ing and emptying, particularly if minimal herniation is present (Fig. 4, *A–F*).

The descriptions of Lerche have been interpreted in various ways by different authors in attempts to incorporate anatomic findings into roentgen and manometric observations.[2,6,18,28,30,34,43] The present author and colleagues[37,38] described roentgen observations in patients with small sliding hiatal hernias which demonstrated that the terminal 3 cm. or so of the esophagus showed functional features, specifically independent contractility, which differed from the remainder of the esophagus. This region was also capable of distending to a greater diameter than the esophagus proximal to it and could reasonably be equated with the vestibule of Lerche. However, evidence for the presence of the "constrictor cardiae" as a discrete contractile ring at the distal margin of the vestibule could not be obtained. Instead, it was assumed that a suitable roentgen landmark for the distal margin of the vestibule, the "B" level, was the circumferential web-like ring described by Templeton.[34] This ring was not included in any of Lerche's descriptions. According to Templeton, it was a normal variation of the phrenic ampulla of no clinical significance, but the present author[37] presented evidence to indicate that the squamocolumnar mucosal junction was close to, if not at, this ring. Schatzki and Gary,[32] and Ingelfinger and Kramer,[17] noted that such a ring could act as an obstruction with an opening so small as to cause periodic dys-

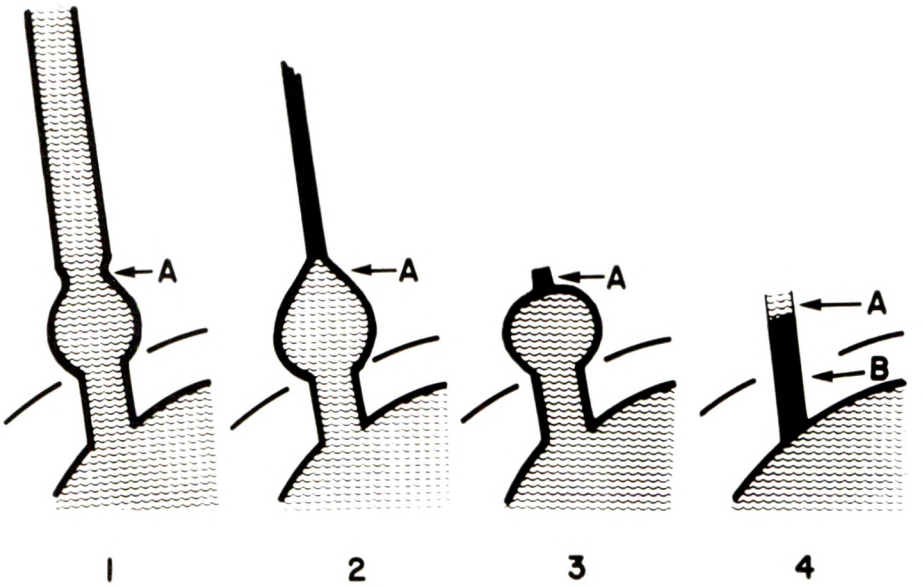


Fig. 3. Diagrammatic representation of normal functional and anatomic features during the course of emptying of the esophagogastric region. The A level is indicated in each panel.

Panel *1* shows a continuous fluid column flowing through the tubular esophagus and the esophagogastric region. Pressure throughout the column (shaded) is essentially equal to fundic pressure. The rapid stripping peristaltic wave is slowed just above the A level (Panel *2*). The apex of the phrenic ampulla is flattened (Panel *3*) and sealed off by contraction at the A level. After complete emptying, a zone of pressure elevated above fundic pressure (Panel *4*, solid black column) persists in the resting phase. This is the normal physiologic inferior esophageal sphincter as described by Code and his co-workers—not the inferior esophageal sphincter described by Lerche. Resting pressure at the A level is about equal to fundic pressure. The approximate position of the B level is indicated in Panel *4*.

phagia. The interpretation of Schatzki and Gary,[32] namely, that rings of this type are static, has been confirmed by subsequent observations.[16,34] MacMahon and co-workers[23] described a specimen of a typical stenotic distal ring studied in detail at autopsy and demonstrated that it consisted of a reduplication of mucosa containing muscularis mucosae and a fibrous extension of the submucosa. This specimen also showed that the squamo-columnar junction was located on the inferior aspect of the ring. Since this indicates that the vestibule is lined by squamous epithelium, it was considered to be part of the esophagus. The esophagus proximal to the vestibule is designated the body of the esophagus or the tubular esophagus since it maintains a tubular configuration during all phases of filling and emptying.

Of special interest in the studies noted above[37,38] was the observation that the *proximal* margin of the vestibule ("A" level) could often be identified by a short contractile ring clearly functional in nature (Fig. 4, *A–F*). This contractile ring at the junction of the tubular esophagus and the vestibule appeared to correspond to Lerche's inferior esophageal sphincter and was therefore so designated. This anatomic-roentgenologic correlation has withstood the test of time.[2,3] In retrospect, however, the acceptance of Lerche's term, inferior esophageal sphincter, for this ring was unfortunate because of the connotation that this ring is the normal sphincteric or antireflux mechanism in the esophagogastric region. This has turned out to be incorrect. In 1956, Fyke and associates[13] reported studies of intraluminal pressures in the distal esophagus during and between swallows in normal healthy individuals. They demonstrated—and this has been confirmed by all subsequent investigators[4,10,11,19,28,35] that the

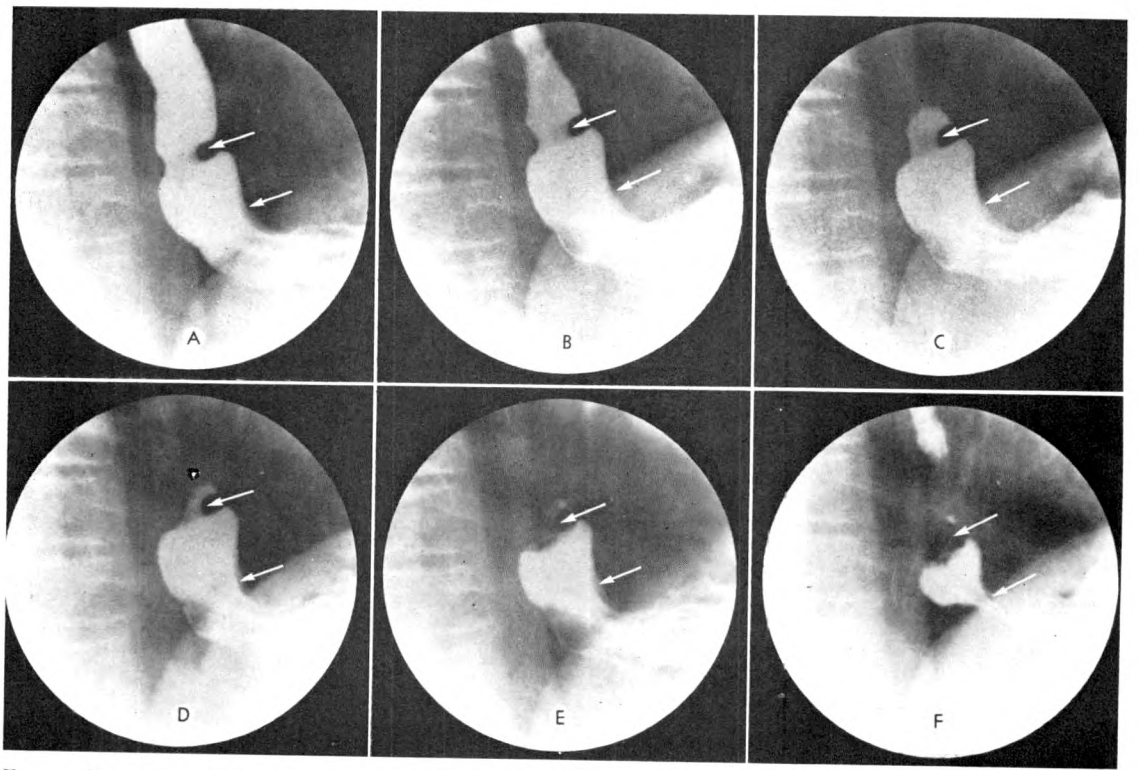


Fig. 4. Six 70 mm. spot roentgenograms of a patient with a small sliding hiatal hernia. In each frame, the upper arrow indicates the A level, the lower arrow the B level. The A level is identified by a functional ring; the B level is identified by faint profile notches and the proximal extent of thick folds.

Frame (*A*) is taken during continuous free flow. An A ring (upper arrow) persists. The submerged segment is effaced—actually widened and displaced upward through the hiatus. The segment between the arrows is the "vestibule" although it is not greatly different from the original phrenic ampulla. In Frame (*B*), the peristaltic wave is emptying the proximal portion of the esophagus. The barium column traversing the hiatus is slightly wider than in Frame *A*. In Frame (*C*), a cap is formed immediately above the A ring. This cap diminishes in size in Frame (*D*) but retains a drop of barium in Frame *E*. Frames (*E*) and (*F*) show contraction of the A ring. In Frame *F*, the herniated portion of the stomach retracts into the hiatal region and reconstitutes a narrow contracted submerged segment. The body of the esophagus, after relaxation of the peristaltic wave elongates, to permit this movement. Barium is temporarily retained between the A ring and the hiatus in a small sac which is lined by squamous epithelium and may be called vestibule or phrenic ampulla. The suprahiatal configurations in the previous frames should not be designated phrenic ampullae. This patient is asymptomatic since in the resting phase, no "hernia," either functionally or anatomically, is present. The appearances seen during swallowing—prominent A ring, excessive elevation and widening of the submerged segment—indicate loosening of the fascial attachments to the crura and widening of the hiatus, *i.e.*, minimal herniation.

terminal 4 cm. or so of the esophagus showed resting pressures higher than fundic pressure and that these pressures decreased to fundic pressure at the onset of a swallow. As catheters connected to pressure transducers are withdrawn from the stomach into the esophagus, resting pressure begins to rise promptly at the cardiac orifice (Fig. 5). It reaches a peak in the region of the hiatus but remains elevated above fundic pressure for a distance of about 2 cm. above the hiatus. This resting high pressure zone therefore straddles the hiatus, with supra- and infradiaphragmatic portions each about 2 cm. in length. It cannot be equated with a short ring-like contractile segment such as the inferior esophageal sphincter of Lerche. It must include a part, if not all, of the phrenic ampulla as well as the submerged segment (Fig. 5). Identification of these

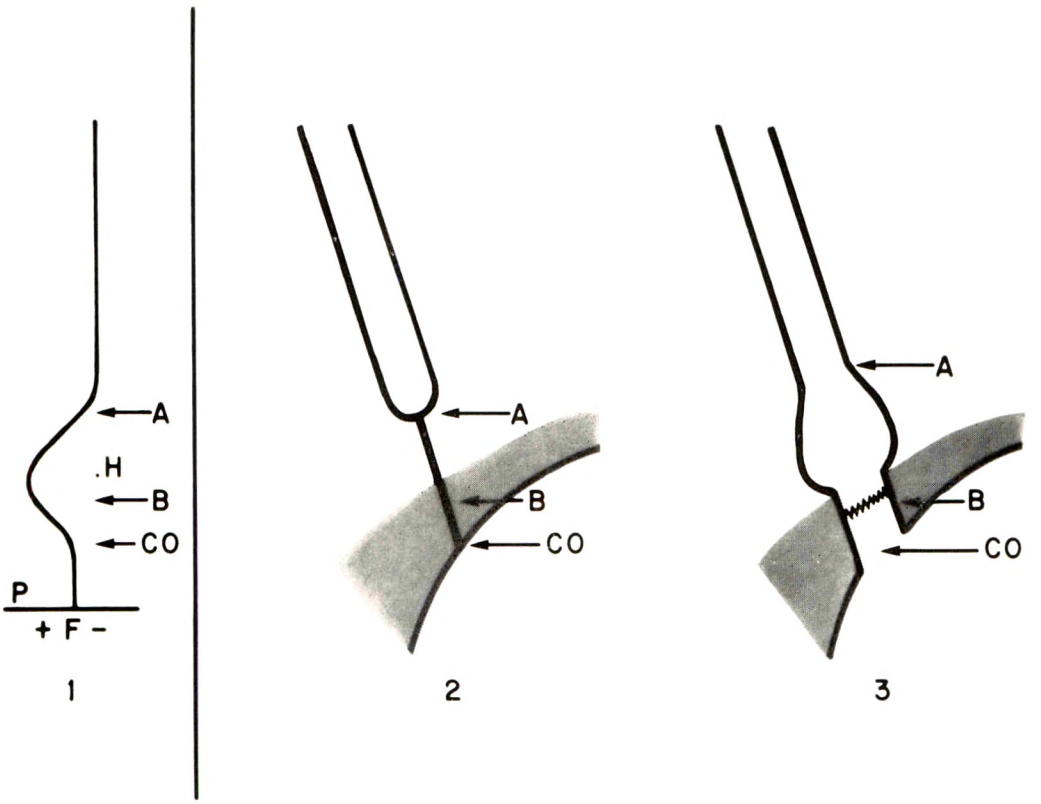


Fig. 5. Diagrammatic representation of the normal or Code esophageal sphincter.

Panel *1*. The resting pressure profile is indicated. "F" represents fundic pressure. The A and B levels as well as the level of the cardiac orifice (CO) and the hiatus (H) are indicated. Increasing pressure (P) is indicated toward the left. Resting pressure begins to rise at the cardiac orifice, reaches a peak in the region of the hiatus and decreases to intrapleural pressure level above A. Panel *2* illustrates an appearance rarely seen normally; *i.e.*, the arrival of barium prior to relaxation of the sphincteric zone which can then be seen to extend from a point about 2 cm. above the hiatus to the cardiac orifice. Panel *3*. The sphincteric zone relaxes and elongates during filling and the typical appearance of a phrenic ampulla and submerged segment is produced. The Z line adjacent to the B level is indicated but is not seen roentgenologically.

roentgenologic segments with the high pressure zone must not be taken literally since the sphincteric zone as such exists only in the empty or resting phase. Such correlations must be considered purely descriptive since, no doubt, during the course of filling and emptying, rearrangements of individual muscle fibers and up and down movement of the esophagogastric region in relation to the hiatus occur. Although the normal high pressure zone is often equated with the vestibule, this zone also includes the most distal portion of the submerged segment, that is, the segment surrounded by the sling fibers (Fig. 1*B*). These sling fibers are generally considered to be the most reliable anatomic landmark for the junction or demarcation between esophagus and stomach. Since they are usually described, however, as part of the stomach, the "sling fiber segment" must be similarly considered. This segment is in the nature of a gap in the wall of the stomach but roentgenologically has the configuration of a short tube and is not part of the sac. Another reason for including the distal portion of the submerged segment in stomach is the fact that the mucosal junction and the constrictor cardiae fibers are located above the sling fibers, that is, within the submerged segment and not at the cardiac orifice (Fig. 1*B*). Moreover, under certain

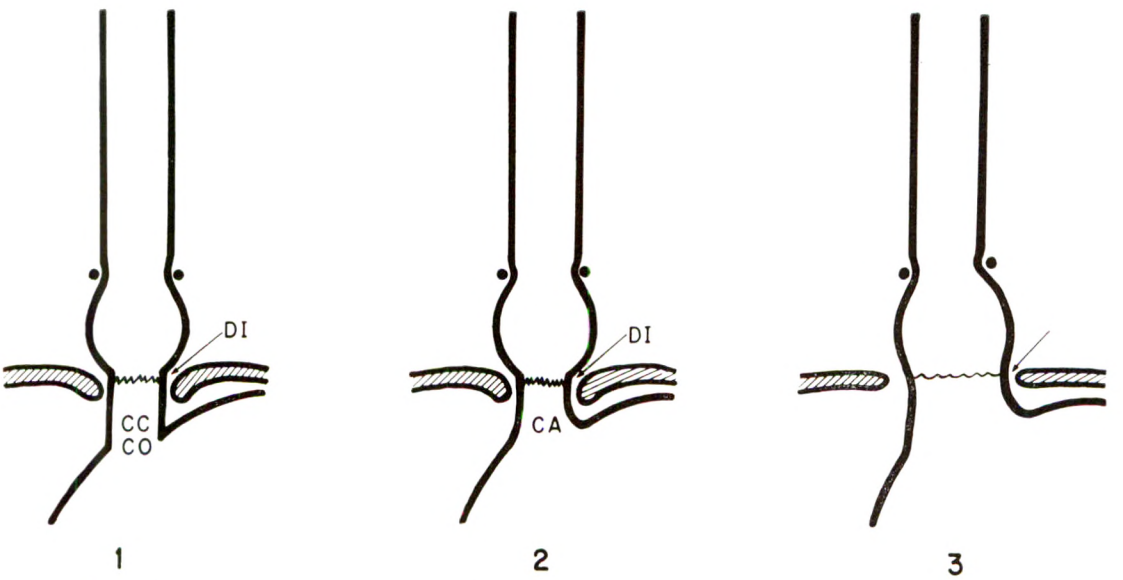


FIG. 6. "Cardiac canal" and "cardiac antrum."

Panel *1* shows the typical normal appearance. The submerged segment may be divided into an intrahiatal portion and a subhiatal portion (cardiac canal, CC) but there is no roentgen demarcation. Occasionally, Panel *2*, the subhiatal portion assumes a funnel-shaped configuration (cardiac antrum, CA) but the intrahiatal portion persists. This must be distinguished from a small hernia, Panel *3*, in which both portions are obviously widened. In Panels *1* and *2*, the caliber of the barium column below the diaphragmatic indentation (DI) is normal. In Panel *3*, the hiatus (arrow) is wide. The Z line is included in this illustration to indicate that "herniation" may occur with little proximal displacement even during a swallow. Note that, if the location of the Z line in the filled state is taken as the upper margin of the cardiac canal, this canal occupies most if not all of the submerged segment.

circumstances, the distal segment may distend and be incorporated into the body of the stomach (Fig. 6).[41] This change, from a short "cardiac canal" to a "cardiac antrum," is rarely seen when the esophagogastric region is normally located but is regularly observed when this region herniates. Normally, there is no roentgen landmark to indicate the junction between the cardiac canal and the remainder of the submerged segment. There is no single way of defining the upper margin of the "cardiac canal"—anatomically, mucosal junction or constrictor cardiae fibers, and functionally, the transition manometrically to a pressure profile of "pure relaxation" during the course of a swallow. Moreover, the fraction of the total length of the submerged segment occupied by the cardiac canal depends on the stage of filling (Fig. 7).[27] When the esophagus and esophagogastric region are completely distended, the mucosal junction

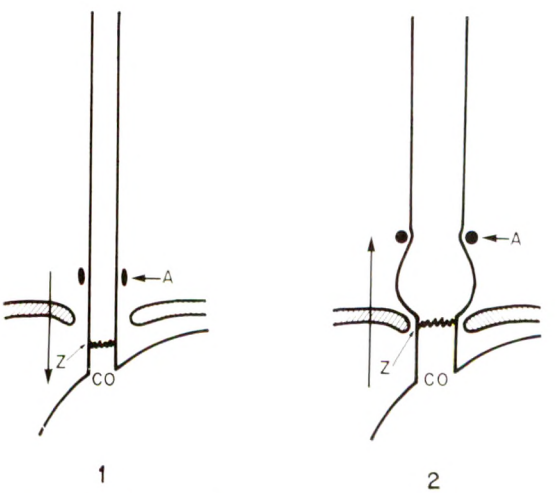


FIG. 7. Reciprocal changes in length of body of esophagus and esophagogastric region during filling and emptying.

Panel *1* and *2*. The esophagogastric region (vertical arrow) elongates during filling. The length of the submerged segment and the position of the cardiac orifice (CO) seem unchanged. However, the Z line and A level are elevated during filling.

appears to ascend to the level of the upper margin of the hiatus, although this no doubt varies to a limited degree from patient to patient. Whether constrictor cardiae fibers also participte in this up and down motion is not clear. Incidentally, the term "cardia" has become too confusing to use, since, while it originally referred to the cardiac orifice, it has also been applied to the entire submerged segment.

Since the manometric or Code sphincter described above is now generally accepted as the normal physiologic sphincter in the esophagogastric region, it is necessary to abandon the designation "inferior esophageal sphincter" for the functional ring at the junction of the tubular esophagus and the vestibule. It serves no useful purpose to argue that Lerche's term, inferior esophageal sphincter, for a contractile ring in this location has historic priority, or that this ring in patients with small hernias has sphincteric characteristics, both roentgenologically and manometrically.[16] Lerche's inferior esophageal sphincter has been referred to as the tubulo-vestibular or TV sphincter[41] to indicate this type of functional activity and to distinguish it from the Code inferior esophageal sphincter. However, the term "A" ring is probably more suitable until a generally accepted definition of a sphincter is available.

The reason why the designation inferior esophageal sphincter for the "A" ring has persisted in the radiologic literature is not difficult to discover. An A ring is often seen in patients with small hernias as part of the associated functional abnormality (Fig. 8, *A–C*; 9, *A* and *B*; and 10). However, the inferior esophageal sphincter of Code is rarely evident roentgenologically. This is to be expected since this zone normally relaxes prior to the arrival of a barium bolus. There might, however, be some additional feature of the normal high pressure zone, such as limited distensibility, which would serve to identify it. Partly on such an assumption, the submerged segment[6,24,26] has been equated with the "inferior esophageal sphincter." Manometric studies combined with the use of radiopaque markers on the hiatus[5,10,40] indicate, however, that the relative lack of distensibility of the submerged segment is related to its location in and below the hiatus. Residual "tone" or incomplete relaxation may also play a role

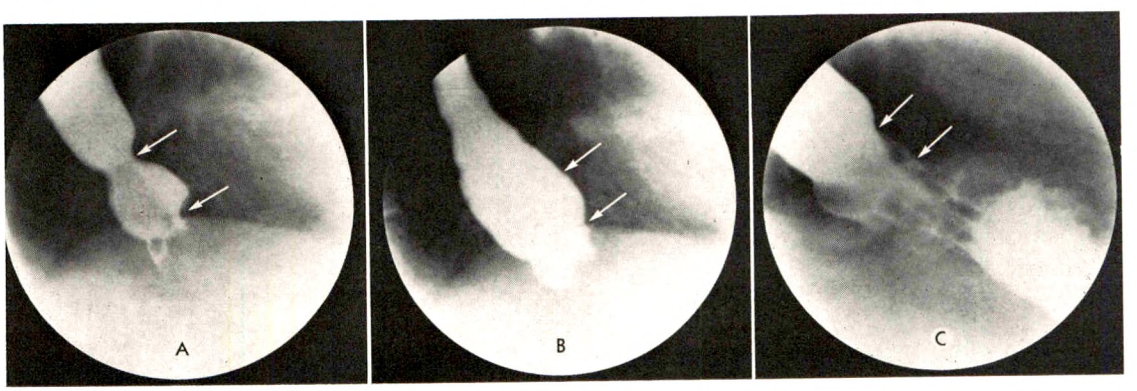


Fig. 8. Three 70 mm. frames from a patient with a small sliding hiatal hernia.

Frame (*A*) during the course of filling shows a distinct A ring (upper arrow) often designated in the radiologic literature as "the inferior esophageal sphincter." Distal notches (lower arrow) indicate the B level. In Frame (*B*) the distal end of the esophagus is maximally distended and there is no evidence of an A ring. The upper arrow indicates its approximate location on previous frames. Notches are still present distally but appear less marked. In contrast to a circumferential web-like ring at this level, such notches change in configuration (constrictor cardiae fibers?) but never form a complete ring which divides the barium column. In Frame (*C*) the A and B levels are again indicated (arrows). A transverse somewhat arcuate linear barium collection (lower arrow) at the B level marks the proximal extent of thick persistent folds. The fold pattern within the gastric channel in the hiatus was never completely effaced indicating limited distensibility.

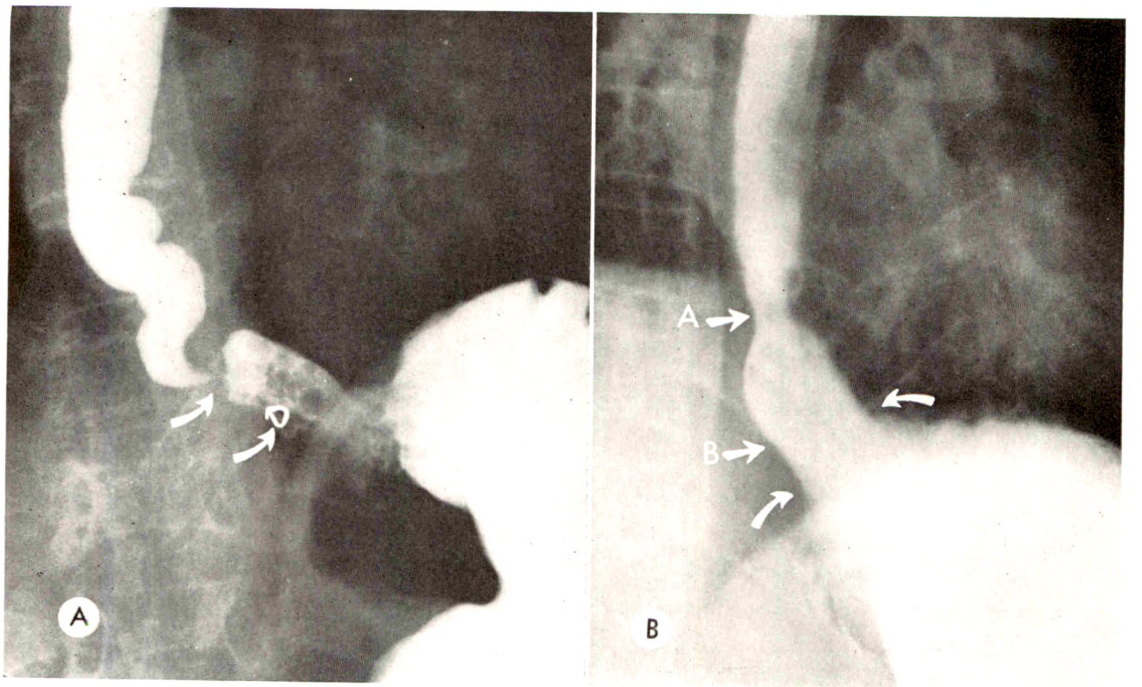


Fig. 9. Patient with small sliding hernia.

(*A*) Roentgenogram taken 3 hours after esophagoscopy at which an opaque clip (lower arrow, retouched) was placed on the squamo-columnar mucosal junction. No distal ring is present in this patient. The A ring (upper arrow) is located between 1 and 2 cm. above the mucosal junction. Tertiary contractions in the body of the esophagus are presumably related to the trauma of esophagoscopy. No discrete contractile activity at the level of the clip is present. (*B*) Roentgenogram taken prior to esophagoscopy shows a wide barium channel traversing the region of the hiatus (between unlabeled arrows) with absence of the submerged segment. The A level is labeled. The approximate location of the squamo-columnar mucosal junction, as determined subsequently by placement of the clip shown in *A* is indicated by arrow "B." The configuration shown on this roentgenogram would be designated as a small sliding hiatal hernia with little question, although the assumption is usually made that the hernial sac begins at the A level. In truth, the proximal portion of this sac is lined by squamous epithelium and must be equated with the original phrenic ampulla or supradiaphragmatic portion of the vestibule. The amount of stomach above the hiatus is minimal and represents the original submerged segment.

but is difficult to demonstrate. When the submerged segment is displaced above the hiatus, it appears to lose sphincteric characteristics particularly in comparison with adjacent segments above and below it (Fig. 11). In fact, this is one of the chief functional features of hiatal herniation. It has also been suggested that the submerged segment, while only the distal portion of the normal high pressure zone, is the true sphincteric region.[10,11] This suggestion was based on the observation that a barium bolus is frequently delayed at the level of the hiatus before entering the submerged segment and the stomach. Intraluminal pressure in the submerged segment, however, usually decreases to fundic level before barium reaches the hiatus. It is likely that delay at the hiatus is caused by another mechanism. Despite relaxation of the high pressure zone, a bolus cannot be delivered into the relatively high pressure abdominal compartment until a contraction behind the bolus occurs and is sufficiently energetic to propel it into the abdomen. In patients with no peristaltic activity in the body of the esophagus, a bolus does not readily enter the abdomen even though the sphincteric zone is absent or destroyed (scleroderma, treated achalasia). In the erect position, gravity is of assistance but, by itself, is not sufficient to

empty the esophagus. Nevertheless, there is no doubt that the submerged segment is the most important part of the normal antireflux mechanism. It is the area directly and continuously exposed to intragastric pressure. Additional protection for this segment is furnished by the cardiac rosette and the cardiac incisura. The integrity of the rosette and the incisura requires normal anatomy and normal function of the submerged segment. It is true that whenever, on roentgen examination, no submerged segment can be demonstrated, a hernia, at least functionally, is present (Fig. 6, Panel *3*). Reflux can be anticipated under these circumstances. Moreover, the best sign of a successful hiatal hernia repair is the reconstitution of a submerged segment. A normal submerged segment on roentgen examination signifies that a normal inferior esophageal or Code sphincter will be found on manometric study. Because of this, in practice, no significant error is made if the radiologist thinks of the submerged segment as the sphincteric region, provided the qualifications noted above are kept in mind. In the same way, in persons with sliding hernias, one may think of the sphincteric region as consisting of the A ring with a portion of adjacent vestibule, provided it is understood that this region is *not* the original submerged segment or the original sphincteric zone.

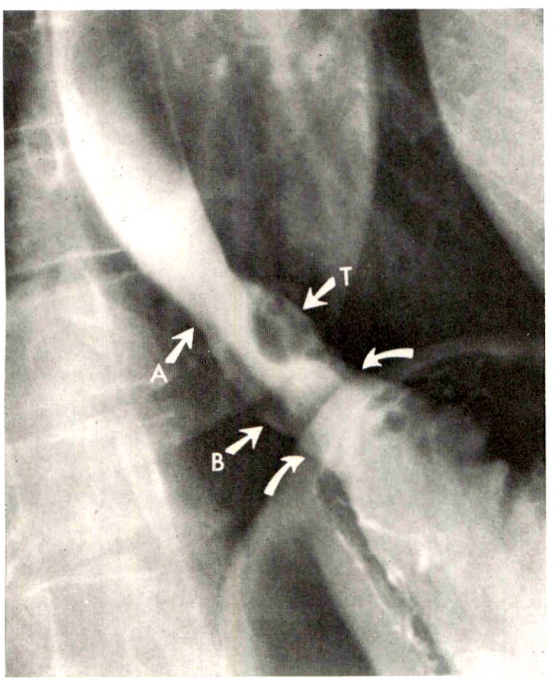


Fig. 10. Another patient with a small sliding hiatal hernia indicated by the wide barium column (between unlabeled arrows) traversing the region of the hiatus and by absence of the submerged segment. The appearance is similar to that shown in Figure 9*B*. The end of the tubular esophagus (A) can be identified. In this patient, a small intramural tumor covered by intact squamous epithelium was confirmed at esophagoscopy. The squamo-columnar junction was located at the distal margin of the mass. The B level or Z line (arrow B) is therefore located at least 2 cm. distal to the A ring.

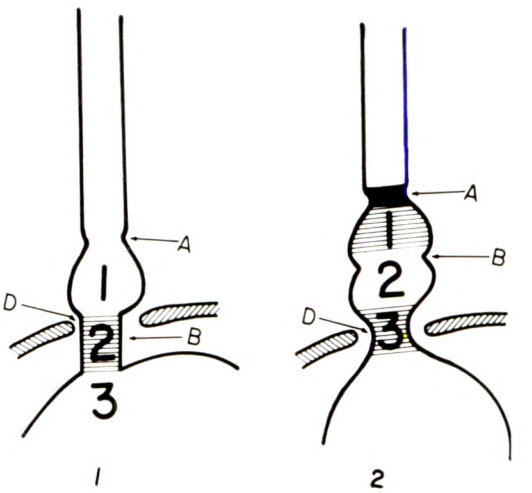


Fig. 11. Diagrammatic representation of functional changes occurring as part of hiatal herniation. A, B and hiatal (D) levels are indicated as well as the regions of the phrenic ampulla (1), submerged segment (2) and body of the stomach (3).

Panel *1*. Normally, the most important part of the pressure barrier is the submerged segment (shaded). Panel *2*. In a typical small sliding hernia, the most prominent "sphincteric" region may be an A ring (solid black). The original ampullary region (1), now designated the vestibule, shows variable elevated resting pressure (shaded) as does the cuff of stomach (3) enclosed within the hiatus (shaded). The original submerged segment (2) shows relatively little "sphincteric" activity.

Although the normal Code sphincter is not evident roentgenologically, evidence for its presence can occasionally be ob-

tained. If a fluid bolus reaches the distal esophagus unusually promptly, transient delay to the passage of the bolus may occur at a point about 2 cm. above the hiatus. Distal flow is rapidly resumed, although, as noted above, more prolonged delay may occur at the hiatus. The best roentgenologic evidence for the existence of the inferior esophageal sphincter is the well-known observation that, in cases of achalasia, a segment about 4 cm. in length which straddles the hiatus fails to open normally (Fig. 12). It often does fill incompletely and fleetingly when the patient is asked to swallow. These roentgenologic features correspond to the manometric findings characteristic of achalasia; *i.e.*, a sphincteric zone which fails to relax normally and contracts prematurely. In late cases of achalasia, however, the progressive dilatation of the body of the esophagus extends into the sphincteric zone and this region may become quite short. It is possible that there are cases of "hypertensive sphincter" in which the esophagogastric region may resemble that seen in achalasia but with maintenance of normal peristaltic activity in the body of the esophagus. This combination has not been convincingly documented. Cases demonstrated manometrically to have hypertensive sphincters have usually been associated with small hiatal hernias[16,20] or with a diffuse functional abnormality of the esophagus.[15]

The function of the A ring under normal circumstances is difficult to discern since it is rarely seen as such. It may serve to close the phrenic ampulla temporarily while the ampulla empties its contents through the hiatus. A normal A ring (Fig. 13, *A–D*) may become evident during the course of a pinchcock maneuver when barium refluxes back into the body of the esophagus. Occasionally, an A ring may be seen fleetingly during filling (Fig. 13, *A–D*) and sometimes persists as a shallow indentation at the end of the tubular esophagus. However, a persistent or prominent A ring is not a normal finding. It is surprising that more attention has not been paid to such A rings since they occur in a variety of conditions (Fig. 14, *A–D*).[42] There appear to be several reasons for this. If interpreted as a physiologic phenomenon, *i.e.*, the inferior esophageal sphincter, it is easy to assume that no special comment is required particularly if the ring appears helpful in preventing reflux. Moreover, an A ring always relaxes to some degree although with varying frequency. Since it is rarely the cause, by itself, of significant dysphagia or esophageal pain, failure to note it rarely leads to difficulty. There is also reluctance in accepting the fact that there are two potential rings in the esophagogastric region with entirely different characteristics. The designation, "lower esophageal ring," for the distal Schatzki type of ring seems to presume that only one ring in this area is of importance. Confusion is compounded when the distal ring is called the "lower esophageal contractile ring"[21] or the "lower esophageal (contractile) ring"[36] since this ring has no independent contractile activity. A stenotic

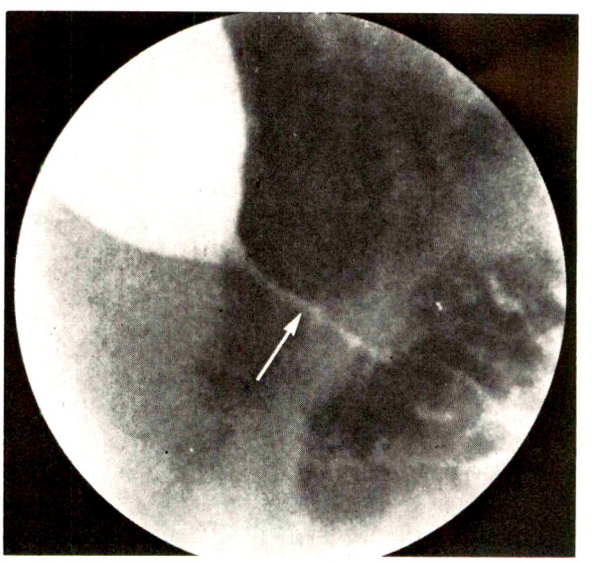


Fig. 12. Patient with typical achalasia shows a narrowed segment 4 or 5 cm. in length which never distended normally (arrow). This segment corresponds to the normal or Code inferior esophageal sphincter and to the manometric findings of failure of relaxation.

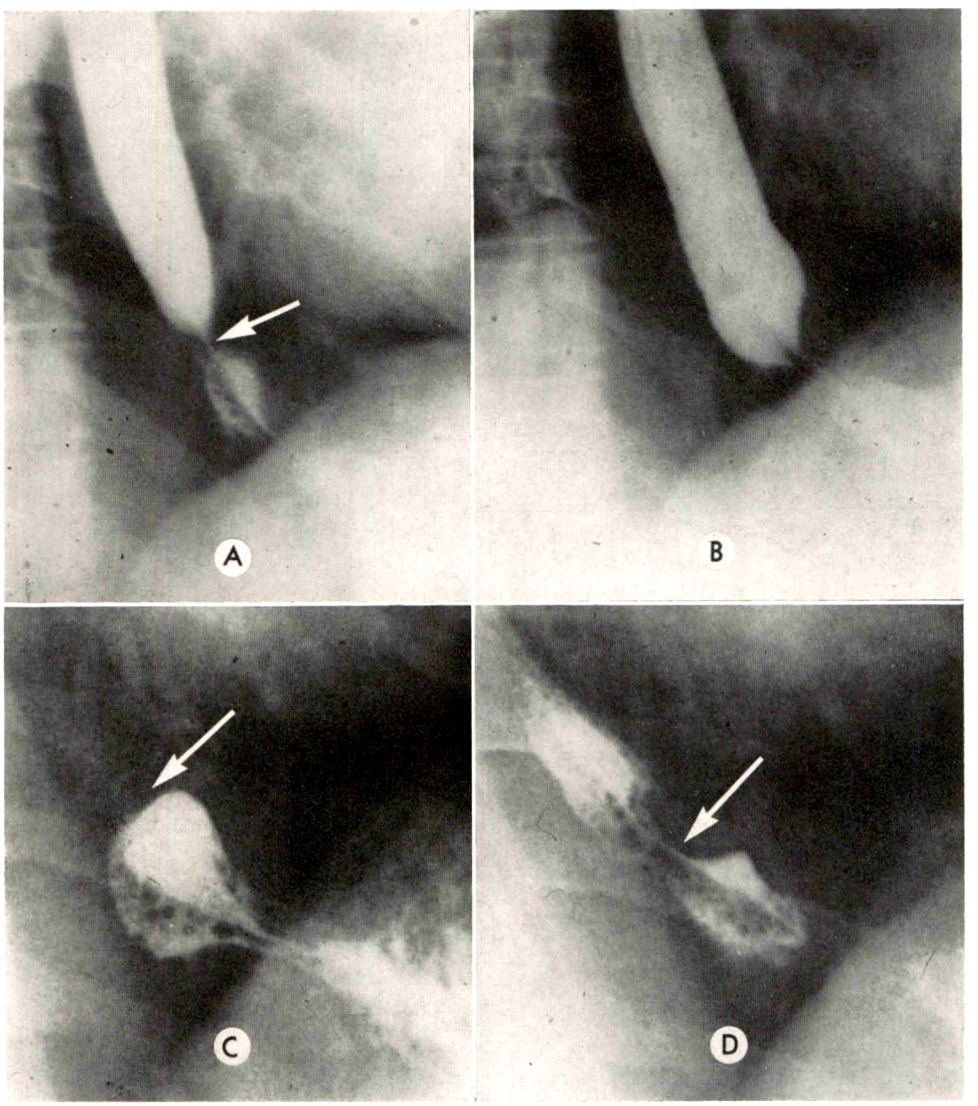


FIG. 13. Four 70 mm. frames in a patient without a hiatus hernia but with evidence of A level activity. Patient was prone.

Frame (*A*) shows the appearance during early filling. A contraction (arrow) is present at the A level. This ring promptly disappeared. Frame (*B*) shows the appearance during complete filling with no evidence of the ring and no evidence of a hernia. A submerged segment is present. Frame (*C*) was taken at the end of emptying of the tubular esophagus. Barium is retained between the contracted A ring (arrow) and the submerged segment within the hiatus. Shortly after this, Frame (*D*), a small amount of barium refluxed into the body of the esophagus. The contraction (arrow) demonstrated in this fashion extends both proximally and distally to the A level.

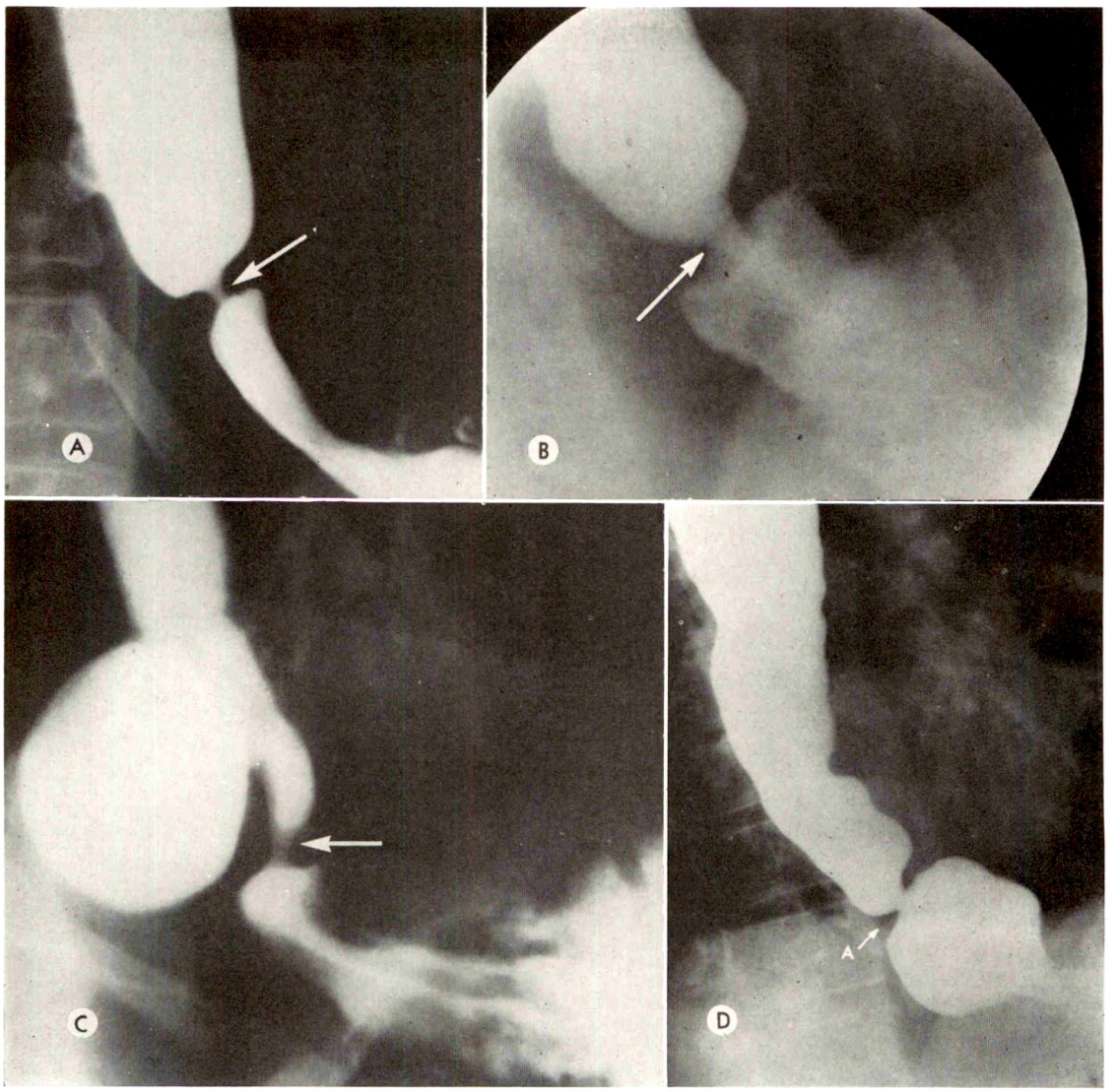


Fig. 14. Four examples of symptomatic A rings.

(*A*) A marked contraction at the A level (arrow) which, however, relaxed and disappeared. This patient complained of occasional dysphagia. A small hernia is presumably present. (*B*) Another patient with a prominent A ring (arrow) which distended to a normal caliber. This patient had 3 episodes of bolus obstruction but was symptom free in the intervals. A small hernia, otherwise asymptomatic, is present. (*C*) Patient with a large epiphrenic diverticulum with occasional dysphagia. A prominent A ring (arrow) as well as a small hernia is present. Peristaltic activity was present in the body of the esophagus. (*D*) A patient, 52 years old, with severe dysphagia but no pain. A persistent A ring (arrow) which never completely distended was demonstrated. Peristaltic activity in the body of the esophagus was markedly diminished although clinically there was no evidence of scleroderma. Tertiary waves did not appear forceful and were present only with maximum distention. No esophagitis or organic stricture was seen on esophagoscopy. Forceful dilatation of the esophagogastric region relieved the patient's symptoms dramatically.

Schatzki ring is organic and can be observed endoscopically and at surgery, while a functional ring is difficult to define at esophagoscopy and may disappear completely under anesthesia or after death. The concept that the A ring is not a discrete entity but a peristaltic wave which travels through the vestibule and finally merges into the distal ring has also been suggested.[8] However, the A ring cannot possibly be a peristaltic wave since it is seen also during filling and recedes proximally from the B level as the vestibule fills. The apparent movement of the A ring toward the B level is due to shortening of the vestibule during emptying which may indeed be so marked as to suggest that the vestibule has disappeared.

The most easily recognized role of an A ring is as a compensatory, substitute, or residual antireflux mechanism in patients with hiatal hernias. Pressure profiles in patients with hiatal hernias[1,9,16,20] are quite variable and not quantitatively predictable (Fig. 15; and 16). A common feature is a short zone of elevated resting pressure at the A level which relaxes in anticipatory fashion at the onset of a swallow. However, relaxation at this level may be delayed, quite transitory or show a prolonged after-contraction. The pressure barrier related to the A ring is rarely a potent antireflux mechanism and is often overcome if there is free communication between the hernia and the stomach below the diaphragm and if intraabdominal pressure is artificially increased. Reflux of barium through an A ring is common if a patient is asked to swallow a mouthful of water. This water swallowing or siphonage test demonstrates that some intrinsic sphincter activity is present which can be reflexly overcome. A roentgenologically "spastic" A ring, *i.e.*, a ring which shows delayed incomplete opening and premature contraction, might serve as an effective antireflux mechanism but would likely cause dysphagia. If combined with excessive resting pressure ("true" spasm) demonstrable manometrically, esophageal pain might result. Dysphagia may be present as a result of a "spastic" A ring but is rarely marked unless peristaltic activity in the body of the esophagus is diminished or disordered.

A surprising observation on both roentgenologic and manometric studies is that the most effective antireflux mechanism available to patients with sliding hiatal hernias is the presence of a collar of stomach within the hiatus which cannot distend normally. Such a narrowed, often slit-like gastric channel occupying the region of the hiatus is frequently seen in patients with sliding hernias.[31] Manometrically, a "submerged" gastric channel or collar of this kind is the site of elevated resting high pressure which diminishes to fundic pressure during fluid filling (Fig. 15; and 16). A compressed portion of the stomach has become "sphincteric." Moreover, an artificial sphincteric zone may be created surgically by fundoplication or an "inkwell" operation which succeeds in reproducing a compressible and contractile tubular segment.[25] The result is independent of the nature of the lining epithelium. The mechanism is not entirely clear but probably represents a nonspecific response of the smooth muscle of the muscularis propria to extrinsic restraints although a "choke valve" effect[7] may also be important. It is not simply the result of direct pressure by the crura of the diaphragm. This phenomenon indicates that the assumption of the presence of a preformed "sphincter" purely on the basis of manometric features must be considered suspect. It also suggests that the normal inferior esophageal sphincter results from a combination of intrinsic and extrinsic factors. It is therefore not surprising that, as a result of herniation, different antireflux mechanisms, both extrinsic and intrinsic, may come into play. Little attention has been paid to this possibility in reports of manometric studies. Any zone which relaxes on swallowing is frequently designated as the inferior esophageal sphincter without further comment. When two

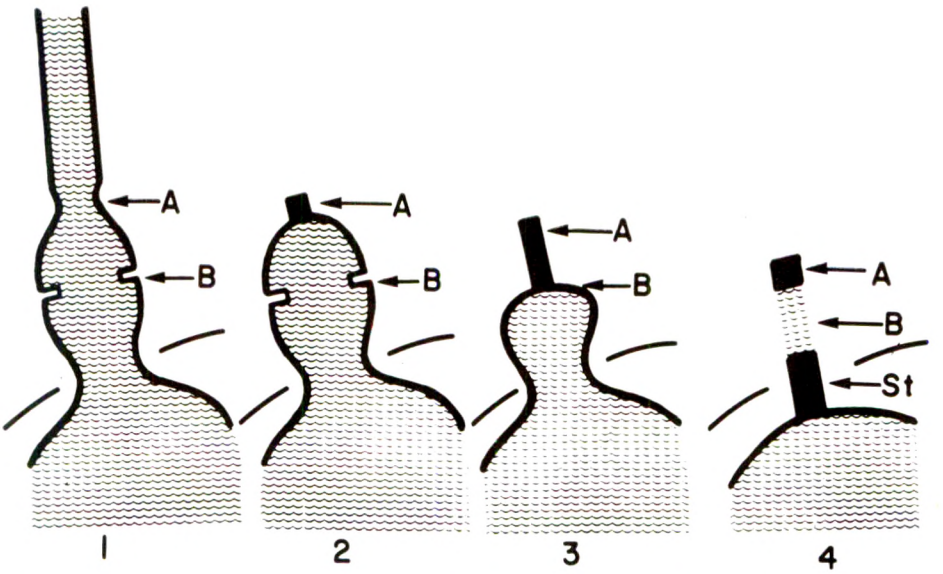


Fig. 15. Diagrammatic representation of emptying phases in a patient with a small hernia and a double peak of resting high pressure.

Panels *1–4*. A and B levels are indicated. Fundic pressure, shaded; pressure above fundic pressure, solid black. With free flow of fluid, pressure throughout the column is equal to fundic pressure. Panel *2* shows contraction at the A level which closes off the distal end of the tubular esophagus. Panel *3* shows emptying of the vestibule between the A and B levels. In the resting phase (Panel *4*), a short high pressure zone is found proximally and a second high pressure zone distally. The proximal zone is related to the A ring with a questionable amount of proximal vestibule. The distal zone is related to a collapsed portion of the stomach (St) within the hiatus. The segment between the two high pressure zones shows resting pressure approximately equal to fundic pressure and includes herniated stomach and distal vestibule.

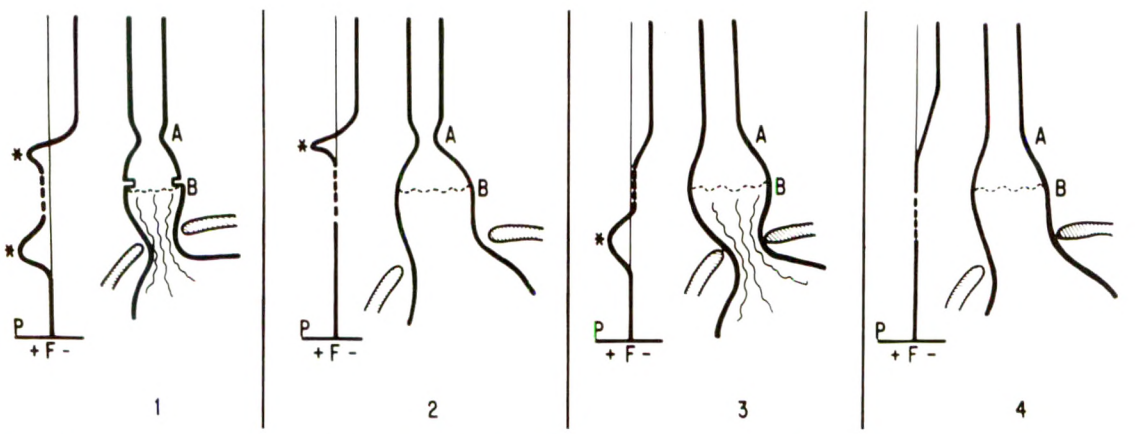


Fig. 16. Diagram illustrating a variety of pressure profiles in patients with sliding hernias.

Panels *1–4*. A and B levels are indicated in each panel. F indicates fundic pressure; pressure (P) higher than F is on the plus (+) side, lower than F on the minus (−) side. Peak pressures are indicated by asterisks (*) on the pressure curves. The length of plateau region varies (dashes) depending on the size of the hernia. Panel *1* shows two peaks of resting pressure, one in the region of the A ring and another at the level of the hiatus. Panel *2* shows one peak related to the A level and a wide barium column traversing the hiatus. Panel *3* shows a single peak in the region of the hiatus with a relatively narrowed gastric channel or collar within the hiatus. Panel *4* shows no peak and a continuous wide barium channel extending from the tubular esophagus into the stomach below the diaphragm. A "B" ring is shown in Panel *1* but may or may not be present in any of these configurations. The Z line, although indicated, is not evident roentgenologically.

such zones are present, the proximal one is assumed to be the inferior esophageal sphincter although its characteristics, for example, its length, are clearly different from the normal sphincter zone. If only one zone is present, it is not always stated whether it is located at the proximal or distal end of the hernial sac. Moreover, on pressure tracings of patients with sliding hernias, the point of respiratory reversal, which normally identifies the hiatal level, may be single or double. If single, it may be located either at the hiatal level or at the proximal margin of the hernia—in each case, corresponding to the location of a high pressure zone. These distinctions are not always made clear in pressure studies, particularly when suitable roentgen studies are not available. Cine studies in the early phases of filling and the last phases of emptying are necessary in efforts to extrapolate roentgen features to resting pressures. It is also important in attempting such correlations to consider the techniques of pressure measurement since these measurements do not correspond to a point and may depend on the state of contraction of adjacent segments.[29,36] At any rate, further observations will be necessary before the roentgen characteristics of hypertensive, hyperacting and hypercontracting sphincters[14] can be delineated.

SUMMARY

1. The term "inferior esophageal sphincter" for a contractile ring at the end of the tubular esophagus was introduced into the radiologic literature as a result of the dissections and descriptions of Lerche. This correlation was based on observations in patients with small sliding hernias rather than on appearances in normal individuals. Subsequently, it was demonstrated that the normal antireflux mechanism is not a ring-like segment above the diaphragm but a zone about 4 cm. in length which straddles the hiatus. The term inferior esophageal sphincter is currently accepted for this region or zone of resting high pressure and therefore should not be used for a discrete ring at the end of the tubular esophagus.

2. There are two potential lower esophageal rings—one functional and the other static. The functional ring is located at the end of the tubular esophagus. The static ring is located more distally adjacent to the squamo-columnar mucosal junction.

3. A prominent or persistent contractile ring at the end of the tubular esophagus is an abnormal finding and frequently a manifestation of a small hernia. It is also a common feature of a variety of functional abnormalities. In the presence of herniation, it may serve as a rather ineffective antireflux mechanism but may also become "spastic" and cause periodic dysphagia.

4. The terms phrenic ampulla, submerged segment, cardia, cardiac canal, cardiac antrum, A and B levels, and vestibule are discussed.

5. Inconsistencies in the use of the term "sphincter" both roentgenologically and manometrically are pointed out.

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ROENTGENOGRAPHIC APPEARANCE OF SYMPTOMATIC LOWER ESOPHAGEAL RINGS AFTER PNEUMATIC DILATATION*

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SYMPTOMATIC lower esophageal rings have been treated successfully in several ways. The ring has been excised or digitally ruptured and the hiatus hernia repaired. Simple bougienage and pneumatic dilatation have been used. Dietary education has improved symptoms. We have treated 10 patients with a Mosher dilator and made roentgenographic observations before and after dilatation.

DESCRIPTION OF THE RING

Schatzki and Gary's[7] criteria for the diagnosis of a lower esophageal ring were used. They described a thin ring-like narrowing in the lower esophagus, present constantly, not changing in position and only visible when the esophagus above and below it is filled enough to dilate to a greater caliber than the diameter of the ring. The thin diaphragm protrudes into the lumen in a plane perpendicular to the longitudinal axis of the esophagus, varies from 2–4 mm. in thickness and usually has a sharp, nearly right angle between the surface of the ring and the wall of the esophagus. It occasionally appears somewhat flexible and solids or large amounts of barium may push the free edge downward. The diameter of the ring measures essentially the same on all roentgenograms of a given examination where maximum distention of the esophagus has been obtained. The best position to visualize the ring is with the patient prone and partially turned on his right side since it is usually not well demonstrated in the upright position. It is best seen after a large bolus of barium is swallowed and the patient takes a deep breath just before the barium reaches the diaphragm. Gastroesophageal reflux is usually not present.

MATERIAL

The age and sex of the 10 patients are shown in Table 1 with the size of the ring prior to dilatation and at various intervals following dilatation. The rings measured 4 mm. to 15 mm. in diameter prior to dilatation. All patients complained of dysphagia with bulky foods for several months to several years. In some the dysphagia was progressive and occasionally aggravated by emotional stress. One patient complained of heartburn in addition to dysphagia (Case 2). All patients underwent esophagoscopy with the area of narrowing only occasionally identified. In some instances inflammatory changes of the mucosa at the esophagogastric junction were present. The duration of follow-up ranged from 5 months to 1½ years.

METHOD

After sedation with demerol and atropine and local anesthesia of the pharynx, a Mosher dilator was advanced into the stomach and partially withdrawn under fluoroscopic control. The bag was then gently inflated and the position adjusted until the indentation seen marking the site of the lower esophageal ring was approximately centered. The pressure was then increased to 12–15 pounds per square inch and held for 1 minute. Figure 1, *A–D*, (Case 8) shows 4 spot roentgenograms of the same patient obtained during pneumatic dilatation. As the bag was inflated the indentation disappeared. The bag was deflated and subsequently reinflated to

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TABLE I

SUMMARY OF CASES

Patient	Age	Sex	Date of Dilatation	Size Pre-dilatation (mm.)	Size (mm.) at Follow-up Postdilatation							Fig.
					Immed.	24 hr.	4 mo.	7 mo.	12 mo.	18 mo.	24 mo.	
1. L.F.	65	M	8/25/67	10		18	11			10		
2. C.F.	28	M	8/25/67	10		15		14	11	10		12, 13
3. H.K.	55	M	9/ 7/67	9		15	15			13		
4. C.K.	47	M	6/ 1/68	15		22	18	18				5
5. L.P.	68	F	6/14/68	13		21	17	17				2
6. M.Y.	53	F	6/21/68	13	24	20		19				3
7. H.P.	65	F	8/ 7/68	11	21	22		15				4
8. V.E.	60	F	8/28/68	11		13	13					1
9. B.B.	43	F	10/18/68	12		22	21					
10. F.A.	41	M	7/12/66*	4		9		7			5	6–10
			10/20/68†	5		16	14					11

* Mercury bougienage.
† Mosher pneumatic dilatation.

15 pounds per square inch pressure. On the second inflation, there was little or no indentation of the bag demonstrable. The patient may feel a mild to moderate stretching discomfort beneath the lower sternum during dilatation. The dilator was usually coated with a small amount of blood and mucus when it was removed but no complications were observed.

Multiple spot roentgenograms of the lower esophagus were obtained before dilatation and at each of the follow-up examinations. Although the ring is a relatively fixed structure, slight variations

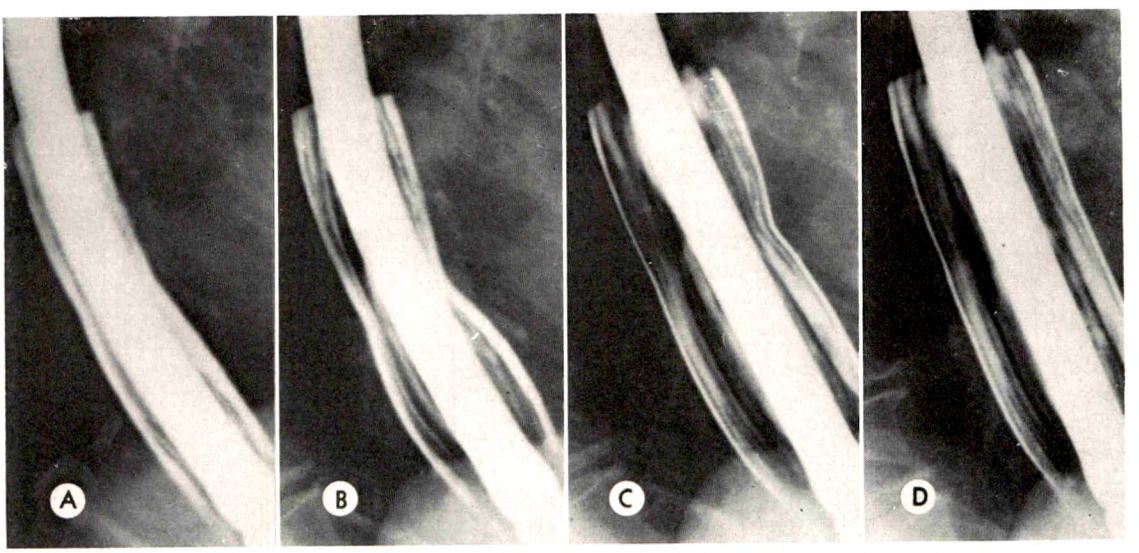


FIG. 1. Patient 8. Spot roentgenograms obtained during inflation of Mosher dilator. (*A*) 3 lb./in.^{2} pressure. (*B*) 8 lb./in.^{2} pressure. (*C*) 10 lb./in.^{2} pressure. (*D*) 15 lb./in.^{2} pressure. The indentation present at the site of the ring is gradually effaced and has disappeared on the final roentgenogram. The inflated balloon measures 4 cm. in diameter.

(1–2 mm.) in size may occur on a single examination. Several roentgenograms were necessary to be certain that the maximum diameter had been demonstrated. All measurements were taken on spot roentgenograms with no correction made for magnification.

RESULTS

Roentgenograms after dilatation showed that all rings increased in diameter. This ranged from 2 mm. to 11 mm. When the luminal cross-sectional area of the ring was calculated ($\pi\frac{1}{2}d^2$), the increase was considerable. For example, a ring that increased in diameter from 12 mm. to 16 mm. actually enlarged the area from 36π to 64π, an increase of 78 per cent. A tenfold increase in area occurred in Patient 10 when his ring increased from 5 mm. to 16 mm.

All patients had evidence of a hiatus hernia below the ring. This is consistent with the general acceptance of the lower esophageal ring as the site of the esophagogastric junction.

Esophagograms obtained shortly after dilatation showed that the ring may become much wider or completely disappear.

Figure 2*A* (Case 5) shows a 13 mm. ring prior to dilatation. The ring was no longer visible 7 hours after dilatation (Fig. 2*B*). In its place were irregular broad projections of barium. On either side of these were flat indentations on the barium column where the esophagus lacked distensibility of the magnitude present above and below. The projections of barium could be due to a mucosal laceration in the esophagogastric region and the indentations may represent the remaining tissue of the ring which has been pushed aside. The same patient 24 hours after dilatation is shown in Figure 2*C*. The projections of barium were no longer present and the ring had reappeared although it was broader and wider than before dilatation, measuring 21 mm. in diameter. Three months later the lower esophageal ring measured 17 mm. in diameter and had returned to a typical configuration. Seven months after dilatation (Fig. 2*D*), the ring still measured 17 mm. in diameter.

Figures 3, 4 and 5 show pre- and post-dilatation esophagograms of 3 different patients. In one of these (Fig. 3, *A–D*), the ring completely disappeared after dilatation; in another (Fig. 4, *A–C*), the ring became wider but was still present; in the third (Fig. 5, *A–C*), the ring became eccentric after dilatation. In all 3 patients, the ring ultimately assumed its predilatation configuration and maintained a diameter larger than before dilatation.

Two patients are of particular interest. The first is a 39 year old white male (Case 10) with dysphagia of 8 years' duration. The dysphagia gradually progressed from large pieces of food to liquids. He had no other gastrointestinal symptoms and the past history was noncontributory. When first seen on June 14, 1966 he had a lower esophageal ring measuring 4 mm. in diameter and 6 mm. in thickness (Fig. 6). Schatzki[8] concurred that this was a lower esophageal ring. During July 1966, the esophagus was dilated to 42F with composition and mercury bougienage. The patient's symptoms improved markedly and he was able to eat solid foods with little difficulty. One month later, his lower esophageal ring measured 9 mm. in diameter (Fig. 7). Over the next 2 years the ring gradually reduced to 5 mm. in diameter (Fig. 8) and the symptoms became worse.

In August 1968, he was readmitted to the hospital with the plan to dilate the lower esophagus by progressive mercury bougienage to a caliber large enough to admit a Mosher dilator. During the mercury bougienage severe muscular spasm occurred and the dilator was removed with difficulty. Four hours later the patient complained of upper chest and right neck pain. Roentgenograms of the neck revealed a large amount of retropharyngeal air (Fig. 9). Water soluble contrast material demonstrated a perforation in the right posterolateral wall of the esophagus, just below the cricopharyngeus muscle (Fig. 10). Within 3 hours, a 2 cm. laceration in this location was closed surgically and recovery

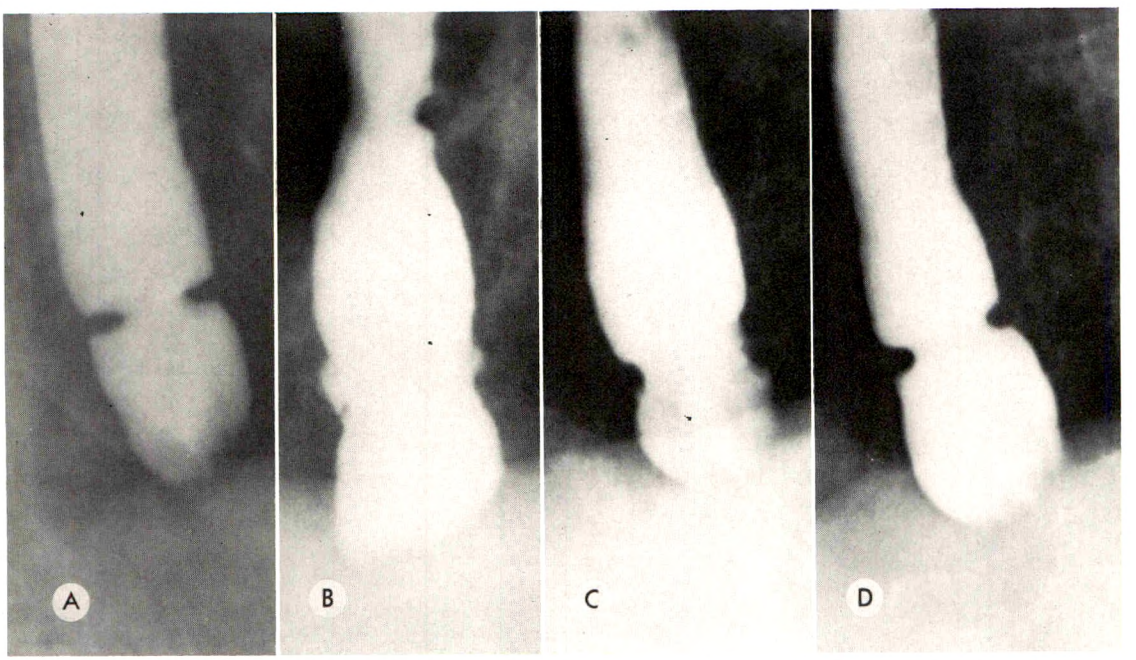


Fig. 2. Patient 5. (*A*) Predilatation: 13 mm. (*B*) 7 hr. after dilatation: 20 mm. Note projections of barium at level of ring. These probably represent mucosal lacerations. Curling of esophagus more cephalad. (*C*) 24 hr. postdilatation: 21 mm. Barium projections no longer apparent. Broad, wide ring. (*D*) 7 mo. postdilatation: 17 mm. Typical configuration of ring. Symptoms have not recurred.

was uneventful. Eleven days later, the lower esophageal ring measured 7.5 mm. in diameter, having increased from 5 mm. during the aborted mercury bougienage. Two months later successful dilatation of the ring was accomplished with a Mosher dilator. Figure 11, *A* and *B*, shows the appearance 24 hours and 2 months fol-

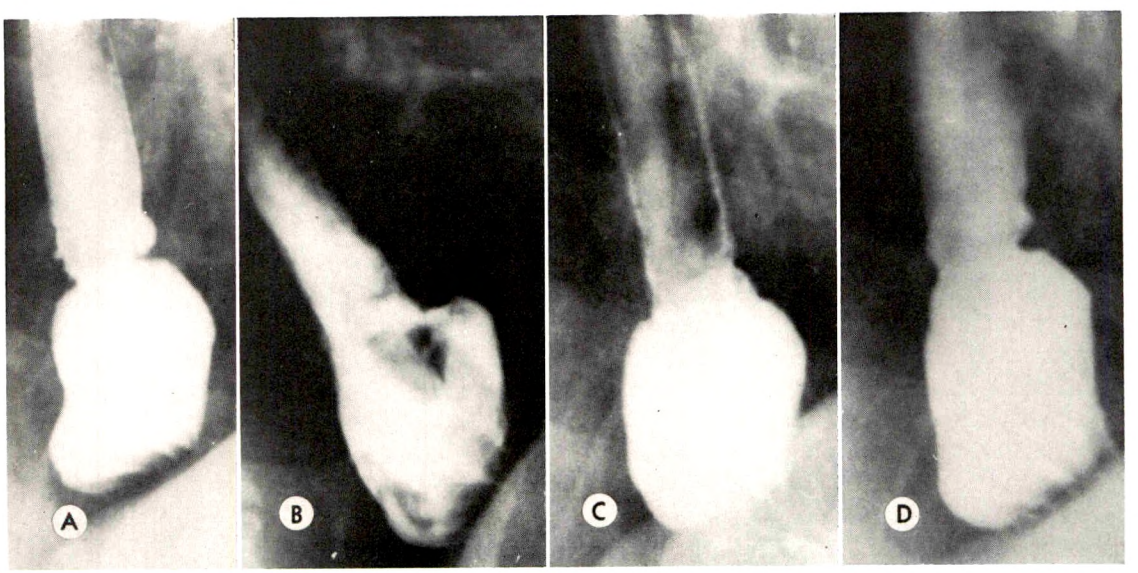


Fig. 3. Patient 6. (*A*) Predilatation: 13 mm. Moderate-sized hernia. (*B*) Immediately after dilatation: 24 mm. Ring has disappeared. Filling defects in distal esophagus and herniated stomach consistent with blood and mucus. (*C*) 24 hr. postdilatation: 20 mm. (*D*) 7 mo. postdilatation: 19 mm. Ring again present. No symptoms.

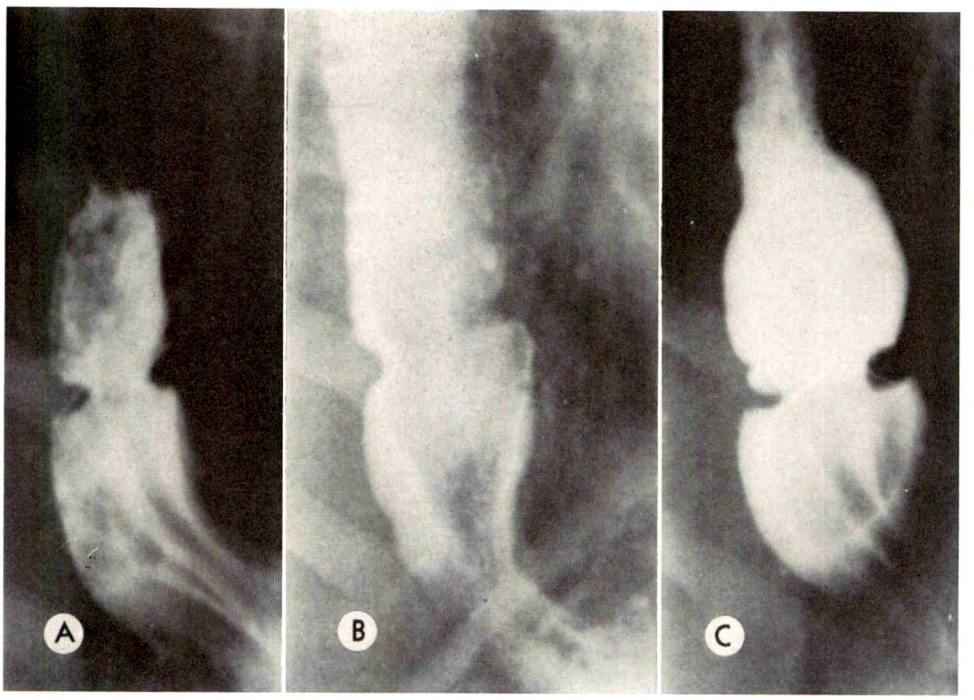


Fig. 4. Patient 7. (*A*) Predilatation: 11 mm. (*B*) Immediately after dilatation: 21 mm. Ring did not disappear completely as in Figure 3. (*C*) 6 mo. postdilatation: 15 mm. Symptoms improved.

lowing pneumatic dilatation when the ring measured 16 mm. and 14 mm., respectively. At that time the patient was asymptomatic.

The second patient is a 28 year old white male (Case 2) with a lower esophageal ring measuring 10 mm. in diameter (Fig. 12). When initially seen, he had dysphagia, heartburn and roentgenographic evidence of gastroesophageal reflux. He was treated with a Mosher dilator on August 25, 1967 and the ring increased to 15 mm. in diameter (Fig. 13*A*). Over the next 15 months, however, it diminished to 10 mm. in diameter but did not assume its predilatation appearance. It then had features of esophageal stricture (Fig. 13, *B* and *C*). Dysphagia had recurred. Esophagoscopy 15 months following dilatation revealed definite localized inflammatory changes and narrowing at the esophagogastric junction. Since this is probably an esophageal stricture, further pneumatic dilatations are not planned in this patient and surgical correction will be undertaken.

All patients reported definite improvement in dysphagia after dilatation. They have all shown a reduction in the diameter of their lower esophageal rings when compared with the size immediately or 24 hours after dilatation but the reduction has not been sufficient to cause symptoms except in 2 patients. Case 2 has apparently developed an esophageal stricture. The ring of Case 1 has returned to its predilatation diameter 18 months following dilatation, although he claims that he is relatively asymptomatic. He has trained himself to eat more slowly.

The size of the hernia below each ring was unchanged by dilatation. Several of these 10 patients had curling of the lower esophagus. This was not changed by dilatation. Four patients had prominent cricopharyngeus muscles. None had evidence of a pulsion diverticulum or peptic ulcer disease.

DISCUSSION

Lower esophageal rings are quite common, being found in 8 to 10 per cent of patients having examinations of the upper

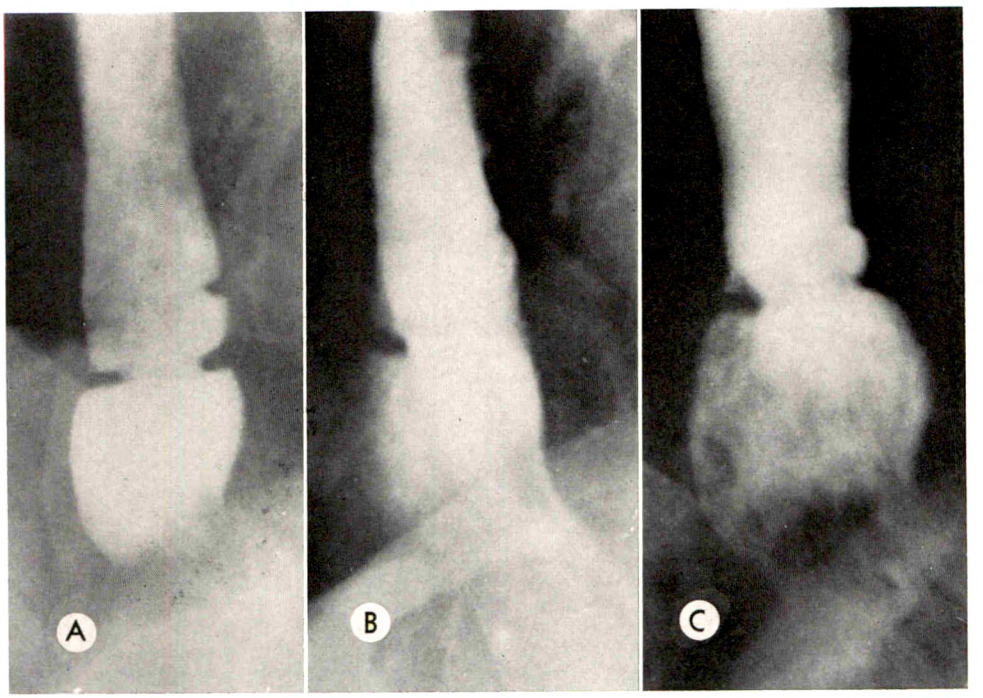


Fig. 5. Patient 4. (*A*) Predilatation: 15 mm. (*B*) 4 da. postdilatation: 22 mm. Ring is no longer concentric. (*C*) 8 mo. postdilatation: 18 mm. Ring now more symmetric. No further symptoms.

gastrointestinal tract. Occasionally, the symptom of dysphagia is not the patient's chief complaint. When a lower esophageal ring is identified and the diameter is ap-



Fig. 6. Patient 10. Ring 4 mm. in diameter and 6 mm. in thickness. Progressive dysphagia for 8 years.



Fig. 7. Patient 10. One month following mercury bougienage: 9 mm. Much less dysphagia.

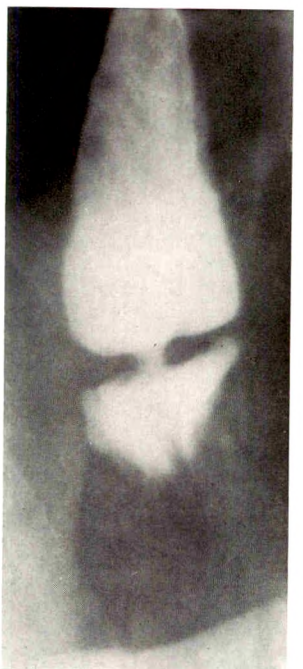


FIG. 8. Patient 10. Two years post-mercury bougienage: 5 mm. Recurrent symptoms.



FIG. 9. Patient 10. Retropharyngeal air 4 hours after mercury bougienage.

proximately 15 mm. or less, the patient will usually admit to dysphagia with bulky foods.

Although pneumatic dilatation invariably results in an initial increase in the diameter of the ring, the maximum diameter obtained does not persist. The rings of 2 of the 3 patients who have been followed the longest have returned to their predilatation size (Cases 1 and 2). Repeat dilatations will be done if symptoms recur and no contraindication exists. Eight of the 10 patients have remained asymptomatic for periods of 4 to 18 months.

The etiology of lower esophageal rings is unknown.[6,9] Surgically excised rings have shown a variable amount of chronic inflammation in the submucosal layer, with round cell infiltration and increased connective tissue. The mucosal junction between the esophagus and stomach is usually at the level of the ring,[5,10] although the ring sometimes is covered only with esophageal mucosa. In only 1 case was an abnormality of the muscular layer of the esophagus described.[2]

Wilkins and Bartlett[10] operated on 15 cases of lower esophageal ring. The hiatus hernia was repaired in all patients. The ring was digitally ruptured in 7, totally excised in 5 and partially excised in 3. Only 1 has developed recurrent dysphagia, a patient who had a gastrotomy with digital rupture of the ring.

Postlethwait and Sealy[4] have used several methods of treatment in 59 patients. They stress individualized therapy depending on whether obstructive symptoms due to the ring or symptoms of reflux esophagitis were present. In 15 patients having hiatus hernia repair and partial excision of the ring, 10 showed recurrence of the ring, 5 of them symptomatic. Hernia repair and digital rupture in 7 patients was followed by recurrence of the ring in 2. Neither had dysphagia, but both had symptoms of reflux. In the group having hernia repair alone, 11 patients had dysphagia. Symptoms recurred in 5 of them and subsequently improved with dilatation using a Hurst bougie. Good results were obtained in another group of 11 patients after dilata-



Fig. 10. Patient 10. Laceration of right posterolateral wall of upper esophagus demonstrated by retention of water soluble contrast material in adjacent soft tissues. A small amount of gastrografin can be seen in the hypopharynx and esophagus.

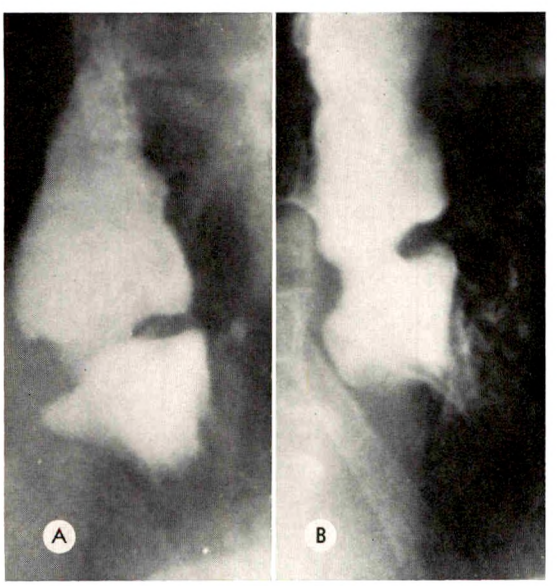


Fig. 11. Patient 10. (*A*) 24 hr. after pneumatic dilatation: 16 mm. (*B*) 2 mo. later: 14 mm. No symptoms.

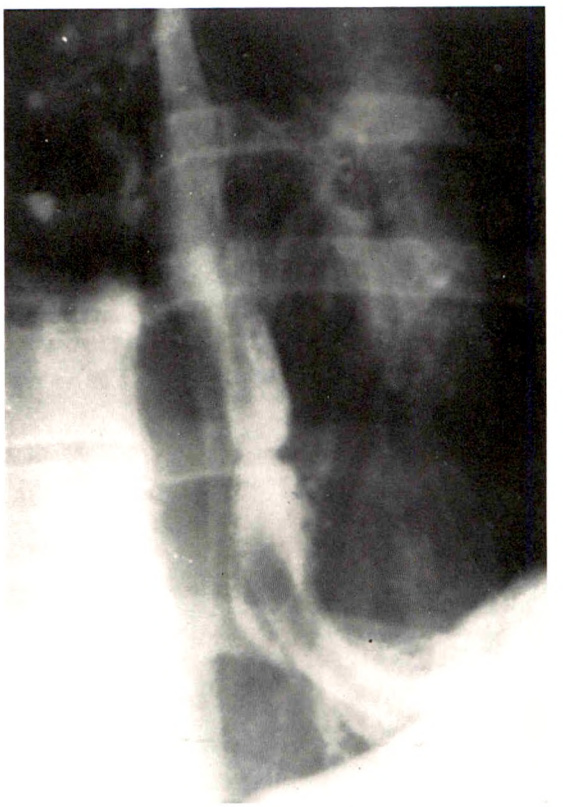


Fig. 12. Patient 2. Predilatation: 10 mm.

tion alone with a Hurst bougie. No patient required subsequent repair of the hernia. Twelve patients were managed merely with instructions to eat showly and chew food thoroughly. Eight of these 12 patients had rings measuring 14 mm. in diameter or less. Pneumatic dilatation gave favorable results in 2 previously reported patients, one with a 17 mm. ring[3] and the other with a 6–7 mm. ring.[1]

Dilatation would appear to accomplish the same end as digital rupture or partial excision of the ring and is a reasonable alternative to thoracotomy in the management of patients with lower esophageal rings causing dysphagia. Patient selection is of some importance as illustrated in Case 2 in our series. Since this patient had gastroesophageal reflux, surgical management might have prevented the development of what appears to be an esophageal stricture. Judging from published material, if surgery

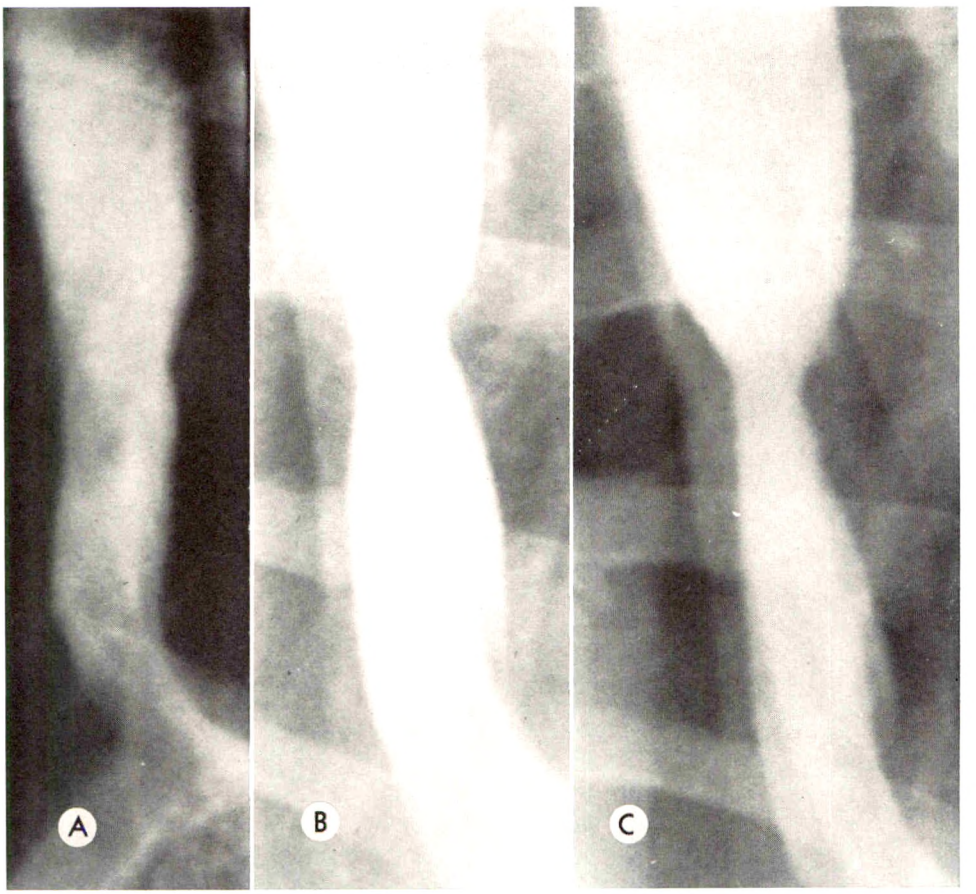


Fig. 13. Patient 2. (*A*) 24 hr. postdilatation: 15 mm. (*B*) 13 mo. postdilatation: 11 mm. Recurrent symptoms. Esophagogastric junction does not have same appearance as before dilatation. (*C*) 18 mo. postdilatation: 10 mm. Probable stricture. This patient had gastroesophageal reflux when first seen.

for a lower esophageal ring is done, total excision of the mucosa forming the ring at the esophagogastric junction and repair of the hernia is probably the method of choice.

SUMMARY

Lower esophageal rings can be treated satisfactorily by pneumatic dilatation.

Studies of the esophagogastric junction before, during, and at varying intervals after dilatation have been made in 10 patients.

Dilatation always increased the lumen of the ring and all patients were initially improved symptomatically. The rings did not maintain the size present immediately or shortly after dilatation but to a variable extent became more narrow with time. Two patients have demonstrated a return to a predilatation diameter. One of these had roentgenographic evidence of gastroesophageal reflux before dilatation and has apparently developed a localized stricture 1 year after dilatation.

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EXPERIMENTAL ESOPHAGITIS: ROENTGENOGRAPHIC FINDINGS AFTER INSUFFLATION OF TANTALUM POWDER*

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THE morphologic abnormalities of acute esophagitis are difficult to demonstrate roentgenographically. In roentgenograms of humans[6] and experimental animals,[4] luminal narrowing caused by esophagitis is the most commonly noted abnormality. Shallow ulcerations and thickened folds, when present, are seldom demonstrated on roentgenograms.

A technique has recently been described whereby esophagograms of excellent mucosal detail were obtained in cats, after the insufflation of powdered tantalum.[5] The present report evaluates tantalum esophagography as a method of demonstrating mucosal ulceration and thickened esophageal folds associated with acute esophagitis in the cat. The adherence of tantalum to areas of esophagitis was also studied.

MATERIAL AND METHOD

PRODUCTION OF ESOPHAGITIS

Esophagitis was produced in 15 adult mongrel cats, each weighing 4 to 6 kg. by a method described previously.[3] Unmedicated supine cats were restrained and their jaws immobilized by rubber bite-blocks. After the tip of a polyethylene tube was positioned in the mid-esophagus, an acid-pepsin solution (hydrochloric acid 25 mEq/L and porcine pepsin 25 μg./cc. HCl) was infused at a rate of 1 cc. per minute for 2 hours. The resultant esophagitis involved predominantly the middle one-third of the esophagus. This middle third was chosen because the pattern of the folds, consisting of 3 to 4 longitudinal folds, is similar to that of the human esophagus.[2] The distal one-third to one-half of the feline esophagus usually is characterized by prominent transverse folds, which are not present in the human. After infusion, the cats were permitted a normal diet.

TANTALUM ESOPHAGOGRAMS

Tantalum esophagograms were obtained on each animal 3 days after the production of esophagitis, since a previous study[4] showed that the inflammatory response from a 2 hour acid-pepsin infusion is maximal at 3 days.

The animals were sedated with phenobarbital (0.5 cc. intraperitoneally) and azepromazine (0.25 cc. intramuscularly). Atropine sulfate (0.2 mg./kg. intramuscularly) was given to diminish salivary secretions. Fifteen minutes after administration of atropine, powdered tantalum was insufflated into the esophagus under fluoroscopic control.[5] Roentgenograms of the esophagus were obtained in the frontal and lateral projections using cardboard cassettes, parspeed film, a focal-film distance of 90 cm., 1/60 sec. exposures, 300 ma., and 124 kv.

ADDITIONAL STUDIES

A lateral roentgenogram of the chest was obtained 2 hours after tantalum insufflation in 1 cat, 12 hours in 2 cats, 1 day in 4 cats,

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This material has been reviewed by the office of The Surgeon General, Department of the Army, and there is no objection to its presentation and/or publication. This review does not imply any indorsement of the opinions advanced or any recommendation of such products as may be named.
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2 days in 1 cat, 3 days in 3 cats, 4 days in 2 cats, 1 week in 1 cat, and 2 weeks in 1 cat. Thereupon each cat was killed by intracardiac injection of pentabarbital. To identify the presence of tantalum beyond the wall of the esophagus, the entire animal was roentgenographed. Because the severity of acute feline esophagitis 3 days after a 2 hour infusion of acid-pepsin solution of identical concentration as the one used in the present investigation had already been documented,[4] sacrifice of the animals immediately after tantalum insufflation was not considered necessary. At 3 days the esophagitis is characterized by mucosal ulceration and inflammatory infiltrate extending into the submucosa.

The esophagi were resected and examined for distribution of tantalum, and gross changes of esophagitis were scored from 1 to 3.[4] For further documentation, roentgenograms were obtained of each specimen on Kodak M film. After fixation for 24 hours

Table I

EXPERIMENTAL ESOPHAGITIS OF 3 DAYS' DURATION DEMONSTRATED ON TANTALUM ESOPHAGOGRAMS OF 15 CATS

Cat	Widened longitudinal folds	Irregularity of luminal contour	Ulcer	Narrowing of lumen
1	+	+	o	o
2	+	+	o	+
3	+	+	o	+
4	+	+	o	+
5	Not identified	o	o	o
6	+	+	+	+
7	+	+	o	+
8	o	o	o	o
9	+	+	+	+
10	+	+	o	o
11	Not identified	o	o	o
12	Not identified	+	+	+
13	+	o	o	o
14	+	+	+	+
15	o	o	o	o
Total abnormal findings	10	10	4	8

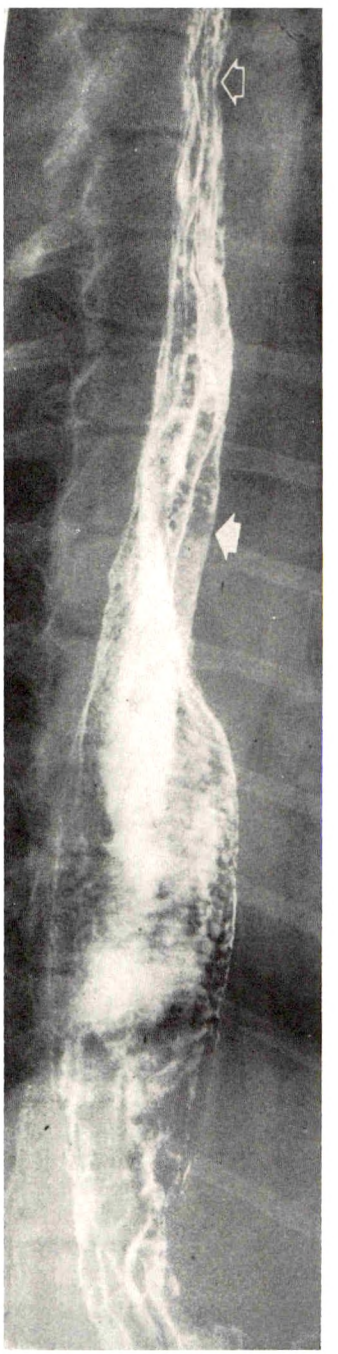


Fig. 1. Cat 9. Tantalum esophagogram. Esophagitis of the middle one-third of the esophagus is manifested by widened longitudinal folds, measuring 2.5 mm. in diameter (closed arrow). Irregularity of the luminal contour and segmental narrowing are also present. Normal longitudinal folds (open arrow) are seen in the upper esophagus, an area that was normal on histologic examination.

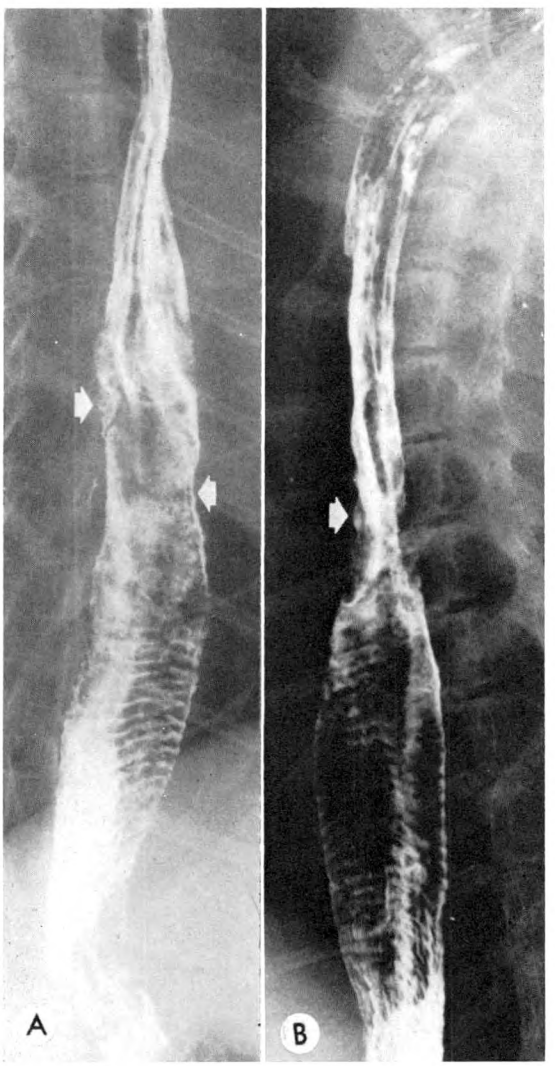


FIG. 2. Cat 7. Tantalum esophagograms. (*A*) Anteroposterior projection. The irregular luminal contour (arrows) is evident in the middle portion of the esophagus. (*B*) Lateral projection. A discrete superficial ulcer is situated on the anterior esophageal wall (arrow). Contour irregularity and luminal narrowing are also evident.

in 10 per cent formalin, the entire thoracic esophagus was sectioned longitudinally and areas of gross esophagitis transversely. The sections were stained with hematoxylin and eosin, phosphotungstic acid hematoxylin, and Wright's stain.

Tantalum esophagograms were also obtained on 15 live control animals, for comparison of luminal contour and fold characteristics. The same roentgenographic factors were used (100 cm. target-to-film distance).

EVALUATION OF ROENTGENOGRAPHIC AND HISTOLOGIC FINDINGS

The tantalum esophagograms were scored by two radiologists (HIG and WJD). Width of folds, luminal contour, and luminal width were evaluated. These esophagograms were compared with those obtained on the 15 control animals. The width of longitudinal folds was measured on roentgenograms of collapsed esophagi. The transverse folds and luminal diameter were measured on roentgenograms of esophagi distended with 30 to 50 cc. of air.

By means of a previously described system of scoring,[4] a pathologist (EHJ) scored the histologic sections from 0 to 6, A to F. This system of scoring esophagitis is based both on depth and on phase of injury. Scores of 1 to 6 indicated extension to the epithelium, lamina propria, muscularis mucosa, submucosa, and inner circular and outer longitudinal muscle layer of the muscularis externa, respectively. Progressive stages of esophagitis during the inflammatory phase were scored as A to C and stages during the reparative

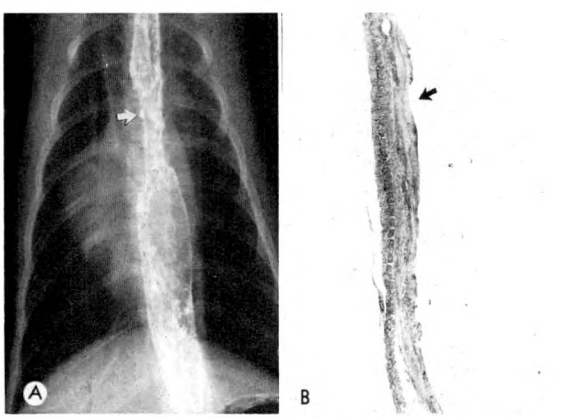


FIG. 3. Cat 3. (*A*) Tantalum esophagogram. The upper middle esophagus is segmentally narrowed. A shallow ulcer (arrow) and irregular contour of the lumen are also present. (*B*) Microscopic section (×5) demonstrating diffuse esophageal ulceration covered by inflammatory infiltrate. The depression (arrow) corresponds to the discrete ulcer shown in *A*.

phase D to F. Normal esophagi were scored ○.

RESULTS

ROENTGENOGRAPHIC FEATURES OF ACUTE ESOPHAGITIS

The roentgenographic features of tantalum esophagograms obtained on 15 animals with esophagitis of 3 days' duration are listed in Table 1. Abnormal findings consisted of widened longitudinal folds, irregular luminal contour, discrete superficial ulceration, and luminal narrowing. Except in 3 instances, the abnormalities were confined to the middle one-third of the esophagus.

Widened longitudinal folds (Fig. 1) were present in 10 animals. In 2 animals the longitudinal folds were normal and in 3

TABLE II

RESULTS OF ROENTGENOGRAPHIC AND HISTOLOGIC EXAMINATIONS
(2 Hours to 2 Weeks After Insufflation of Tantalum)

Cat	Time animal was killed after insufflation	Roentgenographic demonstration of retained tantalum		Pathologic examination	
				Gross	Histologic
		Chest	Specimen	Tantalum (esophagitis score)	Tantalum (esophagitis score)
1	2 hr.	+	+	+ (2)	+ (4C)
2	12 hr.	+	+	+ (1)	+ (3C)
3	12 hr.	+	+	+ (2)	+ (3C)
4	1 da.	+	+	+ (2)	+ (4C)
5	1 da.	○	+	+ (1)	+ (3C)
6	1 da.	+	+	+ (3)	+ (4C)
7	1 da.	+	+	+ (2)	+ (4C)
8	2 da.	○	○	○ (1)	○ (1A)
9	3 da.	+	+	+ (2)	+ (4C)
10	3 da.	○	○	○ (3)	○ (5C)
11	3 da.	○	○	○ (1)	○ (1A)
12	4 da.	+	+	+ (3)	+ (5C)
13	4 da.	○	○	○ (0)	○ (0)
14	1 wk.	○	+	+ (2)	+ (4C)
15	2 wk.	○	○	○ (0)	○ (0)
Total with retained tantalum		8	10	10	10

animals they were not identified. The diameter of the widened folds ranged from 1.5 to 2.5 mm. (normal 1.0 mm. or less). In only 3 animals with abnormal longitudinal folds were the transverse folds in the distal esophagus widened, 1.5 to 2.0 mm. (normal 1.0 mm. or less).

Segmental irregularity of the esophageal luminal contour was noted in 10 animals (Fig. 2*A*). The ragged contour lacked the sharp, smooth margin seen on esophagograms of the control animals. Discrete ulcers were detected in only 4 animals (Fig. 2*B*). In each instance, the ulcer was shallow, measuring less than 1 mm. in depth. Irregularity of luminal contour was associated with widened longitudinal folds in 9 instances. Discrete ulcers were always accompanied with irregular luminal contour and widened folds.

In 8 animals, segmental narrowing of the esophageal lumen resulted in a diameter of 0.5 to 1.8 cm. (normal 2.0 to 2.5 cm.) (Fig. 3, *A* and *B*). Invariably, luminal narrowing was associated with widened longitudinal folds and irregularity of luminal contour. The 4 identified superficial ulcers were in areas of luminal narrowing.

ROENTGENOGRAPHIC-PATHOLOGIC CHANGES AFTER TANTALUM INSUFFLATION

Roentgenographic changes. Roentgenograms of the lateral chest, obtained in from 2 hours to 2 weeks after insufflation of tantalum, showed tantalum labeling in the esophagus of 8 animals (Table II). Residual tantalum was identified as linear streaks of radiodensity in the middle and distal esophagus (Fig. 4*A*). The retained tantalum in 6 of 7 animals was seen on roentgenography performed 2 hours to 1 day after tantalum insufflation. Two to 4 days after insufflation, tantalum was evident

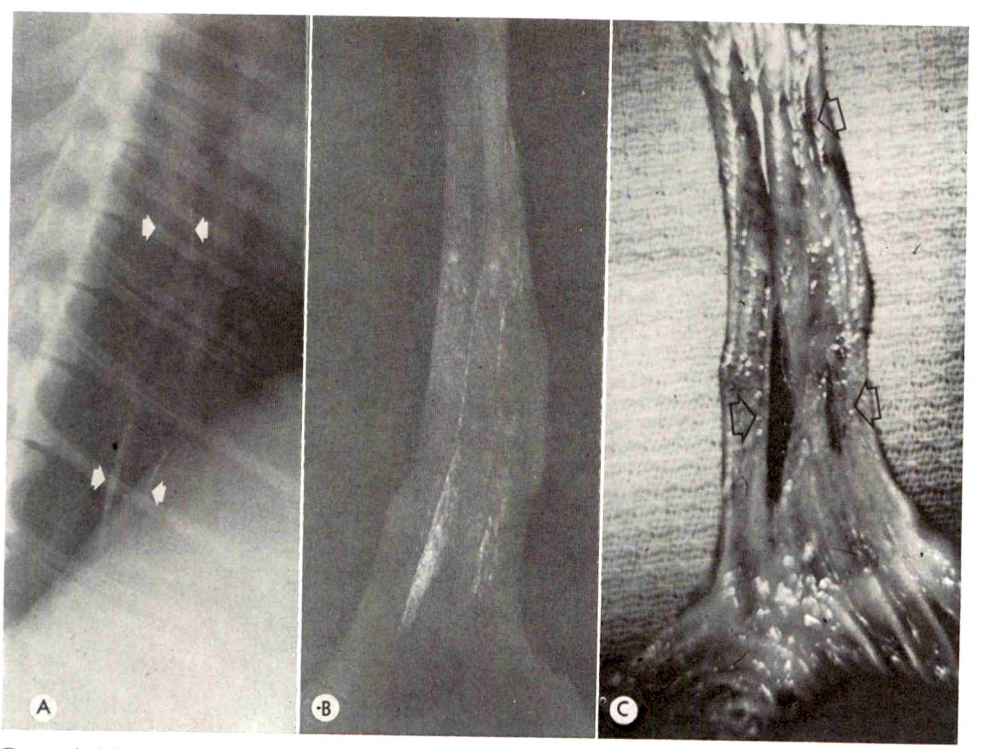


FIG. 4. Cat 4. (*A*) Lateral roentgenogram of the chest obtained 1 day after tantalum insufflation. Tantalum streaks (arrows) are seen in the middle and distal esophagus. (*B*) Roentgenogram of the resected specimen showing that distribution of tantalum corresponds to that seen on the gross specimen. (*C*) Resected esophagus. Black linear streaks represent retained tantalum enmeshed in inflammatory infiltrate (arrows) between thickened longitudinal folds.

on roentgenograms in 2 of 6 animals. No tantalum was seen in the 2 animals roentgenographed 1 and 2 weeks, respectively, after insufflation. Roentgenograms of the resected specimens confirmed that the tantalum identified on chest roentgenograms was within the esophagus (Fig. 4*B*). In only 2 specimens did the roentgenogram demonstrate tantalum, which was not noted on the chest roentgenograms.

Pathologic changes. Tantalum had usually accumulated in recesses between longitudinal folds in the esophageal specimens (Fig. 4*C*). In some specimens, small confluent patches of tantalum were seen on the tops of the longitudinal folds. The distribution of retained tantalum corresponded to that of thickened and discolored longitudinal folds, usually most pronounced in the middle third of the esophagus.

On examination of the histologic sections, clumps of tantalum particles, 10 to 100 microns in diameter were seen only in areas of esophagitis. The tantalum was located in the superficial layer of inflammatory infiltrate that covered areas of ulceration (Fig. 5). Phosphotungstic acid hematoxylin stains for fibrin showed the tantalum particles entrapped with the fibrin matrix of the inflammatory infiltrate. No phagocytosis of tantalum particles, or giant-cell reaction from foreign body was evident. Although edema and infiltrate of the esophageal wall were evident, tantalum was not seen beneath the superficial layer of inflammatory infiltrate. Nor were such particles seen in veins, arteries, or lymphatics. At no time, when tantalum was identified histologically, was it not seen on the gross specimen.

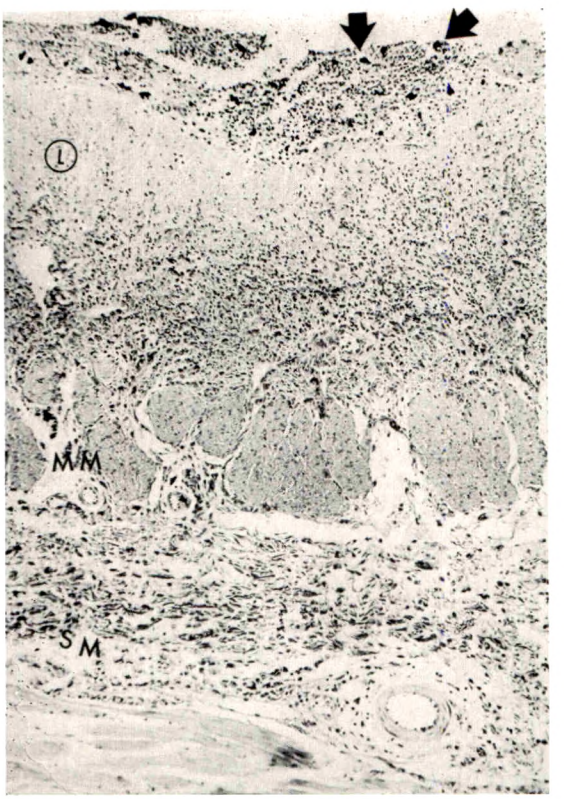


Fig. 5. Cat 7. Microscopic section (×80) of specimen of animal killed 1 day after tantalum insufflation. Esophagitis (score 4C) is evidenced by mucosal ulceration into the lamina propria (L), submucosal (SM) edema, and inflammatory infiltrate extending to the muscularis mucosae (MM). Clumped tantalum particles (arrows) are enmeshed in the superficial layer of inflammatory infiltrate covering areas of ulceration.

Correlation of Roentgenographic and Pathologic Findings

In the 10 animals with evidence of retained tantalum on gross and histologic examinations of esophageal specimens, lateral chest roentgenograms showed tantalum labeling in the esophagus in 8. Roentgenograms of the specimens showed retained tantalum in all 10 animals, distributed similarly as in the gross specimens (Fig. 4, *A–C*). Esophagitis accompanied with acute inflammatory infiltrate, present in these 10 specimens with retained tantalum, was scored 3 to 5 C (mucosal ulceration, edema, and inflammation to muscularis mucosae or externa). Histologically, the 5 specimens without roentgenographic evidence of retained tantalum were scored either as normal (except in Cat 11, scored 5C) or as demonstrating minimal changes (1A).

Discussion

This study indicated that tantalum

esophagography in the cat demonstrated subtle changes caused by acute esophagitis. In addition to luminal narrowing, frequent widened longitudinal folds and irregularity of luminal contour, and occasional superficial ulceration were evident. On barium esophagograms the only abnormal finding in cats with esophagitis of similar severity was segmental luminal narrowing. Widened folds, irregular luminal contour, or shallow ulcers were not demonstrated on the barium esophagograms.

Residual tantalum demonstrated on roentgenograms obtained 1 to 4 days after insufflation is of great interest. Tantalum was retained in the esophagus only when esophagitis was accompanied with acute inflammatory infiltrate. Tantalum particles were entrapped within the fibrin matrix. Apparently, the tantalum disappeared as the pseudomembrane of inflammatory infiltrate sloughed or resolved and healed.

Similarly, as in previous animal studies,[1,5] no toxic manifestations were observed after insufflation of tantalum into the upper gastrointestinal tract. Tantalum retained on areas of esophageal ulceration was not present beneath the superficial layers of acute infiltrate and was not seen in arteries, veins, or lymphatics. The inert tantalum did not cause phagocytosis or foreign-body reaction. Roentgenograms of the entire body did not demonstrate tantalum outside the gastrointestinal tract.

In humans with acute peptic esophagitis, roentgenographic abnormalities are seldom demonstrated by conventional examinations.[6] Tantalum esophagography is feasible in the human[5] and may show subtleties of acute esophagitis, as in experimental animals. The results of this study suggest that in humans, tantalum would be retained in inflammatory infiltrate over areas of esophageal ulceration. Such retained tantalum may permit not only the roentgenographic diagnosis of esophagitis, but may also serve as a means of observing sloughing or resolution of inflammatory infiltrate, and the course of healing.

SUMMARY

Tantalum esophagography was performed on 15 cats with peptic esophagitis of 3 days' duration. The esophagograms demonstrated segmental esophageal narrowing in 8 animals. In addition, wide longitudinal folds and irregularity of luminal contour were shown in 10 animals and discrete superficial ulceration in 4 animals. These latter findings are usually not present on esophagograms obtained with liquid barium. Lateral roentgenograms of the chest obtained 1 to 4 days after tantalum insufflation demonstrated retained tantalum, oriented in linear streaks. On histologic examination, tantalum was seen to be entrapped in inflammatory infiltrate covering areas of ulceration. Insufflated powdered tantalum not only facilitated the roentgenographic demonstration of subtle findings in esophagitis, but also labeled inflammatory infiltrate covering areas of ulceration.

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MIMICRY AND DECEPTION IN THE DIAGNOSIS OF TUMORS OF THE GASTRIC CARDIA*

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THE diagnosis of masses in the gastric cardia is difficult. Mimicry and deception are frequently encountered in this area.[14,22] Benign neoplasms may mimic malignant lesions, extragastric masses may simulate intrinsic gastric tumors, and various pseudotumors may occur. Kirklin stated the basic diagnostic problem: "In the differential diagnosis, the first consideration is to ascertain whether or not a shadow in the cardia represents a lesion." Difficulties in diagnosis are compounded by inherent problems in examining the area.

MATERIAL

When a number of cardia masses were not confirmed on repeat examination, and when several others proved to be non-neoplastic, this study was begun.

Forty cases recently encountered at The New York Hospital in which a mass in the gastric cardia had been diagnosed on the initial gastrointestinal series were reviewed. Masses which were felt to be due to an extrinsic compression were excluded. A variety of both tumors and non-neoplastic conditions was found, despite their striking roentgenographic similarity.

The various tumors and pseudotumors which initially presented as a "mass" in the cardia are listed in Table 1. The final diagnoses are based upon further roentgenographic studies, surgical exploration, or pathologic examination. One or more of these means provided a satisfactory explanation of the mass in the cardia in 38 of the 40 cases.

RESULTS AND DISCUSSION

The non-neoplastic conditions predominated. No true tumor was found in 71 per cent of these cases. When a true neoplasm was present, it was an intrinsic gastric tumor in 73 per cent of the cases. When the cardia mass was due to a true neoplasm, either intrinsic or extragastric, it represented a malignant tumor in 92 per cent of the cases.

Carcinoma is the usual and the most important neoplasm of the gastroesophageal junction.[23] The roentgenographic signs of a cardia carcinoma have been previously described.[1,5,11,18,19] About one-fifth (18 per cent) of the cardia masses in our series were gastric carcinomas, and about two-thirds (64 per cent) of the true tumors were primary gastric malignancies. The diagnosis of a fungating carcinoma in this region is not particularly difficult (Fig. 1). The real problems are the smaller carcinomas which may be mimicked by the other abnormalities described here (Fig. 2). These

TABLE I

FINAL DIAGNOSES IN 40 PATIENTS WITH CARDIA MASS DIAGNOSED ON INITIAL UPPER GASTROINTESTINAL SERIES

Neoplasm	
Carcinoma of stomach	7 cases
Carcinoma of pancreas	2 cases
Carcinoma of colon	1 case
Leiomyoma of stomach	1 case
No Neoplasm	
Hiatus hernia, no neoplasm	10 cases
No hiatus hernia, no neoplasm	8 cases
Gastric varices	3 cases
Extrinsic defect	3 cases
Past hiatus hernia repair, no neoplasm	2 cases
No hiatus hernia, reflux, no neoplasm	1 case
Etiology Undetermined	2 cases

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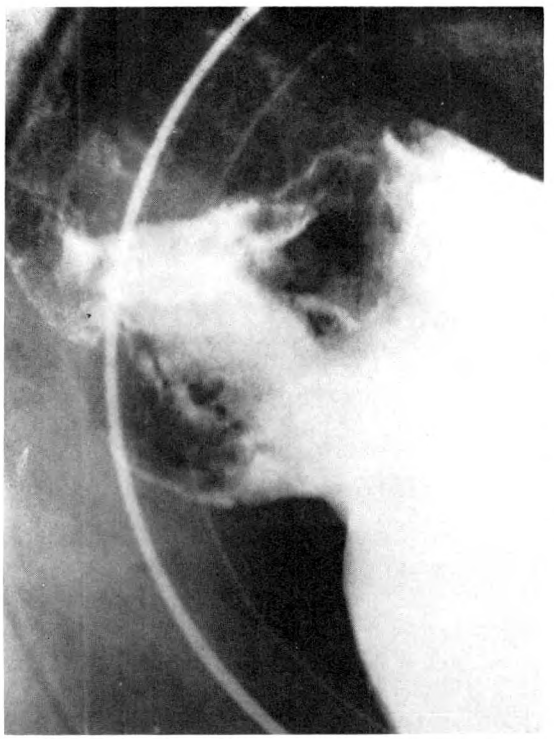


FIG. 1. A large fungating adenocarcinoma of the gastric cardia with extragastric metastases.

smaller tumors often cause little symptomatology and few signs.

Benign tumors of the cardia may present as masses roentgenographically indistinguishable from a small malignant tumor. One such case is illustrated in Figure 3.

Owing to their proximity, tumors and other abnormalities of adjacent organs and structures including the pancreas, liver, spleen, lymph nodes, aorta, colon, kidney, and adrenal, may present as cardia masses. A carcinoma of the pancreas near the gastroesophageal junction is shown in Figure 4. Another configuration indistinguishable from this case and from a primary gastric tumor was produced by a carcinoma of the splenic flexure (Fig. 5, *A* and *B*).

Pseudotumors are also possible causes of cardia masses. We have used this term to indicate those "masses" which were not seen on examinations done for confirmation. These represented 21 per cent of the 38 cases. Examples are shown in Figures 6 and 7. We agree with others that a repeat examination is most important to confirm the presence of a mass in the cardia.[5,18]

A hiatus hernia can produce additional difficulties. It has been shown that its presence substantially increases the incidence of incorrect diagnoses in cases of carcinoma of the cardia.[5] In this series, the presence of a hiatus hernia frequently led to the erroneous diagnosis of a tumor when none was present. Nine "tumor" patients were found to have nothing more than a hiatus hernia on repeat examination. A repeat examination again revealed a cardia mass in 1 case, which at laparotomy was proven to be due to a hiatus hernia. Figures 8, *A* and *B*, and 9 illustrate some cardia masses due to hiatus hernia. The pseudotumors of the gastric cardia found in asso-



FIG. 2. A smaller cardia mass due to an infiltrating carcinoma which produced an achalasia-like syndrome. This case has been reported in detail elsewhere.[23]

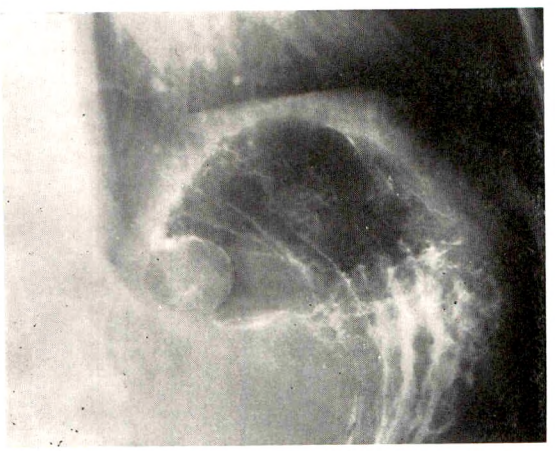


Fig. 3. A leiomyoma of the cardia producing a mass mimicking a smaller carcinoma. The smooth mucosal surface does not exclude an intrinsic malignant lesion.



Fig. 4. A cardia mass produced by an adenocarcinoma of the pancreas, mimicking an intrinsic gastric lesion. The preoperative diagnosis was carcinoma of the cardia.

ciation with hiatus hernias may be due to an invagination or a prolapse of the esophageal mucosa at the cardia.[3,12,15,16,21] Recently, a "notch sign" has been reported as an infradiaphragmatic manifestation of a "reduced" hiatus hernia.[8]

Because of a postoperative distortion of normal anatomic relationships and because of difficulties in obtaining the optimal views of the area, the region of the cardia may be particularly difficult to examine after hiatus hernia repair, subtotal gastrectomy or gastroenterostomy. A "mass" of the cardia seen following such surgery which ultimately required laparotomy for diagnosis is illustrated in Figure 10, *A* and *B*.

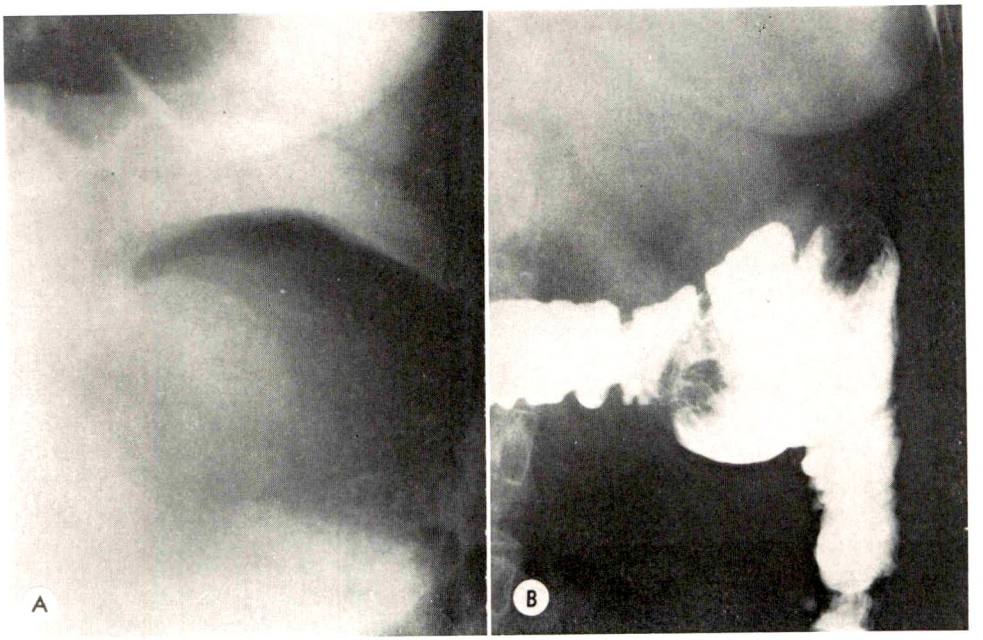


Fig. 5. (*A*) A cardia mass due to a perforating carcinoma of the splenic flexure. Splitting of the barium stream was noted on fluoroscopy. (*B*) A subsequent barium enema examination roentgenogram illustrates both the intraluminal and the extracolonic mass at the splenic flexure.



FIG. 6. A pseudotumor of the gastric cardia. No abnormality was shown on subsequent gastrointestinal series.

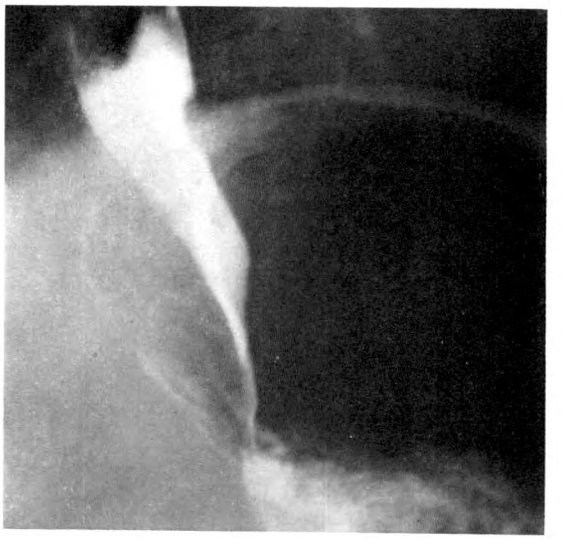


FIG. 7. Another cardia pseudotumor found on initial gastrointestinal series, not confirmed on subsequent studies.

Although the repeat examination may confirm the presence of a mass, it may not reveal its etiology. For example, gastric varices can mimic a neoplasm of the cardia (Fig. 11, *A* and *B*; and 12, *A* and *B*).

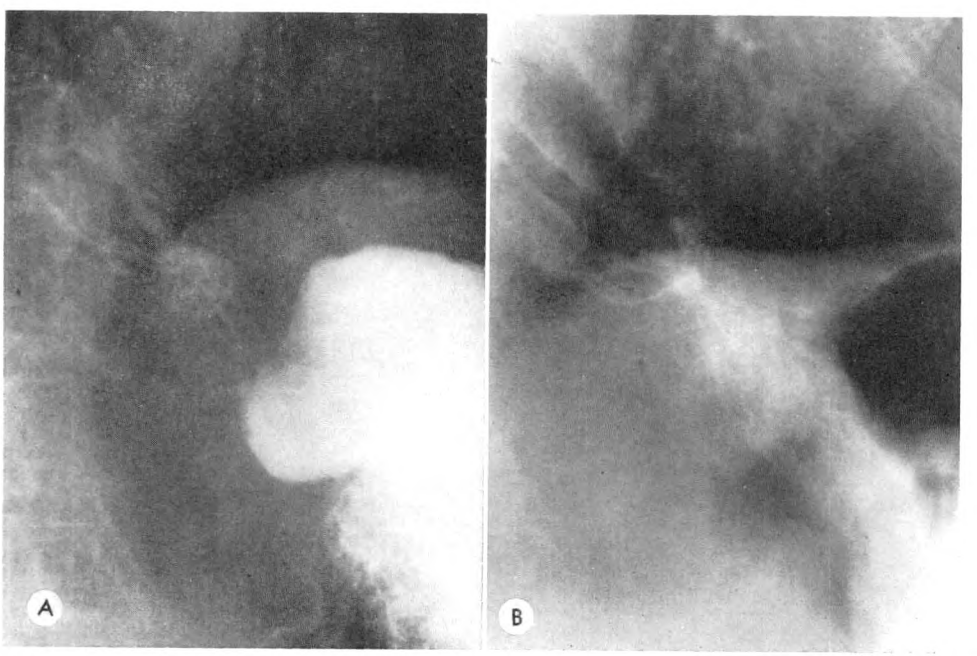


FIG. 8. (*A*) Supine and (*B*) air-contrast studies of a pseudotumor of the cardia related to a hiatus hernia. The preoperative diagnosis was carcinoma of the cardia with a small hiatus hernia. At operation, only a hiatus hernia was found.



Fig. 9. Another cardia pseudotumor. Subsequent studies showed only a hiatus hernia but no mass.

Masses due to gastric varices are particularly difficult to diagnose in the absence of esophageal varices, splenomegaly, or a history of cirrhosis.[2,4,6,10,20] Moreover, both splenomegaly and esophageal varices may be seen with other tumors owing to invasion and obstruction of the splenic or portal vein.

Table 11 briefly lists the entities which should be considered when a mass in the gastric cardia is encountered on the initial upper gastrointestinal series.

The technique for studying the cardia should be adapted to the problem at hand. We recommend several views of the cardia and fundus in the Trendelenburg left posterior oblique position as a bolus of barium enters the gastroesophageal junction. Multiple views of the gas-distended fundus should be obtained with the patient erect as barium enters the region of the cardia and in positions "around-the-clock." The use of carbonated beverages should be avoided if possible, as they tend to dilute and flocculate the barium suspensions. Additional studies made in the anteroposterior projection when the fundus and cardia are dependent, as well as a left lateral view may be helpful. Occasionally a supine translateral view may be useful. The quality of the examination is primarily a product of the ingenuity and the

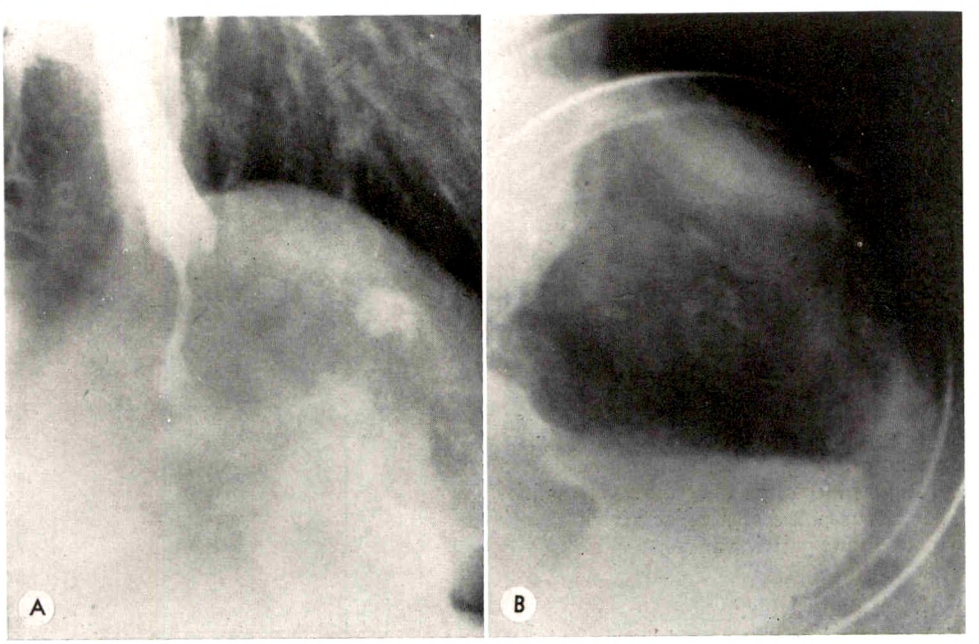


Fig. 10. (*A*) Deformity at the gastroesophageal junction and (*B*) a cardia mass in a patient with a prior hiatus hernia repair. Roentgenographic and endoscopic findings suggested a cardia carcinoma. At laparotomy, evidence of the previous hiatus hernia repair was found, with mild peptic esophagitis, but no tumor was seen or found on histologic examination.

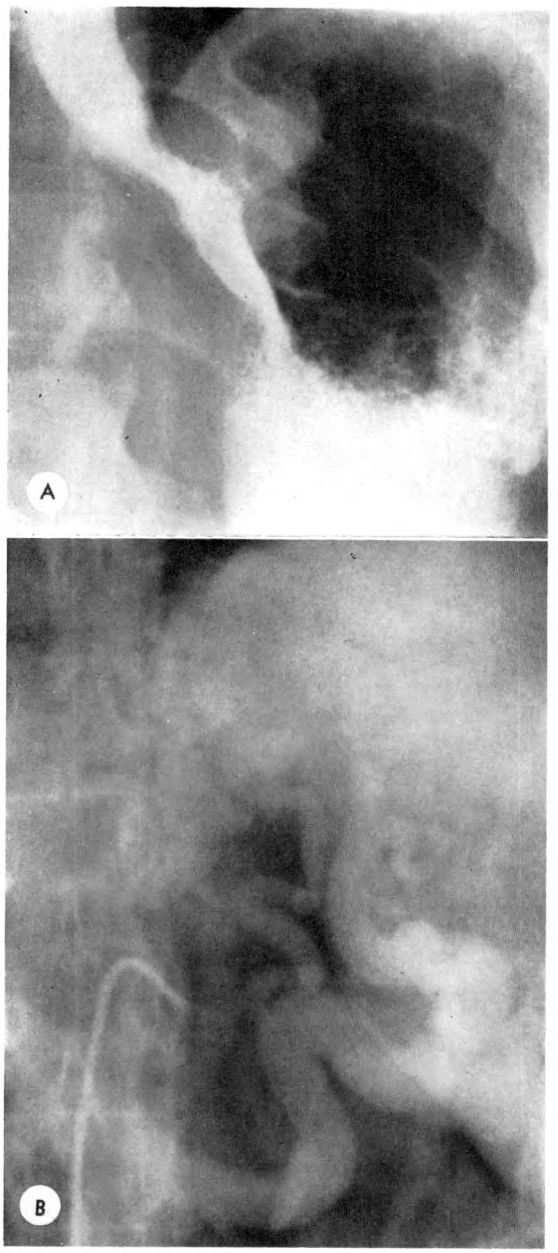


FIG. 11. (*A*) A cardia mass due to gastric varices in a patient in whom there was no splenomegaly and in whom no esophageal varices could be demonstrated. Rigid endoscopy showed no abnormalities. (*B*) The venous phase of a celiac arteriogram in this patient shows gastric varices.

efforts of the examiner.[18]

Additional roentgenographic techniques may be useful in evaluating cardia masses. A barium enema examination may provide the diagnosis, as illustrated in Figure 5, *A* and *B*. Occasionally, tomography of the gastroesophageal junction may be of value,[7] as shown in Figure 13, *A-D*. Arteriography will help to diagnose gastric varices (Fig. 11, *A* and *B*), as well as the pseudotumor due to tortuous aorta, carcinoma of the pancreas, or masses arising in the left lobe of the liver. Splenoportography may be of value with gastric varices (Fig. 12, *A* and *B*) or carcinoma of the pancreas invading and obstructing the portal or the splenic vein.

Radioisotope scanning may help distinguish intrinsic lesions from extrinsic defects caused by the lobe of the liver and

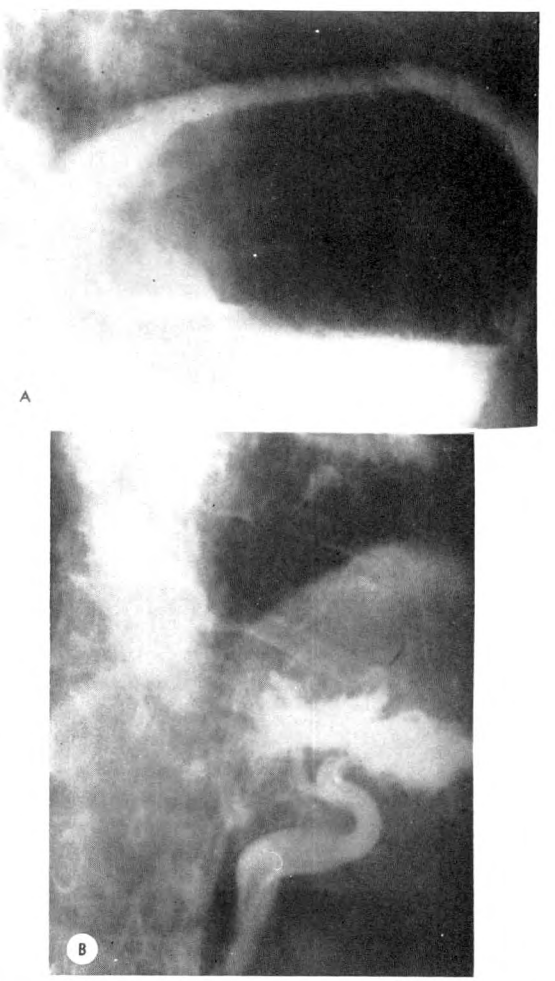


FIG. 12. (*A*) Another cardia mass due to gastric varices. Small esophageal varices were seen on endoscopy. (*B*) A splenoportogram shows both gastric and esophageal varices.

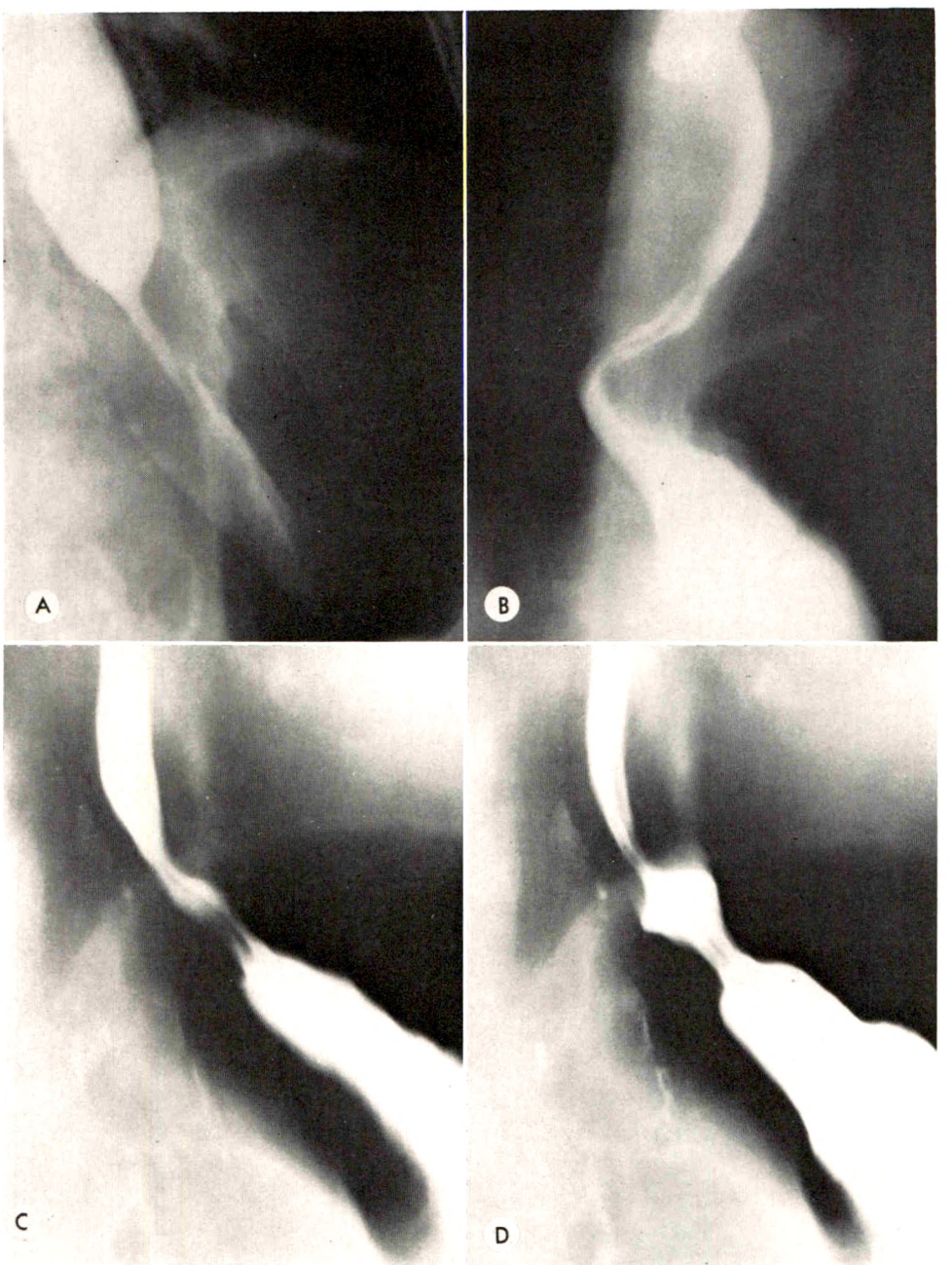


Fig. 13. (*A*) Another cardia mass due to the imprint of a tortuous aorta. (*B*) Anteroposterior and (*C* and *D*) oblique laminagrams show the aortic shadow imprinting on the cardia and no mucosal lesion.

neoplasms of the pancreas, spleen, or kidney.

Endoscopy is a valuable adjunct in evaluating these lesions and the area of the cardia. In particular, the gastric camera can provide a clear view of the cardia and fundus.[9,17]

SUMMARY

Although carcinoma of the cardia of the stomach is clearly the most important neoplasm of the area from the standpoint of diagnosis, the various other tumors and non-neoplastic conditions which may appear as masses in the cardia and mimic a

TABLE II

CONDITIONS PRESENTING AS CARDIA MASSES

I. *Normal Variants* causing apparent cardia masses

II. *Endogastric Causes* of cardia masses
- A. True Neoplasm
  - 1. Carcinoma of stomach
  - 2. Lymphoma of stomach
  - 3. Leiomyoma/leiomyosarcoma of stomach
  - 4. Other benign neoplasms (*e.g.*, adenoma, lipoma, etc.)
- B. No True Neoplasm
  - 1. Pseudotumor associated with hiatus hernia
  - 2. Gastric varices
  - 3. Masses seen in postoperative stomach
  - 4. Giant rugae in the cardia

III. *Exogastric Causes* of cardia masses
- A. True Neoplasm
  - 1. Carcinoma of body or tail of pancreas
  - 2. Carcinoma of splenic flexure of colon
  - 3. Lymphoma or metastatic carcinoma in adjacent lymph nodes
  - 4. Tumors of other adjacent organs (*e.g.*, liver, adrenal, kidney)
- B. No True Neoplasm
  Imprint of adjacent organs or structures (*e.g.*, liver, tortuous aorta, spleen)

carcinoma present difficult diagnostic problems.

Confirmation of the presence of a true mass lesion is the single most important additional diagnostic study. Repeat upper gastrointestinal series with attention to the area is the simplest means to do this. Some technical modifications for the study of this area are suggested. By this means most pseudotumors and non-neoplastic conditions mimicking a cardia carcinoma can be eliminated. Particular attention should be given to the possibility of a hiatus hernia, for pseudotumors are often associated with this abnormality.

Additional roentgenographic techniques such as barium enema examination, tomography, arteriography, and radioisotope scanning may be helpful in establishing the correct preoperative diagnosis. The gastric camera is a potentially valuable aid in evaluating this area. It can confirm the presence of a mass, and may reveal its etiology.

Thus, although many pseudotumors and tumors or other abnormalities of the cardia or adjacent organs or structures may mimic a carcinoma of the cardia in the initial upper gastrointestinal series, the use of additional roentgenographic techniques and the gastric camera should make it possible to eliminate many or most of these.

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PROLAPSED GASTRIC MUCOSA THROUGH A GASTROJEJUNOSTOMY*

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HERNIATION of the gastric mucosa through a gastrojejunostomy is a condition which is not very well known. In 1935, Kirklin,[8] discussing the different causes of vomiting after a gastroenterostomy, mentioned prolapse of the gastric mucosa through the stoma but cited no cases or references. Similar allusions without documentation were made by Bockus[5] and others. It was Gutmann, however, who first reported a case in 1937[6] and in 1947 published the first roentgenograms in his book.[7]

There have been several reports in the European literature[3–5] since that time but the first published roentgenogram in the American literature was the case report of Maisel and Weintraub[10] in 1951. This subject did not appear again in the American literature until 1963 when Levine *et al.*[9] presented 14 cases of what they felt were examples of gastrojejunal mucosal prolapse, although only 1 case was surgically confirmed. They were able to reproduce the roentgenologic appearance in a dog by doing a partial gastrectomy and leaving redundant mucosa which then prolapsed through the gastrojejunostomy. Two additional cases were reported by Shane, Szemes and Amberg[14] at the December meeting of the Radiological Society of North America in 1968. Because of the paucity of reports and the importance of correctly identifying this condition, our experience with 24 cases will be presented and the roentgenologic and clinical aspects discussed.

MATERIAL

Twenty-four patients with roentgenologic evidence of prolapsed gastric mucosa through a gastrojejunostomy have been observed at the Columbia-Presbyterian Medical Center during the past 5 years. Although only 4 of these patients have been re-operated upon, the roentgenographic appearance is quite characteristic so that an accurate, reliable diagnosis can be made by this method.

The age and sex distribution were not remarkable. Information as to the precise nature of the original surgery was not always available but in most instances, it was the Hofmeister modification of the Billroth II partial gastrectomy. The interval between the original surgery and the development or recognition of the gastric mucosal prolapse varied widely, ranging from 5 months to 32 years. In most instances, when there was a long time interval between the original operation and the detection of prolapsed mucosa, there were no intervening roentgen examinations so that the precise time of onset could not be determined. The median time between the initial surgery and detection of the prolapse was 6 years. The reason for the original operation was duodenal ulcer in 17 patients, gastric ulcer in 3, gastric carcinoma in 1 and indeterminate in 3. This seems consistent with the usual incidences of duodenal and gastric ulcer.

Prolapsed gastric mucosa into the anastomotic jejunal loop may be unaccompanied by symptoms. If large enough, partial obstruction may result and this occurred in 6 patients, was mild in 3 instances, was severe enough to recommend surgery in 3 instances but was only carried out twice.

Pain was a prominent symptom in 6 patients. This was attributed to a marginal ulcer which was confirmed roentgenologi-

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cally in 3 cases. These cases were treated by vagectomy (Fig. 6).

Bleeding was the most common clinical manifestation and occurred in 9 patients. In 7, the bleeding was characterized clinically by positive guaiac stools without melena or severe anemia. The hemoglobin and hematocrit levels were normal or near normal in all of these patients except 1 who had a hemoglobin of 5 gm. per cent and a hematocrit of 21 per cent and another patient with a hemoglobin of 9 gm. per cent. Despite repeated roentgenographic examinations, no other explanation for the blood loss was found. Three of these patients were also gastroscoped. Except for blood replacement in the severely anemic patients, no specific treatment was given and the bleeding stopped spontaneously.

In 2 other bleeding patients, an additional potential source of hemorrhage was found. In 1, a bleeding point in the efferent loop was found at surgery although no definite pathologic lesion was described. In a second patient, the possibility of an ulcer of the duodenal stump was raised following a roentgenographic examination.

GASTROSCOPIC FINDINGS

Seven patients were examined gastroscopically because of persistent epigastric pain or unexplained gastrointestinal bleeding. Large erythematous polypoid rugae protruding into the stoma were noted in 2 patients (Fig. 1; and 2, *A–C*) with symptoms of partial obstruction of the stoma.

In another patient, large, hyperemic edematous folds were seen in the region of the stoma. The stoma was not well visualized and no mention was made as to whether the folds actually protruded into the jejunum. Grimoud *et al.*[5] mention that gastric folds extending into the stoma may be seen gastroscopically and state that Moutier had observed this phenomenon in 3 patients of a series of 338 gastroscopies performed after partial gastrectomy. In a fourth patient, an 8×8 mm. ulcer was seen on the gastric side of the stoma and the jejunum was deemed unremarkable.

In the remaining 3 patients, no abnormalities of the gastric remnant or anastomotic jejunal loop were recorded. This is difficult to explain in view of the obvious roentgenographic abnormality (Fig. 3; and 4, *A* and *B*). This might be explained on the basis of unfamiliarity with the entity. Shane *et al.*[14] and Grimoud *et al.*[5] mention that the prolapsed mucosa may retract into the stomach when the patient is horizontal.

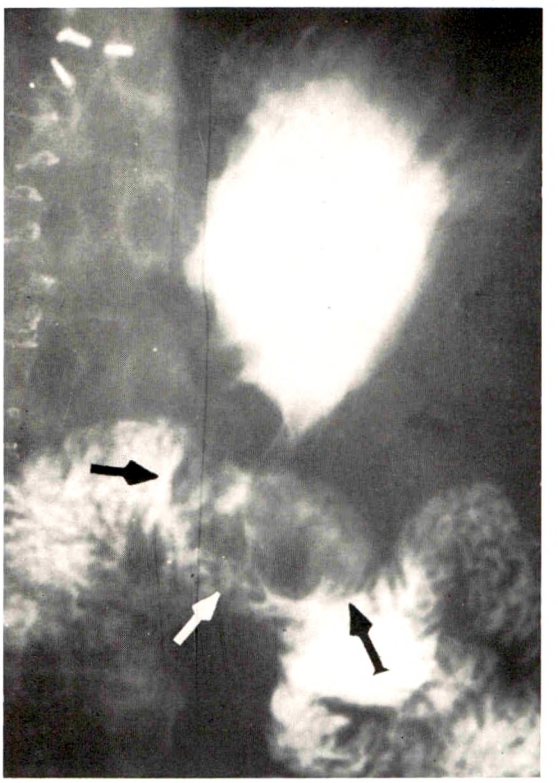


Fig. 1. Case 11. A 55 year old woman with persistent epigastric pain 6 months after a Hofmeister partial gastrectomy for gastric ulcer. Large erythematous folds projecting into a narrowed stoma were observed gastroscopically and roentgenographically. The prolapsed gastric mucosa was confirmed at the time of surgery. Note typical Hofmeister defect along the medial aspect of the gastric remnant.

ROENTGENOLOGIC ASPECTS

The roentgenographic appearance of prolapsed gastric mucosa is quite characteristic and easily recognized. The herniation produces a sharply marginated, smooth, occasionally scalloped, intraluminal mass in the adjacent jejunal loop (Fig. 3; 4, *A* and

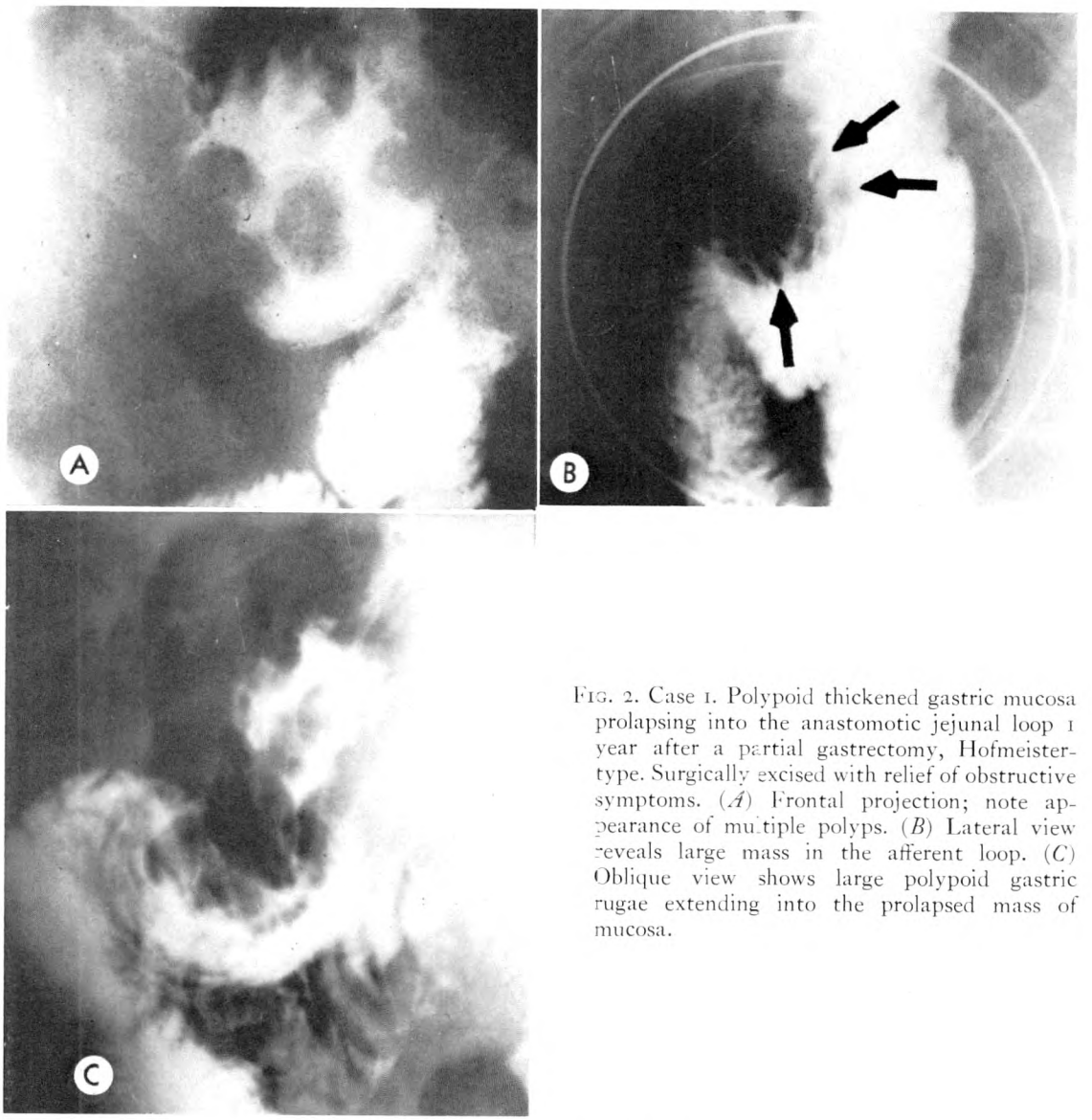


Fig. 2. Case 1. Polypoid thickened gastric mucosa prolapsing into the anastomotic jejunal loop 1 year after a partial gastrectomy, Hofmeister-type. Surgically excised with relief of obstructive symptoms. (*A*) Frontal projection; note appearance of multiple polyps. (*B*) Lateral view reveals large mass in the afferent loop. (*C*) Oblique view shows large polypoid gastric rugae extending into the prolapsed mass of mucosa.

B; 5, *A* and *B*; and 10). The prolapse may originate symmetrically from anterior and posterior margins of the stoma and produce the appearance of 2 separate masses (Fig. 9, *A* and *B*). The superimposition of the 2 masses may be responsible for the bilobed appearance occasionally seen (Fig. 3; and 5, *A* and *B*). In other instances, the prolapse is asymmetric resulting in 2 masses of disparate size, the larger usually located in the efferent loop probably because of the direction of the peristaltic gradient (Fig. 3; and 7). In some instances only one mass can be demonstrated (Fig. 4, *A* and *B*), although this single appearance can be due to superimposition of 2 masses (Fig. 8, *B* and *C*). An appearance of multiple polyps may also occur (Fig. 2*A*).

Since the mass is soft and flexible, it may be easily influenced by external compression or jejunal peristalsis and change in size and shape under the influence of these 2 factors (Fig. 4, *A* and *B*; and 9, *A* and *B*). The size varies greatly (Fig. 5, *A* and *B*; 6; 8, *A–E*; and 9, *A* and *B*) and when large, may be responsible for partial obstruction, particularly when associated with a small stoma (Fig. 1; 2, *A–C*; 7; and 8, *A–E*). This

may be associated with dilatation, delayed emptying of the gastric remnant (Fig. 7; and 8*E*) and retention of ingested food.

The margins are usually smooth but may appear serrated due to overlying jejunal mucosa (Fig. 4, *A* and *B*). Barium caught in folds of the prolapsed mucosa may simulate ulcer craters (Fig. 1; 5*B*; and 8*B*).

Progression of the lesion occurs very slowly. Multiple observations were available in 10 patients covering time intervals varying from 4 months to 6 years. Increase in size was found in only 4. In 2 patients, observed for 4 and 6 years, respectively, the size of the prolapsed mucosa more than doubled (Fig. 5, *A* and *B*), while in 2 others observed for 1 and 5 years, the increase was minimal (Fig. 8, *A–E*). Several cases are of unusual interest and are reported in more detail.

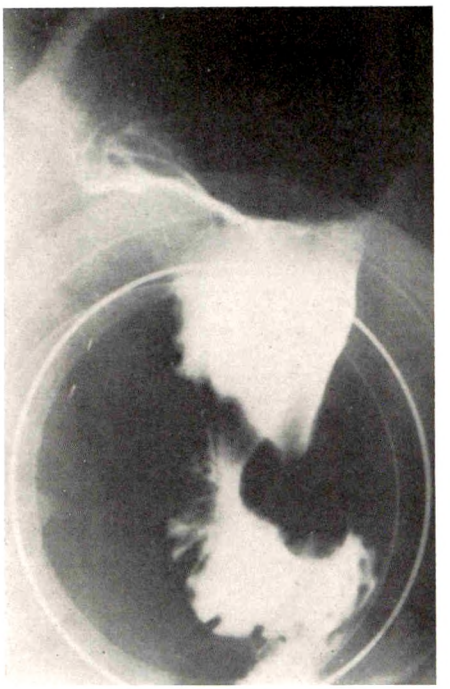


Fig. 3. Typical roentgen appearance of prolapsed gastric mucosa. Note sharp, lobulated contour and asymmetry of the prolapse with the larger component located in the efferent loop.

REPORT OF CASES

Case 1. P.V. 179-65-88. One year following a vagectomy and Hofmeister-type partial gastrectomy, this 51 year old male was readmitted to the hospital because of abdominal pains, weight loss, diarrhea and positive guaiac stools. A mild anemia (Hb. 13 gm. per cent) was demonstrated.

Roentgenographic examination revealed enlarged polypoid rugae in the gastric remnant which had prolapsed through the stoma into the

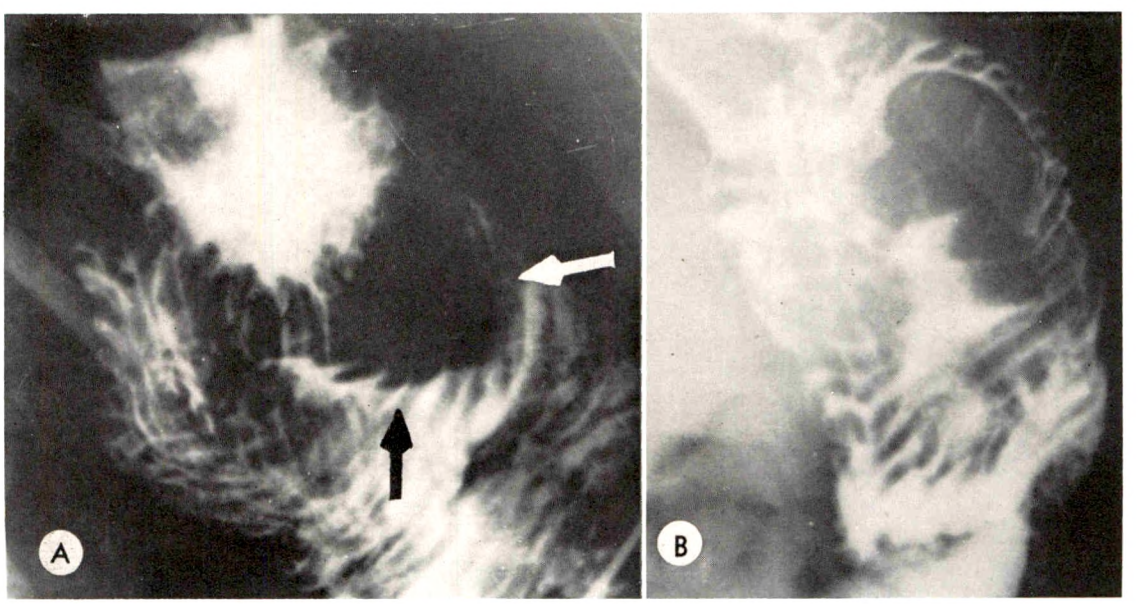


Fig. 4. (*A* and *B*) Asymmetric prolapse of gastric mucosa presenting primarily in the efferent loop. The irregular contour of the inferior aspect is due to superimposed jejunal pattern as seen in *B*.

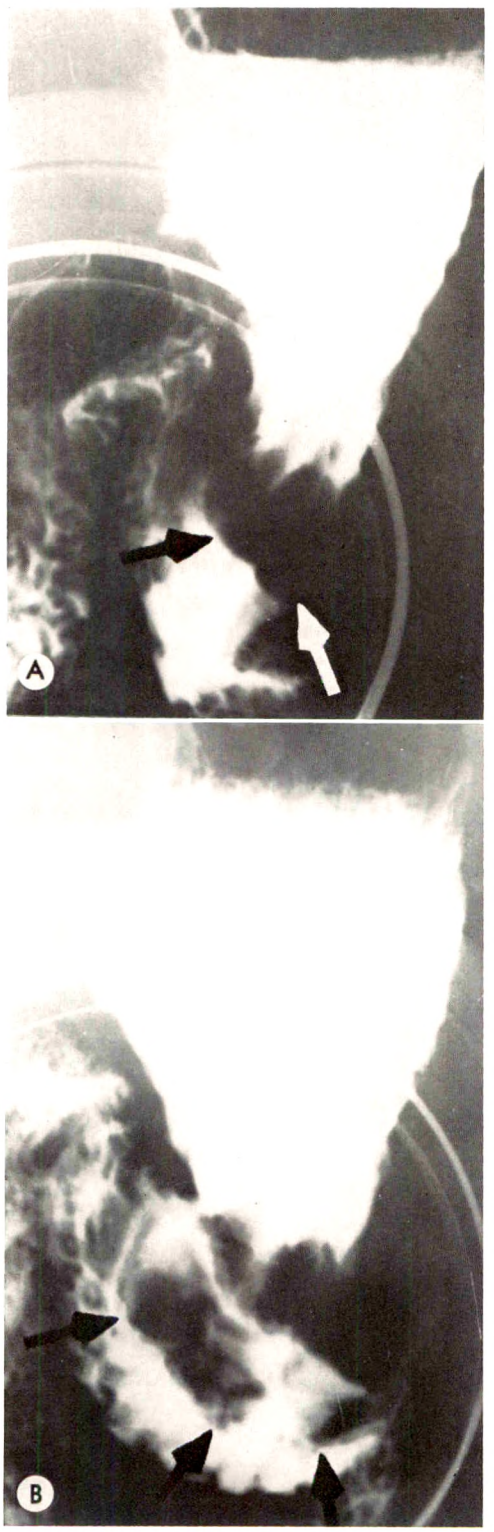


Fig. 5. (*A*) Example of minimal prolapsed mucosa detected in a 63 year old male with 4 + guaiac stools, 2 years after a partial gastrectomy for duodenal ulcer. (*B*) Four years later, the size of the prolapsed mucosa had more than doubled. Superimposed barium probably lies within the fold of the mucosa. There was no evidence of an ulcer, either roentgenologically or clinically.

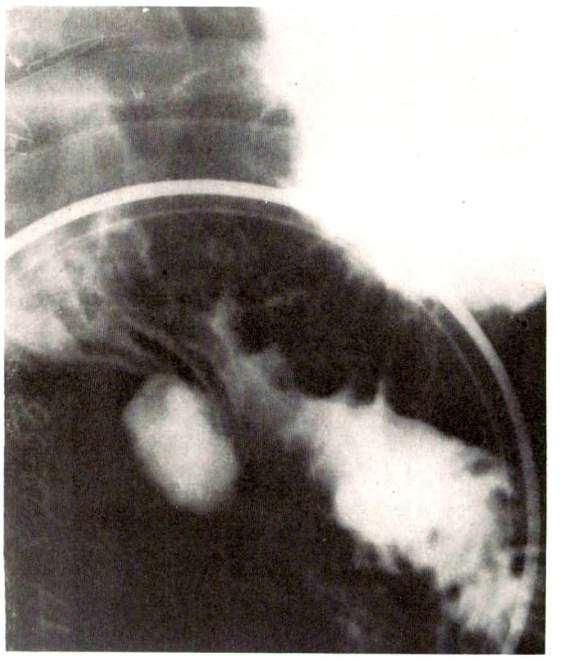


Fig. 6. Minimal prolapse of gastric mucosa in a patient with a jejunal ulcer.

jejunum (Fig. 2, *A–C*). At times, it had the appearance of a single, smooth mass arising from the anterior wall of the stoma and other times, depending upon the projection, seemed multiple.

On gastroscopy, it was noted that the mucosa around the stoma was thrown into large polypoid folds which merged with large mucosal folds on the posterior wall of the gastric remnant.

The patient was re-operated upon and huge folds of gastric mucosa were found to protrude through the gastrojejunostomy stoma into the jejunal lumen. The stoma was opened, the redundant mucosa excised and the gastric mucosa sutured to the jejunal mucosa.

A polypoid mass of gastric mucosa measuring 3.5×2.0 cm. was submitted for pathologic examination. Microscopically, a tremendously thickened mucosa with a loose, edematous connective tissue stroma was observed. There was hyperplasia of the glandular elements with areas of marked cystic dilatation. These changes

were suggestive of giant rugal hypertrophy (Menetrier's disease), but since the gastric mucosa in the first surgical specimen showed only intestinal metaplasia and thinning of the mucosa (chronic atrophic gastritis), it was felt that the edema and hyperplasia in the second specimen were secondary to the herniation of the gastric mucosa.

Comment. Grimoud *et al.*[5] also mention that in 1 of their cases the excised mucosa looked adenomatous with marked glandular hyperplasia. The microscopic appearance of the gastric mucosa excised from the 2 cases reported by Shane *et al.*[14] also showed extensive edema, moderate lymphocytic and plasmacytic infiltration and was diagnosed as benign polypoid hyperplasia.

Case II. E.G. 104-70-61. This 58 year old woman had a partial gastrectomy, Hofmeister-type, and vagectomy for a recurrent gastric ulcer. Several months after discharge, she began to complain of postprandial vomiting of undigested food without blood or bile. She also noted severe mid epigastric pain. Upper gastrointestinal examination performed 5 months after the surgery disclosed a narrow stoma with a lobulated filling defect in the anastomotic jejunal loop representing prolapsed gastric mucosa. There was also evidence of slow gastric emptying (Fig. 1).

The gastroscopist described large erythematous folds protruding into the stoma. At the time of surgery, a narrowed stoma was found, through which several large, edematous, erythematous folds of gastric mucosa were prolapsing. The region of the stoma was excised and dropped into formalin. On receiving the fixed specimen, the pathologist could not identify any prolapsed gastric mucosa.

Comment. It is of interest that the prolapse of gastric mucosa which was observed by the radiologist on 2 separate occasions and confirmed by the gastroscopist and surgeon, was not found by the pathologist. Since the latter only observed the fixed specimen, the explanation must be the tissue shrinkage incident to the fixing process.

Case III. J.W. 152-25-28. A partial gastrectomy, Hofmeister-type, was performed in this

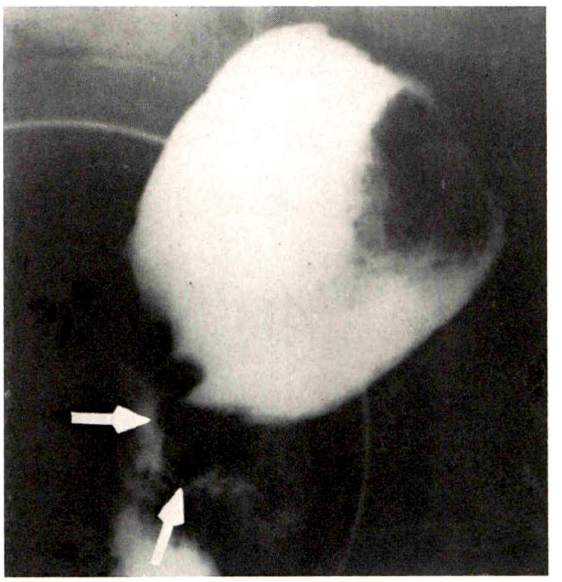


Fig. 7. Prolapsed gastric mucosa causing partial obstruction at the stoma as evidenced by the dilated gastric remnant and retention of food.

53 year old man because of a duodenal ulcer. This was followed by a transthoracic vagectomy for a marginal ulcer 6 months later. Because melena recurred 5 months after the second operation, roentgenographic examination of his upper gastrointestinal tract was requested and it showed herniated gastric mucosa into the anastomotic stoma (Fig. 11). Re-examination 14 months later showed no change.

Two years later a laparotomy was done because of continued bleeding. The region of the anastomosis was carefully palpated and no abnormalities were noted. The next day, the patient was re-explored and a bleeding point found in the efferent loop. *En bloc* excision of the efferent and afferent loops was done. No mucosal lesion was found.

Case IV. J.B. 119-11-11. A 70 per cent Hofmeister-type partial gastrectomy was performed at age 34 in this man because of a duodenal ulcer. Four years later, a marginal ulcer was treated by transthoracic vagectomy. Prolapse of the gastric mucosa was present, in retrospect, on an examination done 2 months after his second operation.

He then did well for 6 years when a third upper gastrointestinal examination was ordered because of dysphagia. The size of the prolapsed gastric mucosa was three times larger. At this

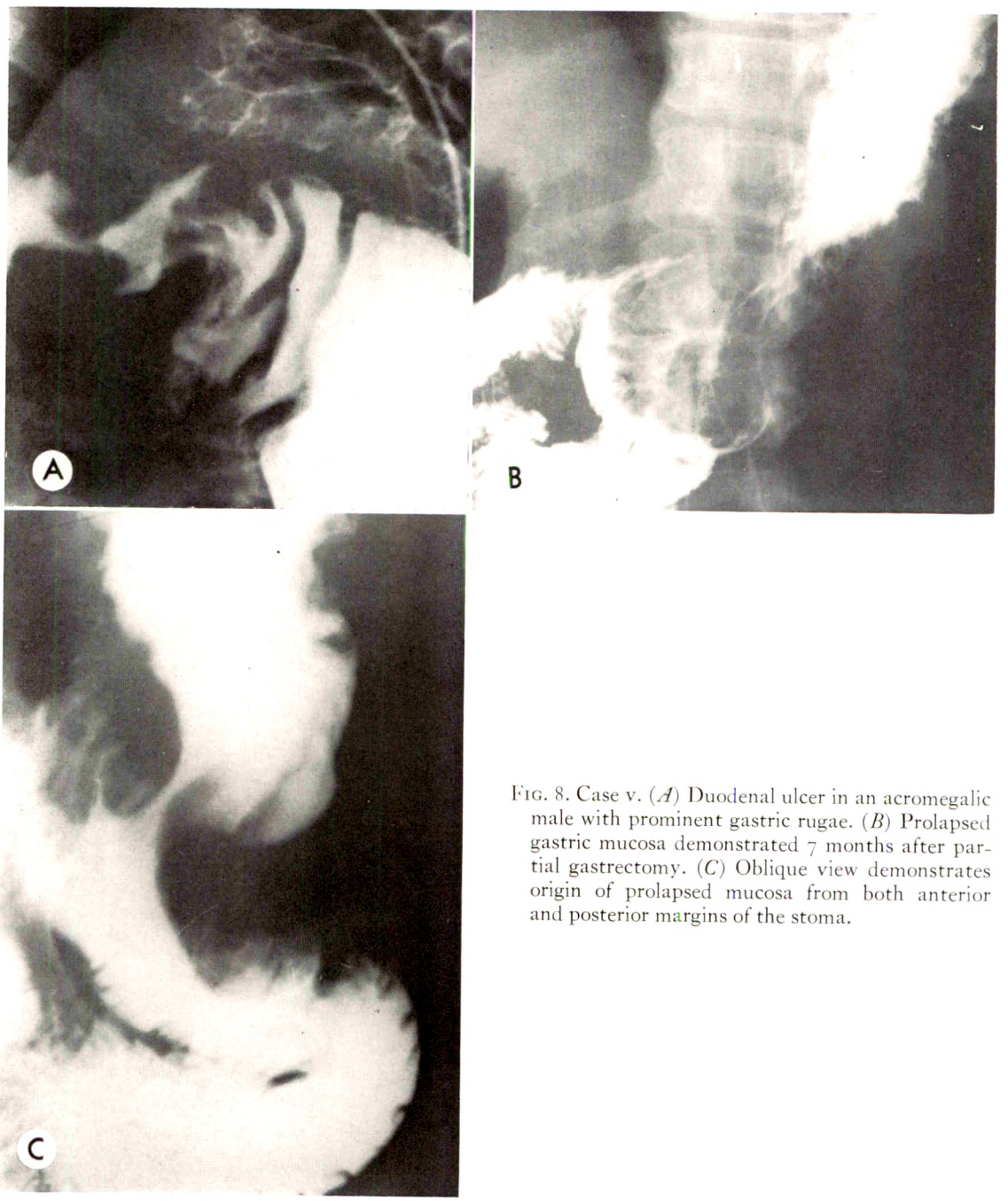


FIG. 8. Case v. (*A*) Duodenal ulcer in an acromegalic male with prominent gastric rugae. (*B*) Prolapsed gastric mucosa demonstrated 7 months after partial gastrectomy. (*C*) Oblique view demonstrates origin of prolapsed mucosa from both anterior and posterior margins of the stoma.

time, there was evidence of a mild anemia and guaiac positive stools. The stoma and both afferent and efferent loops were visualized gastroscopically and no abnormalities observed, although re-examination by roentgenography again demonstrated prolapsed gastric mucosa.

A laparotomy was carried out 2 weeks later for cholelithiasis. The gastrojejunostomy stoma was normal to palpation although the bowel was not opened. He continued to complain of symptoms compatible with "dumping" but no additional gastrointestinal examination or laboratory studies have been done.

Comment. Because the prolapsed mucosa is soft and flat, it is not surprising that it

may not be palpable externally. Shane *et al.*[14] state that the prolapsed mucosa may retract completely into the gastric pouch. In 1 of their patients, even though the jejunum was open, no abnormality was seen until the operating table was tilted producing the prolapse. It seems evident that the diagnosis can not be eliminated surgically unless the jejunum is opened and the patient elevated. Manning and Gunter[11] expressed similar thoughts about prolapse of gastric mucosa into the duodenum when they noted that external palpation at the time of laparotomy frequently failed to demonstrate the mucosa and that it was necessary to inspect the mucosa directly.

Case v. C.N. 143-86-94. A Billroth II, Hofmeister-type, partial gastrectomy was performed on this diabetic Negro male at age 39 because of duodenal ulcer. Acromegaly was diagnosed at age 36 and a parathyroidectomy carried out at 42 years of age for hyperparathyroidism.

Ever since the gastric surgery, the patient had complained of recurrent postprandial epigastric distress and nausea without vomiting or weight loss.

The first upper gastrointestinal examination showed a deformed duodenal bulb with an ulcer niche and prominent gastric rugae throughout the entire stomach which may have been a manifestation of his acromegaly (Fig. 8*A*).

Seven months after the gastric surgery, reexamination demonstrated herniation of the gastric mucosa into the anastomotic jejunal loop measuring 7 cm. in diameter (Fig. 8*B*). In the appropriate projection, this could be shown to have arisen from both the anterior and posterior walls (Fig. 8*C*).

Three years later (Fig. 8*D*), the prolapsed mass had increased and the patient complained of obstructive symptoms of moderate severity which were confirmed roentgenographically by the delayed emptying of the gastric remnant (Fig. 8*E*).

The last examination, more than 5 years after the initial demonstration of the prolapsed mucosa showed very little change. The patient's obstructive symptoms are slowly progressing. Surgery was recommended but refused by the patient.

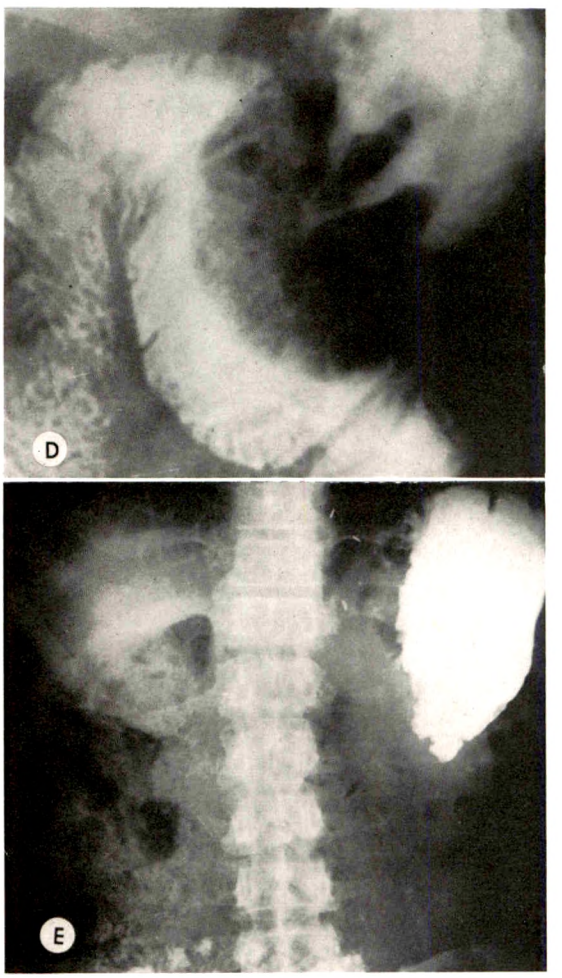


Fig. 8. (*D*) Slight increase in size 3 years later. (*E*) Abnormal retention of barium in gastric pouch on a 1 hour roentgenogram. Patient developing symptoms of obstruction.

DISCUSSION

The gastric mucosa has now been observed to prolapse through every available orifice, although the exact pathogenesis of this phenomenon is unclear.[1,11–13,15] Certainly, redundancy, unusual laxness of the underlying submucosa and mucosal hypertrophy are all important factors, although the latter can apparently be the result of the prolapse, as was suggested in Case 1.

The basic mechanism for the production of prolapsed gastric mucosa is the free movement of the mucosa over the underlying muscular layers as initially empha-

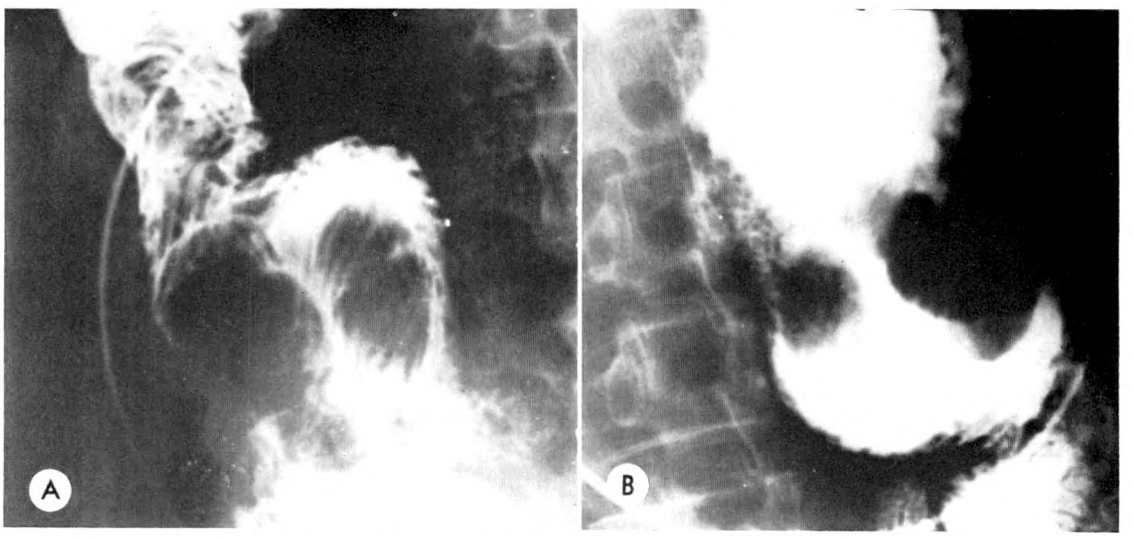


Fig. 9. (*A*) Typical symmetric double, smoothly contoured filling defects produced by prolapsed gastric mucosa. (*B*) Note change in size and appearance produced by variations in peristalsis, compression and amount of barium.

sized by Forssell.[2] The looseness of the submucosa increases with age, permitting greater redundancy of the mucosa and this probably partially accounts for the prominent antral mucosal patterns seen in older patients. Prolapse of gastric mucosa into the duodenal bulb is abetted by the vigorous peristaltic activity of the antrum. However, motor activity is practically



Fig. 10. Classic intraluminal filling defect produced by prolapsed gastric mucosa occurring 23 years after a partial gastrectomy.



Fig. 11. Case III. Small protrusion of gastric mucosa into the jejunum in a patient with gastrointestinal bleeding.

never observed in gastric remnants which function merely as a passive reservoir and conduit.

Levine *et al.*[9] suggest that the smaller stoma created by the Hofmeister gastrojejunostomy lends itself more readily to mucosal prolapse than the larger stoma of the Pólya gastrojejunostomy. They also postulate that the spur created by partially closing the gastric stump may act as a leading point. Only 2 of their 14 cases were of the Pólya type. Of the 11 patients in our series in whom the information was available, all had had the Hofmeister-type of gastrojejunostomy. We have also seen prolapse of the gastric mucosa into the duodenum following a Billroth I partial gastrectomy.

The differential diagnosis is not difficult since the roentgenographic appearance is so typical. The most important consideration is cancer, particularly if the initial operation was done for malignant disease. The softness and flexibility of the lesion should be sufficient to exclude this possibility. Retrograde jejunogastric intussusception is another possible source of confusion, but, in this condition, the mass is in the gastric remnant as well as the jejunum and may exhibit fine, parallel striations due to the small intestinal mucosal pattern. Poppel[13] described a case of gastrojejunal intussusception into the efferent loop produced by a larger gastric leiomyoma whose roentgenograms were very similar to prolapse of gastric mucosa. True gastrojejunal intussusception is extremely rare and is associated with a very short gastric stump and a dilated jejunum containing it.

SUMMARY

Twenty-four patients with prolapse of gastric mucosa through a gastrojejunostomy stoma following a partial gastrectomy were studied.

The roentgenographic appearance is quite characteristic.

Although many of these patients are asymptomatic, prolapse of the gastric mucosa may be a cause of stomal obstruction or gastrointestinal bleeding.

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Case 1 is included by courtesy of Dr. John Prudden.

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THE GASTROCAMERA—A METHOD FOR *IN VIVO* ROENTGENOLOGIC-PATHOLOGIC CORRELATION*

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ALTHOUGH the roentgenographic examination remains the most important diagnostic method in the investigation of disorders of the gastrointestinal tract, additional aids are often employed.[1] Gastroscopy with the gastrocamera has become an increasingly important and more frequently employed diagnostic procedure in the investigation of gastric lesions.[3,4,7] We, as radiologists, should become familiar with the photographic appearance of the stomach as it is recorded by the gastrocamera and should develop an understanding of the advantages and shortcomings of the technique. In some institutions the radiologists may even perform the examination.[6]

The first intragastric camera was described in 1898, but technical problems were great and the procedure was abandoned. There had been several other unsuccessful attempts to develop an instrument for the purpose of photographing the interior of the stomach *in vivo* until 1950, when the Olympus Optical Company of Tokyo perfected such an instrument. After several years of modification, it was introduced at the First World Congress of Gastroenterology in 1958.[7] Since then the instrument has become smaller and more flexible, and in 1964 a combination gastrocamera-fiberscope was devised.

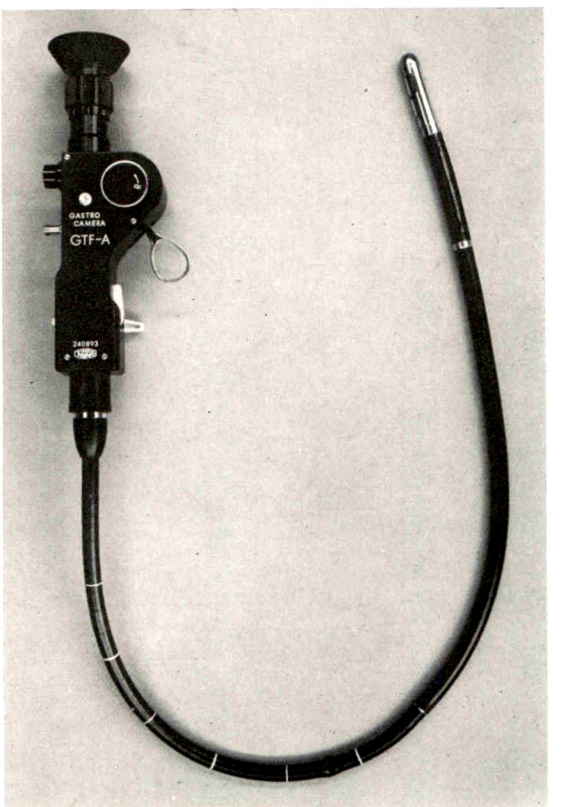


FIG. 1. *Gastrocamera (GTF-A).* Camera proper containing visual lens; photographic lens and film capsule are in the tip; and control unit is at proximal end.

INSTRUMENT

The instrument currently in use in our institution, the GTF-A, incorporates a 3 cm. camera proper containing visual lens, photographic lens, film capsule, and lamps for both viewing and photography in the tip of a tube which is connected to a control unit by an 85 cm. vinyl covered tube which is 10.2 mm. in diameter. The lens has a

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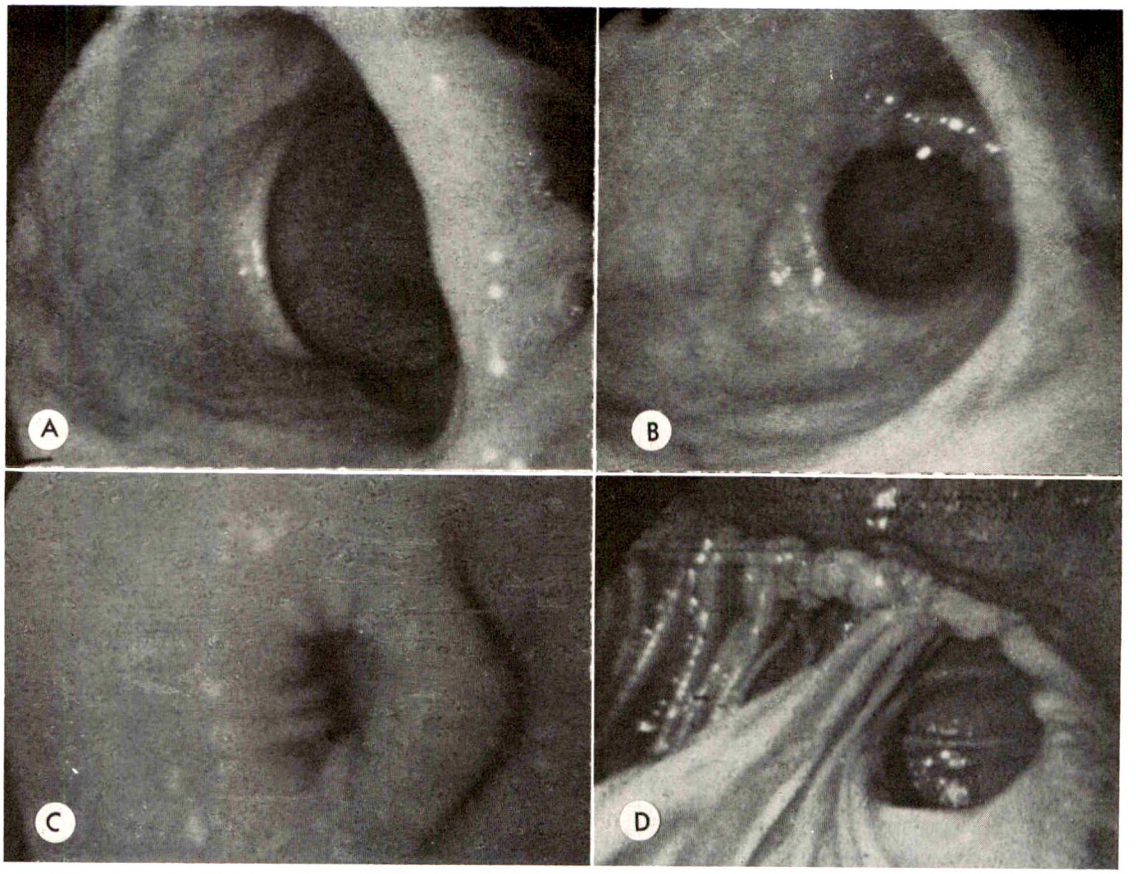


FIG. 2. (*A*) *Gastrocamera view of the gastric antrum.* The camera is in the body of the stomach viewing the antrum. The broad band of tissue on the right is the incisura. The greater curvature is on the left, the lesser curvature on the right. The anterior wall is superior and the posterior wall inferior. (*B*) *Distal gastric antrum.* The orientation is as described in *A*. The darker area in the center represents the more distal antrum *en face*. (*C*) *Normal pylorus.* The folds encircling the opening represent a contraction wave. (*D*) *Normal Billroth II anastomosis.* The ridge in the middle of the picture is the anastomotic ring. All of the area beneath this to the left of the picture is jejunum. The gastric remnant is above.



3.6 mm. focal length and allows clear photography of objects between 2 and 10 cm. from its front surface. The camera lens gives an angular coverage of 80°. The tip of the gastroscope containing the camera can be made to move 70° upward or downward and thus provides the means to sweep the entire stomach with proper manipulation. The film width is 4 mm. and the individual picture size is 4 by 5 mm.

FIG. 3. *Benign gastric ulcer.* Upper gastrointestinal study shows a large crater on the posterior wall of the stomach. (*Insert*) A large crater with smooth margins is seen in the lower portion of the gastrocamera frame. Folds radiate right up to the edge of the crater. The other darkened area is the more distal antrum *en face*.

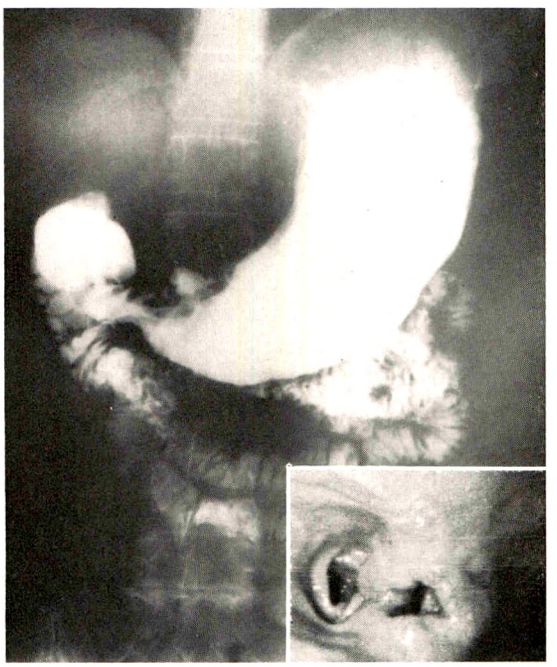


Fig. 4. *Malignant gastric ulceration.* An irregular lesser curvature antral ulceration with a suggestion of nodularity at its base is seen on the barium study. (*Insert*) Gastrocamera view shows triangular shaped ulcer with a nodular heaped-up margin on the right. The pylorus is on the left.

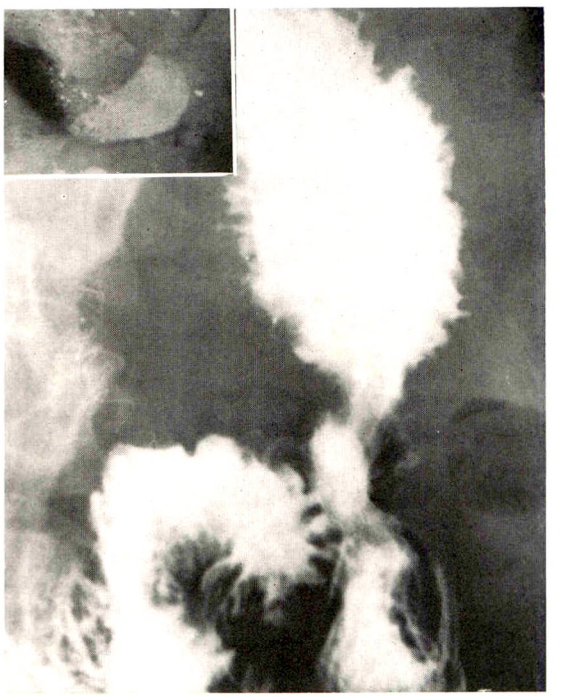


Fig. 5. *Marginal ulcer.* Suggestion of a large ulceration in the area of anastomosis in this patient who is after subtotal gastrectomy for ulcer disease. (*Insert*) A large, antacid filled crater with edematous margins is seen. The darker area represents one of the loops of the enterostomy on end.

MATERIAL

Since the procedure was first introduced in our institution, approximately 75 gastrocamera examinations have been performed. Examples are shown in Figures 1 through 11. It must be kept in mind that these are black and white reproductions of the gastrocamera color films and are magnified many times the 4 by 5 mm. picture of the film strip. This results in some loss of detail and magnification of artifacts.

METHOD

Preparation of the patient is simple. The evening prior to the examination, the patient receives a clear liquid supper and then N.P.O. Occasionally, gastric lavage is required in patients with gastric bleeding or pyloric obstruction. Premedication is suggested before the procedure. Topical pharyngeal anesthesia is obtained with 1 per cent Xylocaine.

After passage of the instrument, the stomach is insufflated with air and the patient is placed on his left side. The cam-

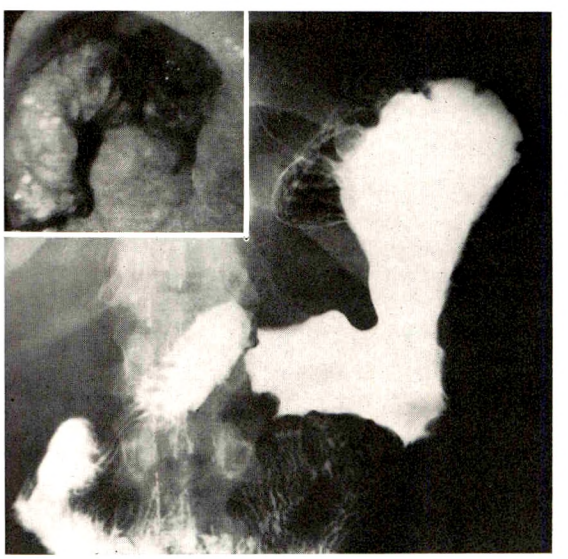


Fig. 6. *Carcinoma of the stomach.* Mucosal destruction, nodularity, and fixation along most of the greater curvature were seen on the barium study. (*Insert*) Gastrocamera view shows massively and irregularly infiltrated folds in most of the stomach.

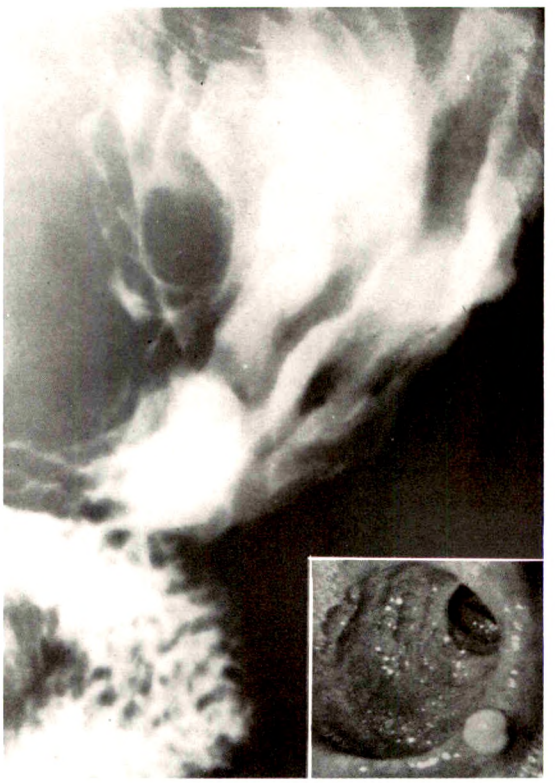


FIG. 7. *Adenomatous polyp.* This 33 year old female had a subtotal gastrectomy for ulcer disease. Postoperative barium study showed a filling defect in the gastric remnant questionably related to previous surgery. (*Insert*) Actual polyp rather than plication defect was found on gastrocamera examination.

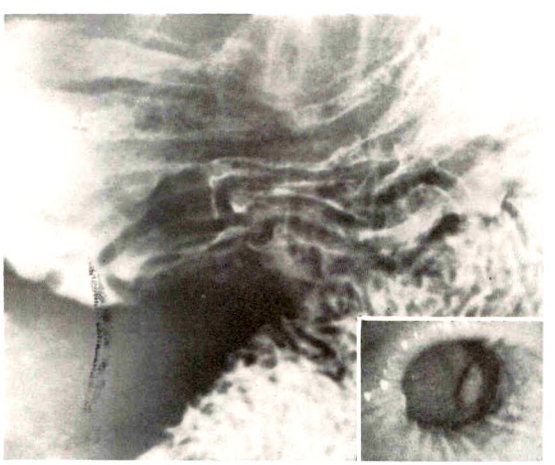


FIG. 8. *Pancreatic rest.* Small submucosal filling defect is seen in the antrum. (*Insert*) Smooth lesion in the antral region. Contraction wave surrounds the antrum.

era will then slide along the greater curvature and photographs are taken at various levels from pylorus to cardia. If a plan is devised ahead of time, the 32 exposures in the camera should in most instances survey the stomach in an adequate manner. The examination has been shown to be safe for the patient. In a series of 1,700 patients of Morrissey *et al.*,[7] there were no perforations. The only absolute contraindications of gastroscopy with this particular instrument are a large Zenker's diverticulum, recent myocardial infarction, aneurysm of the thoracic aorta and coma from any cause.

DISCUSSION

A program of gastrocamera examination following gastrointestinal barium studies in selected patients has been in effect in our institution for over 1 year. The availability of this film record can be of value to the radiologist in several ways:

1. The examination can confirm the presence of a questionable lesion suspected from the barium study. In this way, it serves as an almost immediate roentgenologic-pathologic correlative aid and can be especially valuable to the radiologist in training.

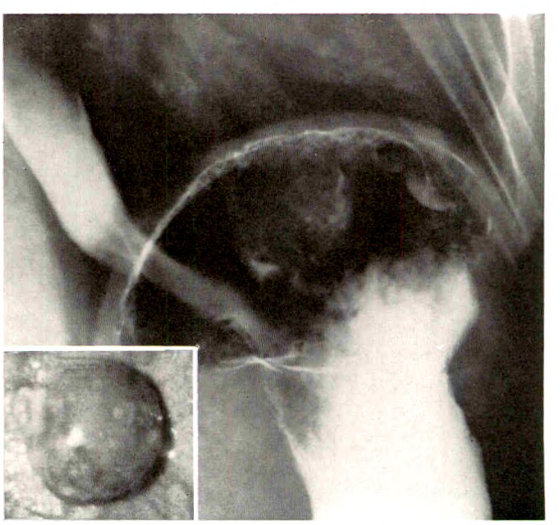


FIG. 9. *Leiomyosarcoma.* A large sessile mass is seen arising from the fundus. (*Insert*) This is a large mass without ulceration and a smooth surface. Color films showed this to be quite vascular.

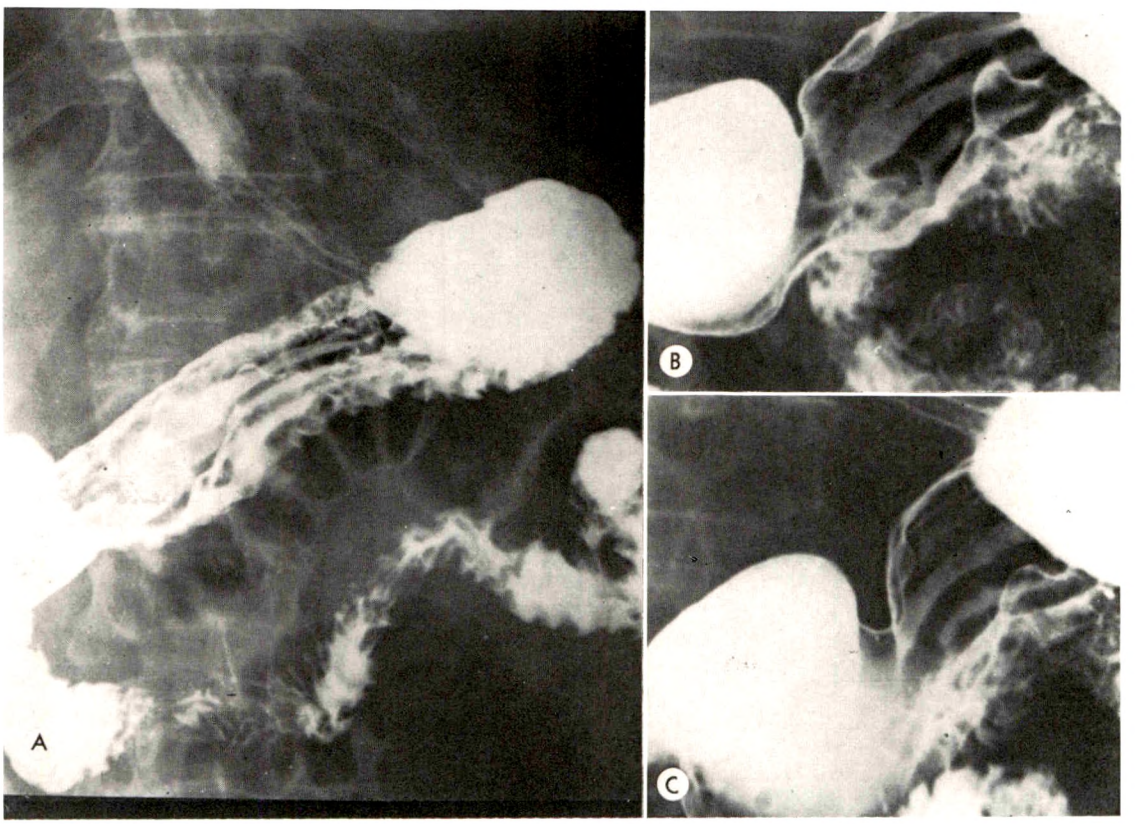


Fig. 10. (*A–C*) *Kaposi's sarcoma.* This 72 year old male with a 3 year history of Kaposi's sarcoma had a recent onset of occult bleeding. Roentgenograms show numerous small submucosal filling defects in the stomach and small bowel.

2. At times, the gastrocamera will allow a more definitive diagnosis than is possible on roentgenograms. For example, the etiology of narrowing and irregularity of the gastric antrum on the roentgenographic examination of the stomach may be unclear. The gastrocamera examination may help to distinguish between neoplasm, redundant folds, or perhaps extrinsic pressure deformity. The examination may also further help to distinguish between ulcerations of benign of malignant origin after the roentgenologic criteria have been applied. In patients with roentgenographic evidence of pyloric obstruction, the gastrocamera examinations may help to differentiate between inflammation *vs.* neoplasm.

The postoperative stomach is notoriously difficult to evaluate roentgenographically. Anastomotic ulcers and tumor recurrences may appear more obvious on gastrocamera examination than on the roentgenograms, where we may find difficulty in distinguishing pathology from changes in anatomy



Fig. 10. (*D*) In addition to multiple submucosal nodules, a macular violaceous eruption on the gastric mucosa is seen which has the same appearance as the patient's skin lesion.

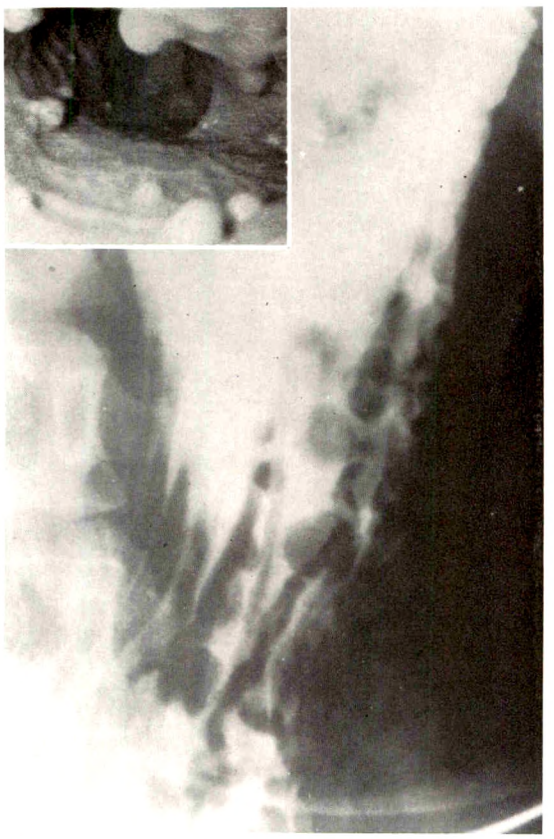


FIG. 11. *Peutz-Jeghers syndrome.* This 19 year old female had numerous small bowel obstructions. Barium studies revealed multiple polypoid defects present in the stomach, small bowel and colon. (*Insert*) There are numerous small tumors of varying sizes in all parts of the stomach. This is a picture taken of the body of the stomach.

secondary simply to the surgical procedure.[2]

3. The study may also show a lesion which may not be suitable for conventional roentgenologic demonstration. Examples are small, shallow ulcerations and possibly small telangiectasias of gastric mucosa.[9] Also fundal abnormalities may be clearly shown, this area often being particularly difficult to evaluate roentgenographically. In these cases, viewing the roentgenograms and gastrocamera results side by side may provide new roentgenologic criteria for lesions previously thought undiagnosable by roentgenography.

Therefore, some of the value of the examination to the radiologist is that it may confirm his roentgenographic impression, it may further explain a lesion or finding he suspects from his study, or it may uncover a lesion that roentgenography is not able to depict in a symptomatic patient. The examination may be of further value in the monitoring of gastric lesions such as gastric ulcers, the complete healing of which may be difficult to assess accurately roentgenographically.[5,7]

The greatest disadvantage of the procedure is the large number of technically unsatisfactory examinations.[4,8] There may be photographic problems with the film being either over- or underexposed. Unfortunately, this does not become apparent until after the examination is completed and the film developed. The endoscopic impression must then be relied upon as the film strip is not available for intensive study. Overlapped frames may also be a problem.

Although theoretically with the newer instruments it is possible to survey the entire stomach, full coverage may not be obtained because of distortions in anatomy due either to unusual normal variation or distortion secondary to disease or infiltration.

Blurring of the lens by mucus may be a factor in a suboptimal examination. In some cases, lack of patient cooperation is a factor as in inability to distend the stomach sufficiently with air. The instrument is also fragile and expensive to repair.[4]

SUMMARY

A selection of gastrocamera studies of a variety of gastric abnormalities is presented in the hope of familiarizing the radiologist with this increasingly popular visual diagnostic instrument.

The advantages and shortcomings of the technique are discussed.

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ANGIOGRAPHIC FINDINGS IN DISEASES OF THE STOMACH*

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BARIUM study, gastrocamera, biopsy and other methods available for diagnosis of gastric diseases have made a remarkable progress in recent years. These diagnostic methods are intended to examine the gastric wall from the mucosal aspect. On the other hand, angiography is employed for the purpose of observing the state of the gastric wall from the aspect of the serous membrane. The combined use of these 2 different diagnostic procedures makes it possible to obtain an accurate diagnosis of gastric diseases, as regards their clinical development including the site of the lesion and the infiltration.

It is the purpose of the authors to report the angiographic findings of gastric diseases, and discuss the clinical significance in comparison with the histopathologic assessment.

MATERIAL AND METHOD

The gastric arteries consist of the branches from the celiac axis: left and right gastric arteries, gastroduodenal artery, short gastric arteries, and left and right gastroepiploic arteries.

Selective celiac angiography was performed in 34 patients with gastric cancer, 10 patients with gastric ulcer, and 5 patients with other gastric diseases that were subsequently proved by surgery or autopsy.

The Seldinger technique of catheter introduction from the femoral artery was used, and a preformed green Kifa catheter was inserted into the celiac axis and occasionally the superior mesenteric artery.

Prior to the injection of the contrast medium (urografin 76 per cent), effervescent tablets were administered perorally in order to distend the gastric cavity.[10]

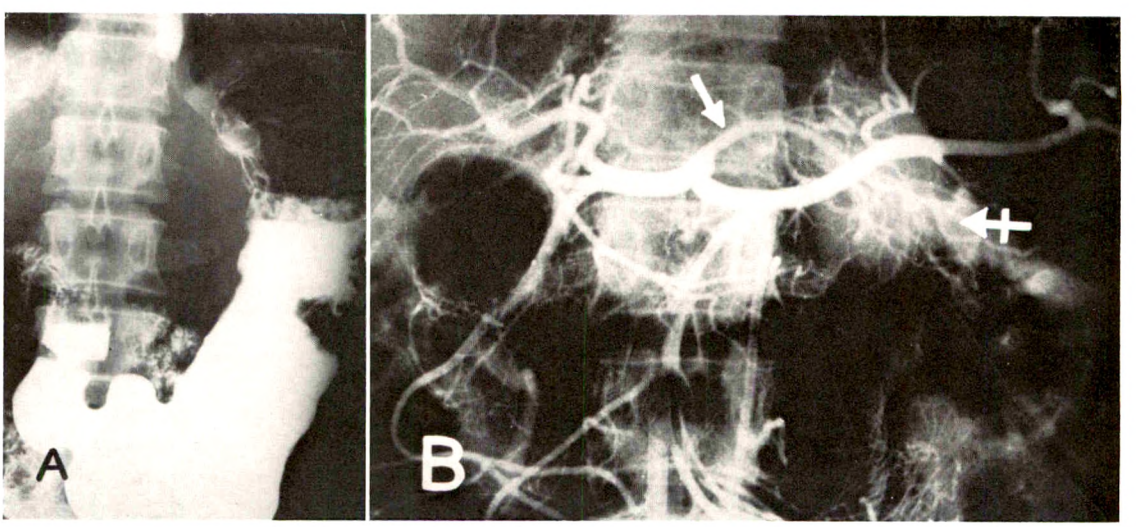


FIG. 1. A 35 year old woman with tubular adenocarcinoma of the cardia. (*A*) Roentgenogram taken in upright position. Coarse irregularity is observed on the lesser curvature in the cardiac portion, with an incisura demonstrated on the greater curvature. (*B*) *Arterial phase of superior mesenteric arteriography.* Because of complete obstruction at the orifice of the celiac axis, the branches of the celiac axis are visualized via the pancreaticoduodenal and transverse pancreatic arteries. The left gastric artery (↑) is distinctly recognized showing neoplastic vascularity, and a tumor stain (‡) is demonstrated in the cardia.

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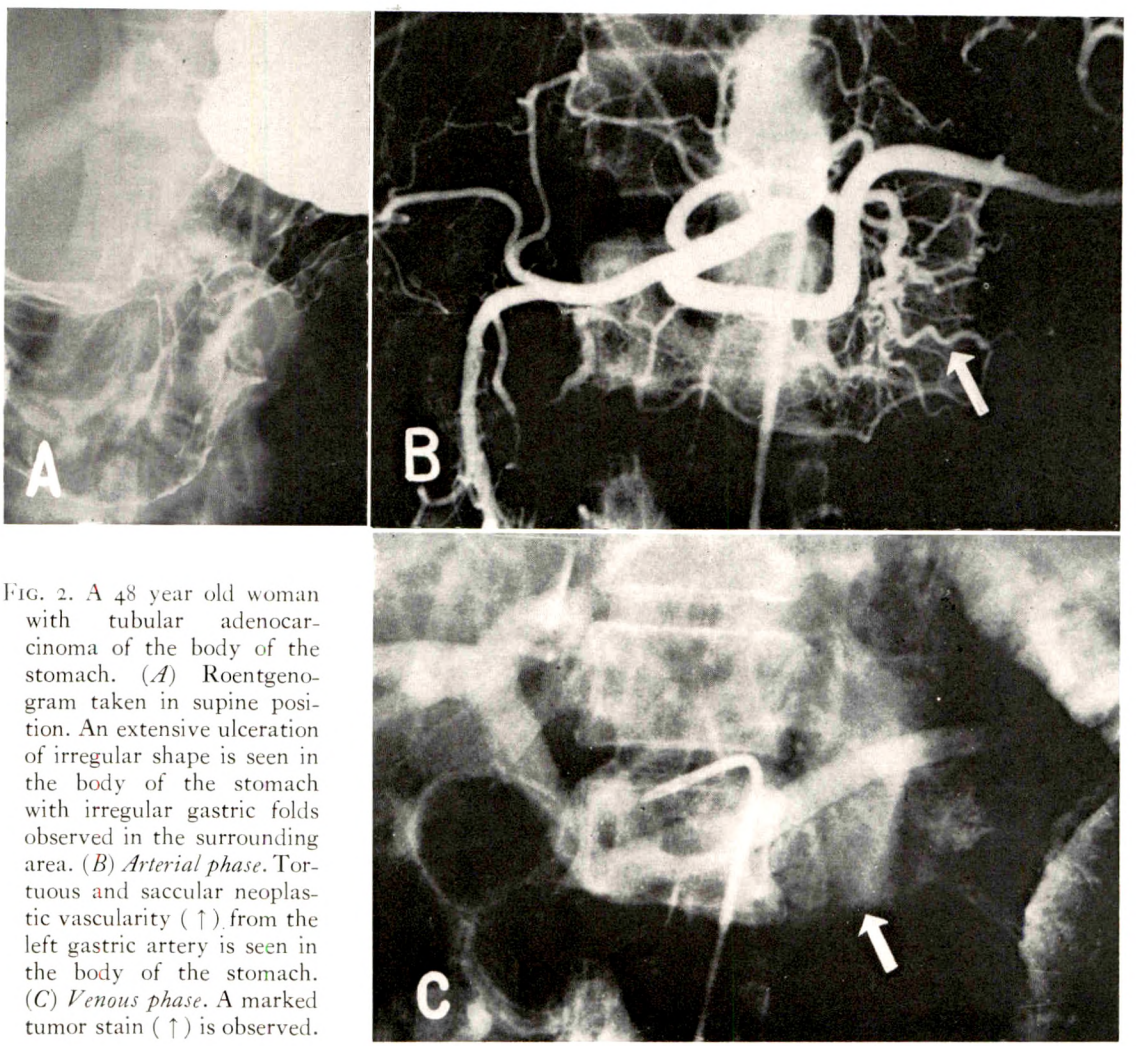


Fig. 2. A 48 year old woman with tubular adenocarcinoma of the body of the stomach. (*A*) Roentgenogram taken in supine position. An extensive ulceration of irregular shape is seen in the body of the stomach with irregular gastric folds observed in the surrounding area. (*B*) *Arterial phase*. Tortuous and saccular neoplastic vascularity (↑) from the left gastric artery is seen in the body of the stomach. (*C*) *Venous phase*. A marked tumor stain (↑) is observed.

Serial roentgenograms were made, with 2 films exposed per second for 5 seconds and 1 film at intervals of 4 seconds for an additional 15 seconds.

RESULTS

Hypervascularity and puddling are very important angiographic findings for the diagnosis of tumor, in the form of neoplastic vascularity and tumor stain,[6] as interpreted in the roentgenograms. The angiographic findings of hypervascularity and puddling may also be observed in cases of inflammatory changes.[4] It is, therefore, necessary to differentiate the hypervascularity and puddling due to tumor from those ascribable to inflammatory changes, and to identify the tumor vessel by observation of all the findings obtained in every phase. An angiogram should be characterized by the pattern of the tumor vessel when it shows a blood vessel giving off numerous and irregular branches, saccular branches and tortuous branches.[9]

As shown in Table 1, of 34 cases of gastric carcinoma, neoplastic vascularity was observed in 31 cases, and tumor stain in 22 cases. The cases were grouped by the site of the lesion. In carcinoma of the cardia, all the cases exhibited both neoplastic vascularity and tumor stain, almost corresponding to the extent of the tumor invasion (Fig. 1, *A* and *B*). In carcinoma of the body of the stomach, neoplastic

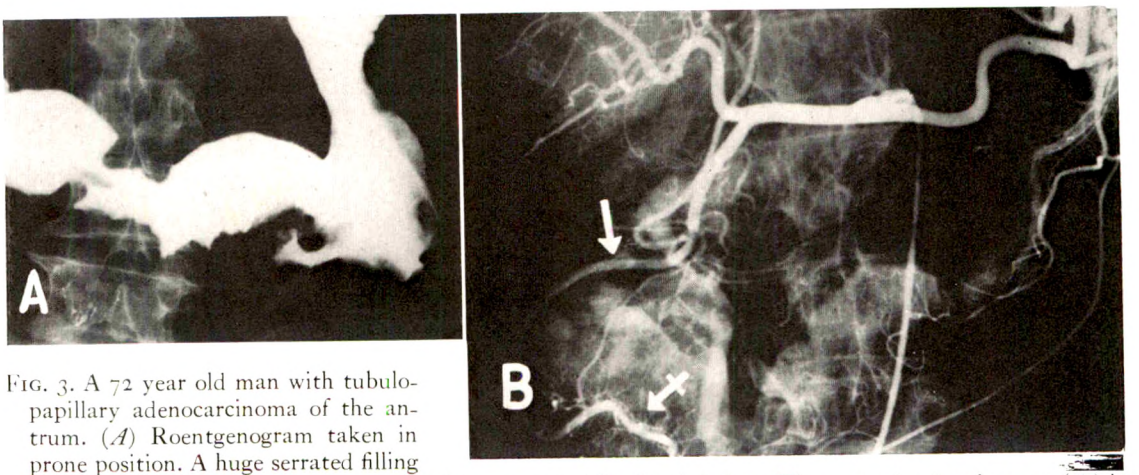


FIG. 3. A 72 year old man with tubulopapillary adenocarcinoma of the antrum. (*A*) Roentgenogram taken in prone position. A huge serrated filling defect is seen on the greater curvature of the antrum. (*B*) *Arterial phase*. The gastroduodenal artery (↑) is displaced to the right side, and the right gastroepiploic artery (↕) shows tortuosity at the involved area. Neoplastic vascularity and marked tumor stain are observed on the greater curvature of the antrum.

TABLE I

SITES OF GASTRIC CANCER AND ANGIOGRAPHIC FINDINGS

	No. of Cases	Neoplastic Vascularity	Tumor Stain
Cardia	9	9	9
Body	9	9	6
Antrum	11	9	3
Pylorus	5	4	4
Total	34	31	22

vascularity was observed in 9 cases, and tumor stain in 6 cases (Fig. 2, *A–C*). In antral carcinoma, neoplastic vascularity was found in 9 cases, and tumor stain in 3 cases (Fig. 3, *A* and *B*). Although the presence of tumor may be indicated by neoplastic vascularity, it was generally difficult to demonstrate the whole tumor tissue of the main lesion in the antrum as a tumor stain. There were occasional cases which exhibited neither neoplastic vas-

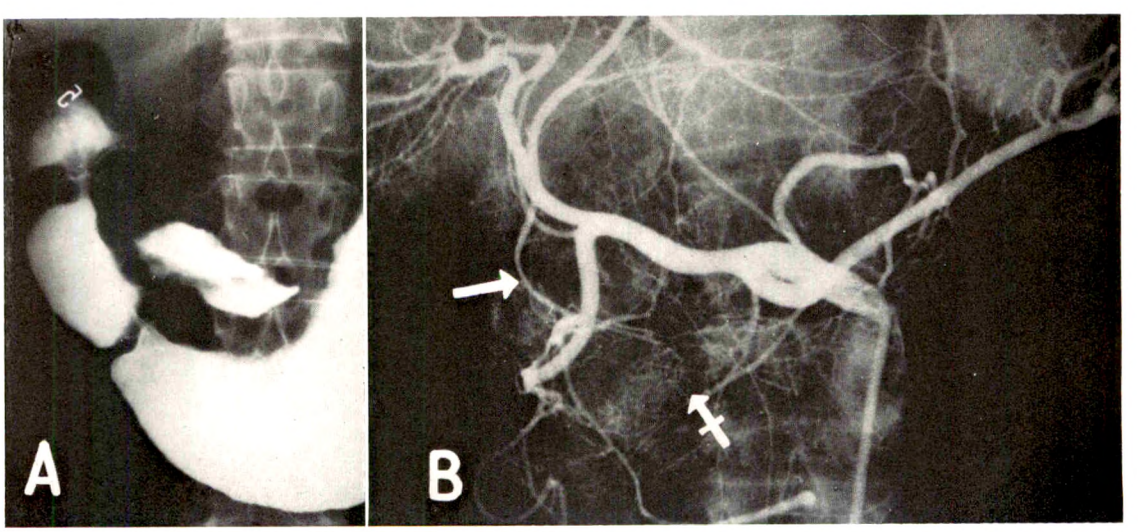


FIG. 4. A 68 year old woman with tubular adenocarcinoma of the antrum. (*A*) Roentgenogram taken in prone position. A large crater is observed in the lesser curvature of the antrum. (*B*) *Arterial phase*. The right gastric artery (↑) is displaced inferiorly and is disrupted by neoplastic vascularity of the lesion. Also fine irregular vessels (↕) are demonstrated in the surrounding of the lymph nodes. The gastroduodenal and pancreaticoduodenal arteries are irregularly narrowed.

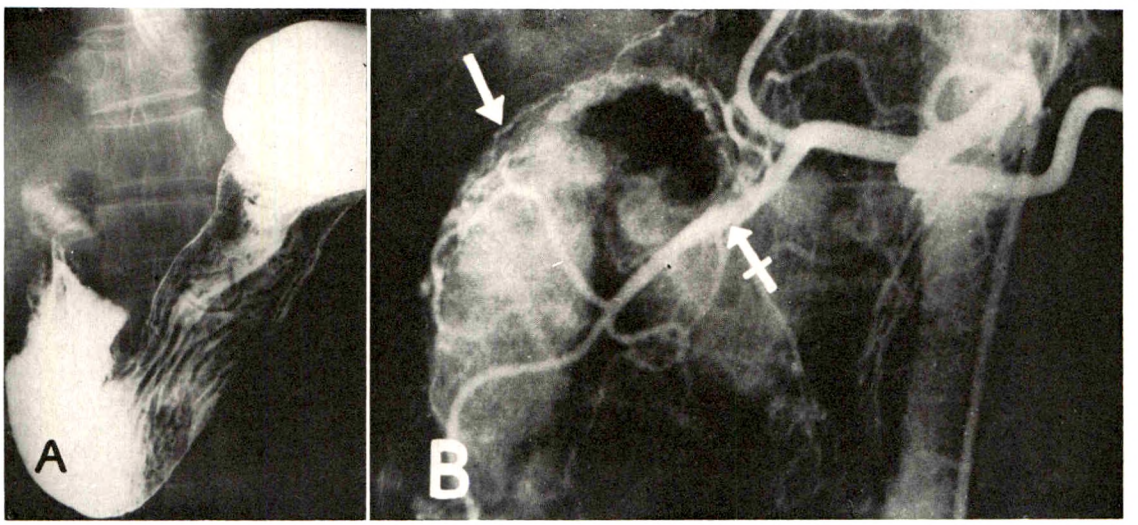


Fig. 5. A 65 year old woman with tubulopapillary adenocarcinoma of the pylorus. (*A*) Roentgenogram taken in supine position. A filling defect is seen in the pyloric portion, and its margins are irregular. (*B*) *Arterial phase*. A well defined tumor stain (↑) is observed in the regions of both lesser and greater curvatures of the pylorus, and the gastroduodenal artery (‡) is slightly displaced to the right side.

cularity nor tumor stain. In antral carcinoma with cancerous ulcer appearing as a main lesion, neoplastic vascularity was demonstrated in the area surrounding the ulcer, but no marked tumor stain could be observed (Fig. 4, *A* and *B*). In pyloric carcinoma, neoplastic vascularity was observed in 4 cases, and tumor stain in 4 cases. The tumor stain was marked. Especially when pyloric stenosis was present, a tumor stain often appeared in the area consistent with the portion invaded by carcinoma. Consequently, the extent of tumor invasion in the gastric wall was often clearly demonstrated within the area shown as a filling defect by the barium study (Fig. 5, *A* and *B*). In another case where similar pyloric stenosis was observed, however, neither neoplastic vascularity nor tumor stain was obtained; the angiogram indicated only tortuosity and disruption of the gastric arteries. On laparotomy, a carcinoma was found infiltrating the stomach from the pyloric portion to the antrum and body, which was nonresectable.

It was characteristic that both neoplastic vascularity and tumor stain were exhibited in many cases with carcinoma of the cardia and body of the stomach. Antral carcinoma exhibited neoplastic vascularity in most cases, but tumor stain in only a few. Pyloric carcinoma showed both neoplastic vascularity and tumor stain in all except 1 case.

Histologic diagnosis of the operative specimens was made in 26 cases with gastric carcinoma. The classification included papillary, tubular, and tubulopapillary adenocarcinomas.

As shown in Table II, the histologic diagnosis was correlated with the angiographic findings of neoplastic vascularity and tumor stain. It was characteristic that neoplastic vascularity and tumor stain were obtained in most cases with papillary and tubulopapillary adenocarci-

Table II

Histologic Patterns of Gastric Cancer and Angiographic Findings

	No. of Cases	Neoplastic Vascularity	Tumor Stain
Papillary adenocarcinoma	4	4	3
Tubular adenocarcinoma	15	13	7
Tubulopapillary adenocarcinoma	7	7	7

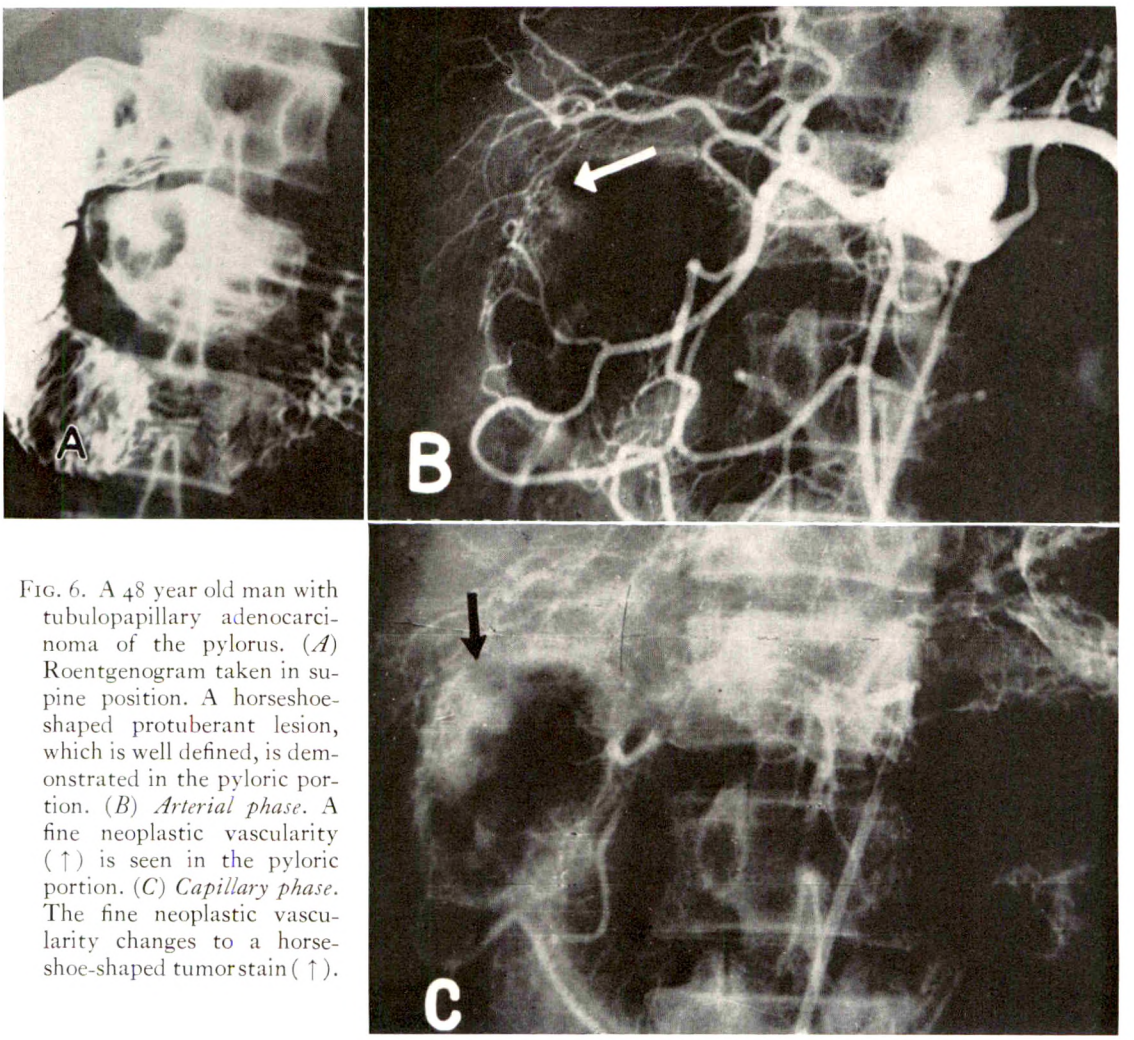


FIG. 6. A 48 year old man with tubulopapillary adenocarcinoma of the pylorus. (*A*) Roentgenogram taken in supine position. A horseshoe-shaped protuberant lesion, which is well defined, is demonstrated in the pyloric portion. (*B*) *Arterial phase*. A fine neoplastic vascularity (↑) is seen in the pyloric portion. (*C*) *Capillary phase*. The fine neoplastic vascularity changes to a horseshoe-shaped tumor stain (↑).

nomas. In tubular adenocarcinoma, tumor stain was demonstrated in fewer cases. However, both neoplastic vascularity and tumor stain were exhibited only in tubular adenocarcinoma in the cases with carcinomas of the cardia and body of the stomach. In pyloric and antral carcinomas, the presence of neoplastic vascularity and marked tumor stain depended on the stage and histologic pattern of the carcinoma.

DISCUSSION

Neoplastic vascularity is demonstrated to be consistently present in the tumor, but does not always appear in combination with tumor stain due to the type of the tumor.

A positive assessment of tumor stain is made when the tumor vessels are recognized and the whole tumor tissue is stained in all phases of angiography. For such a finding, contrast medium should flow in earlier and be retained for a longer period in the tumor tissue than in the normal one; in other words, arteriovenous shunt and absence of vein become prerequisites.[1,7] For this reason, comparison of the angiographic findings in the arterial, capillary and venous phases is important to differentiate malignant disease from a benign lesion.

The diagnosis of carcinoma of the cardia and upper portion of the gastric body can be established on the basis of neoplastic vascularity and tumor stain,[2] whereas it is difficult to diagnose a lesion in these re-

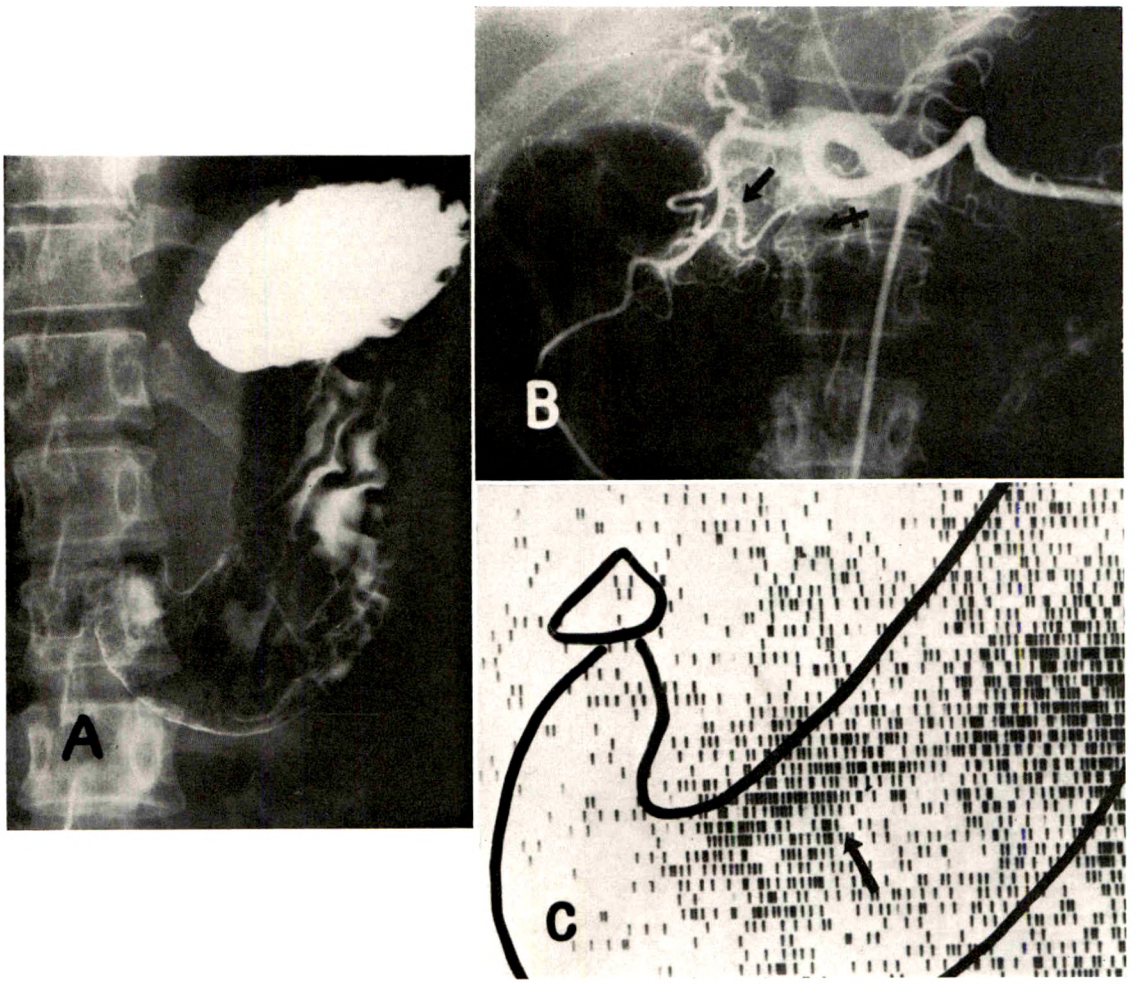


Fig. 7. A 36 year old woman with tubular adenocarcinoma of the body of the stomach. (*A*) Roentgenogram taken in supine position. The mucosal folds of the lesser curvature of the body radiate away from their base in spoke fashion, and the edges of the crater tend to become slightly undetermined. (*B*) *Arterial phase*. The branches from the right gastric artery (↑), slightly dilated, are visualized. There is a fine neoplastic vascularity (‡) but a tumor stain is not recognized at the lesion. (*C*) *Angioscanogram* (after injection of I^{131}-MAA through the celiac axis). A positive scintigram (↑) is obtained consistent with the lesion.

gions by barium study and endoscopic examination. Furthermore, the angiographic findings can disclose lesions that may be unsuspected on the barium study or endoscopic examination, and readily determine the extent of infiltration of the main lesions (Fig. 1, *A* and *B*; and 2, *A–C*). The cardia and the upper portion of the gastric body are supplied by the left gastric artery (larger in caliber than the other gastric arteries), which originates directly from the celiac axis, and by the short gastric arteries (branches of the splenic artery), which are of rather increased caliber;[8] therefore, a large amount of contrast medium flows into these parts of the stomach. Cases of antral carcinoma which do not demonstrate neoplastic vascularity or tumor stain should be considered as being far advanced; the angiographic findings of neoplastic vascularity and tumor stain are replaced by findings indicating displacement, tortuosity, and disruption of the gastric blood vessels. The situation is similar in the pyloric carcinoma. As a possible cause, it may be considered that the

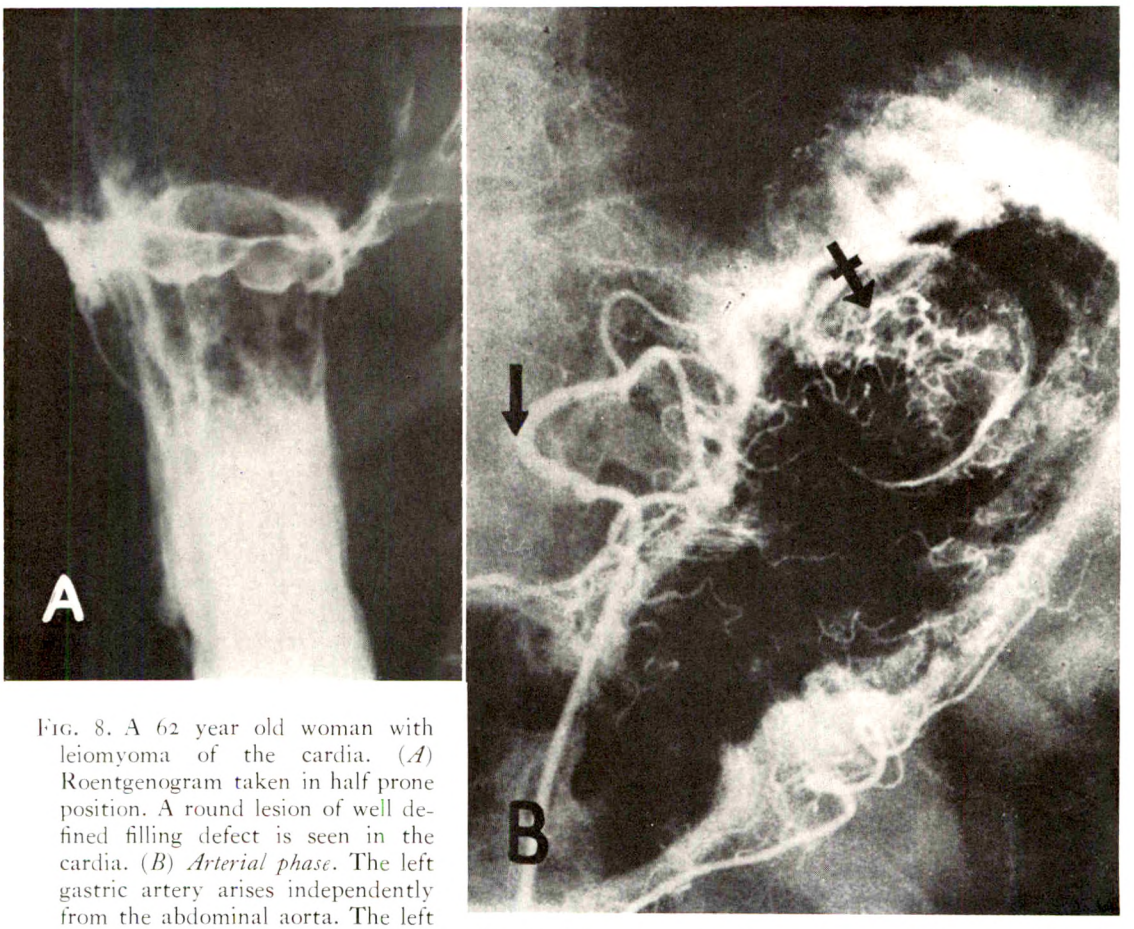


Fig. 8. A 62 year old woman with leiomyoma of the cardia. (*A*) Roentgenogram taken in half prone position. A round lesion of well defined filling defect is seen in the cardia. (*B*) *Arterial phase*. The left gastric artery arises independently from the abdominal aorta. The left gastric artery (↑) supplying the lesion fills rapidly, and neoplastic vascularity (‡) is seen within the lesion. A well defined tumor stain is also demonstrated.

injected contrast medium can flow only in a very small quantity into the pyloric and antral portions of the stomach because they are supplied by the right gastric artery, the smallest in caliber among the gastric arteries, and by the right gastroepiploic artery, the most distal branch from the celiac axis.[8]

A marked tumor stain develops in some histologic patterns of gastric cancer. Papillary adenocarcinoma exhibits a marked tumor stain regardless of its site, thus enabling a clear distinction between the tumor tissue and the normal tissue. Tubulopapillary adenocarcinoma shows a tumor stain before the tumor tissue has been necrotized (Fig. 3, *A* and *B*; and 5, *A* and *B*).

When the tumor tissue grows and develops into an advanced stage in papillary and tubulopapillary adenocarcinomas, it undergoes necrosis in part because of disruption, stenosis and other changes of the gastric arteries, producing a circulatory disturbance. In consequence, neoplastic vascularity becomes scarce and the tumor tissue is no longer recognized as a tumor stain.

In the case of tubular adenocarcinoma, tumor stain cannot be observed in the pyloric and the antral portions, only in the cardia and in the upper portion of the gastric body.

Two cases with early gastric carcinoma that invaded only the gastric mucosa and the submucosal layer are discussed. One had a protuberant lesion and exhibited neoplastic vascularity and tumor stain in the

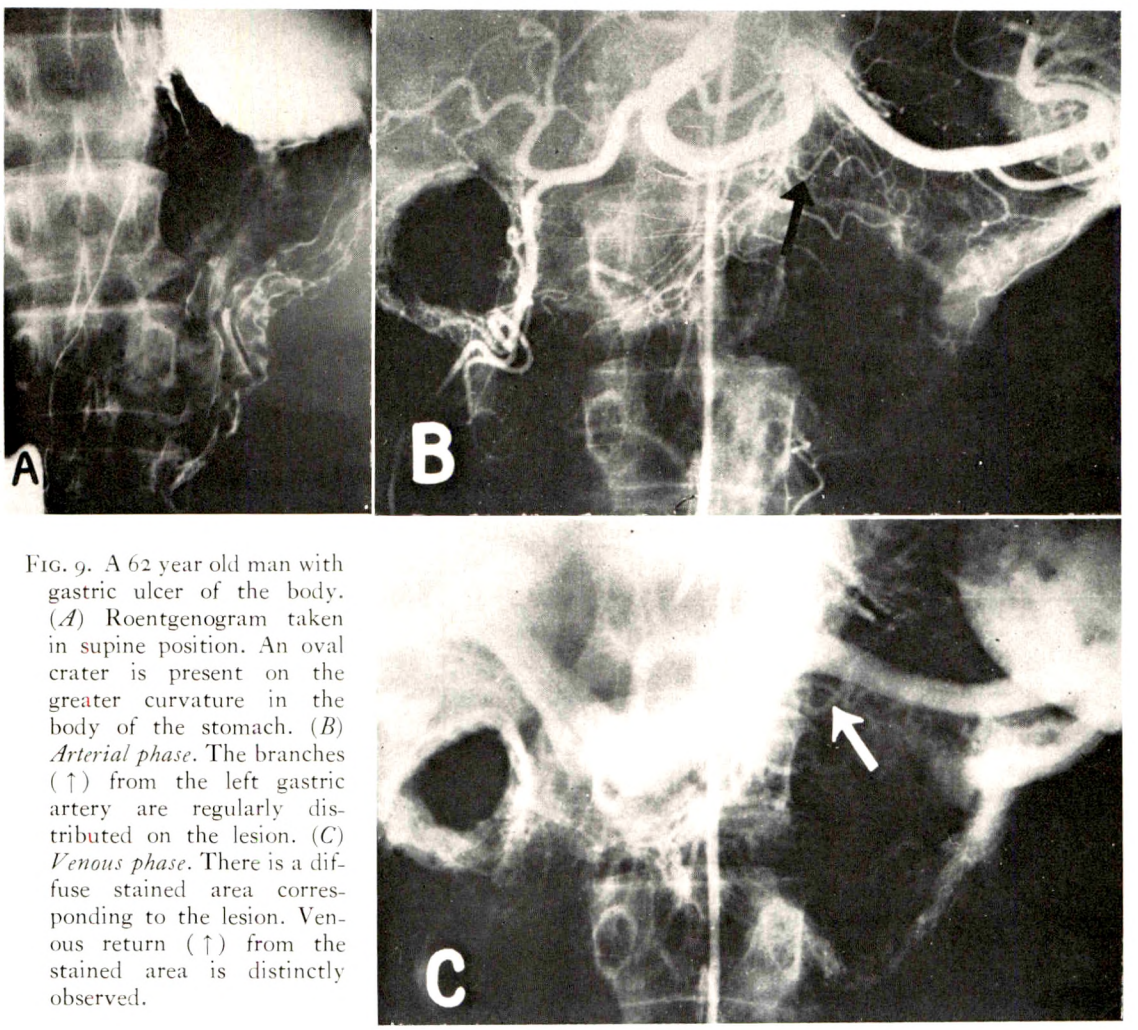


Fig. 9. A 62 year old man with gastric ulcer of the body. (*A*) Roentgenogram taken in supine position. An oval crater is present on the greater curvature in the body of the stomach. (*B*) *Arterial phase*. The branches (↑) from the left gastric artery are regularly distributed on the lesion. (*C*) *Venous phase*. There is a diffuse stained area corresponding to the lesion. Venous return (↑) from the stained area is distinctly observed.

area corresponding to the lesion. This tumor stain was well delineated. No abnormality was demonstrated at all in the venous phase of the gastric wall at the lesion. The histologic diagnosis was tubulopapillary adenocarcinoma (Fig. 6, *A–C*). The other case had a cancerous ulcer, and exhibited neoplastic vascularity in the area corresponding to the lesion in the arterial phase, but no tumor stain. Angioscanography was performed following injection of I^{131}-MAA into the celiac axis, and a positive scintigram was obtained in the area corresponding to the tumor tissue.[5] Angioscanography was very effective in this case since there was no tumor stain (Fig. 7, *A–C*).

Early papillary or tubulopapillary adenocarcinoma showing protuberance is readily diagnosed, proving that early gastric cancer may be recognized by the combined use of angiography and angioscanography.

In advanced gastric cancer, swellings of the regional lymph nodes due to metastases from the main lesion cause displacement, stretching and disruption of the gastric blood vessels, and produce neoplastic vascularity indicating formation of fine irregular vessels in the capsule of the lymph node. However, they fail to show stain (Fig. 4, *A* and *B*). Infiltration into the adjacent organs is indicated by abnormality due to displacement, stretching and other changes of the splenic, pancreatic

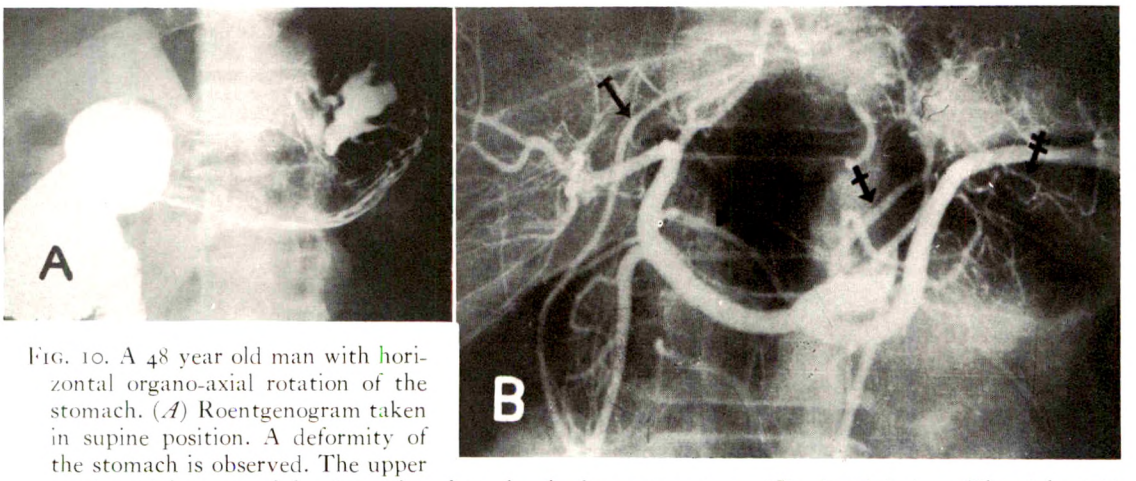


Fig. 10. A 48 year old man with horizontal organo-axial rotation of the stomach. (*A*) Roentgenogram taken in supine position. A deformity of the stomach is observed. The upper portion of the stomach has been thought to be the lesser curvature. (*B*) *Arterial phase* The right (↑) and left (‡) gastric arteries make an arcade in the lower portion of the stomach which has been thought to be the greater curvature; in reality it is the lesser curvature. The right (↑) and left (‡) gastroepiploic arteries make an arcade in the upper portion of the stomach which has been thought to be the lesser curvature; in reality it is the greater curvature.

and hepatic vessels. The angiographic findings of celiac axis are particularly important for diagnostic assessment of hepatic metastases.

Polyp of the stomach was not demonstrated angiographically, while leiomyoma of the stomach was the most vascular tumor. The artery supplying the tumor filled rapidly, and there was early, moderately dense filling of the drainage veins. Many irregular vessels were seen within the tumor and the accumulation of the contrast medium was dense; the tumor had a well defined border (Fig. 8, *A–C*).

The angiographic finding of gastric ulcer was not so greatly different from that of a normal stomach. It is characteristic of the fresh ulcer that the branches of gastric arteries supplying the involved area are a little dilated and show neither irregularity nor stenosis. When an ulcer is healed, the supplying arteries become rather thin as compared with other arteries of the gastric wall. However, if inflammatory change occurs in the serous membrane of an unhealed ulcer, an irregular dense stain is observed in the surrounding area of the ulcer. It may be sometimes difficult to differentiate this angiogram from tumor stain but the differentiation can be made easily by thorough observation of the angiographic findings appearing on all the phases of the stained area. Especially when venous flow of the stained area is demonstrated in the venous phase, can a diagnosis of ulcer be made (Fig. 9, *A–C*).

Although gastric disease can be diagnosed by barium study, we have not been able to differentiate a malignant from a benign lesion in some cases. Angiography of the stomach provides additional information unsuspected on the barium study, and also is effective for making a diagnosis of intramural gastric tumors, extragastric tumors, and such anomalies as organo-axial rotation and volvulus of the stomach (Fig. 10, *A* and *B*).

SUMMARY

Angiography was performed in cases with gastric diseases for the purpose of determining the clinical significance in comparison with histologic assessment.

In carcinoma of the stomach, neoplastic vascularity and tumor stain are demonstrated in the area corresponding to the main lesion. The tumor stain is dependent upon the stage and histologic pattern of the carcinoma. As a carcinoma advances, neoplastic vascularity and tumor

stain are replaced by displacement, stenosis and disruption of the gastric blood vessels.

Angiography of the stomach gives additional information not anticipated from the barium study as regards the gastric serous membrane, and is very helpful in the diagnosis of intramural gastric tumors, extragastric tumors, and such anomalies as organo-axial rotation and volvulus of the stomach.

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PASSING THE CANTOR TUBE IN A PATIENT WITH A GASTROENTEROSTOMY: AN UNUSUAL COMPLICATION*

By HARRY SHULMAN, M.D.
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THE radiologist is often called upon to monitor the intestinal location of the Cantor tube. This report describes an unusual complication of passing the Cantor tube in a patient with a gastroenterostomy, and alerts the radiologist to its roentgenologic appearance.

REPORT OF A CASE

The patient, a 68 year old female, was admitted to The New York Hospital for evaluation of sinus bradycardia. While in the hospital, she developed a small intestinal ileus of undetermined cause, and the Cantor tube was passed to relieve abdominal distention.

A scout roentgenogram of the abdomen was obtained and showed the mercury-filled bag of the distal Cantor tube to be within the second portion of the duodenum (Fig. 1). The clinician was advised that the tube could be advanced in the routine fashion.

A second scout roentgenogram, taken the following day, was interpreted as showing the tube coiled within the stomach (Fig. 2). Further evaluation, however, suggested the possibility of the Cantor tube being coiled through the gastroenterostomy. A hypaque contrast medium

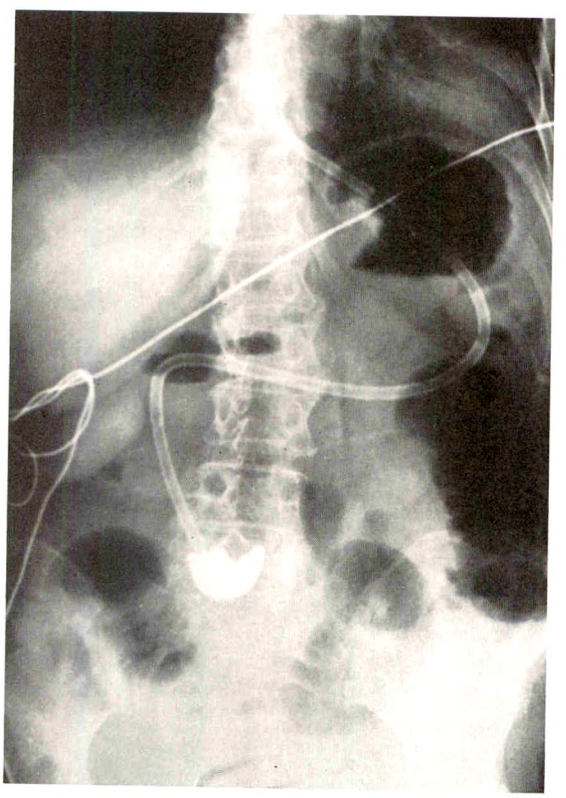


FIG. 1. The mercury-filled bag is in the second portion of the duodenum. The duodenal bulb is gas-filled. The gallbladder is opacified. Distended small bowel loops are present.

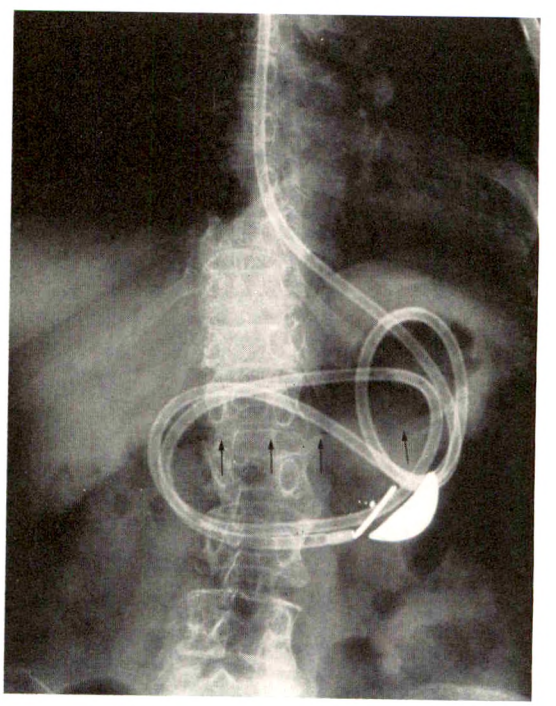


FIG. 2. The Cantor tube was initially interpreted as being coiled in the stomach. The distal stomach in the supine position, however, is air-filled (arrows) and suggests that the 2 caudad loops are outside the stomach and within the duodenum.

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swallow was performed which showed the tube passing into the duodenum and twice re-entering the stomach through the gastroenterostomy, forming the 2 coiled loops seen on the scout roentgenogram (Fig. 3). Attempted bedside removal resulted in a tightening of the coil (Fig. 4).

The loops were unwound under fluoroscopy, and the Cantor tube was removed without surgical intervention.

DISCUSSION

It is customary for the radiologist to monitor the position of the Cantor tube via a scout roentgenogram of the abdomen. Visualization of the mercury-filled bag of the distal tube in the second portion of the duodenum is usually considered sufficient evidence to freely advance the tube. In the presence of gastroenterostomy, however, monitoring should be done more frequently, and possibly with the aid of fluoroscopy, until the mercury bag has passed the site of anastomosis. Should the appearance suggest the possibility of a trapped tube, contrast medium examination is suggested to confirm the entrappment. Attempted removal of the coiled tube under fluoroscopy is the treatment of choice.

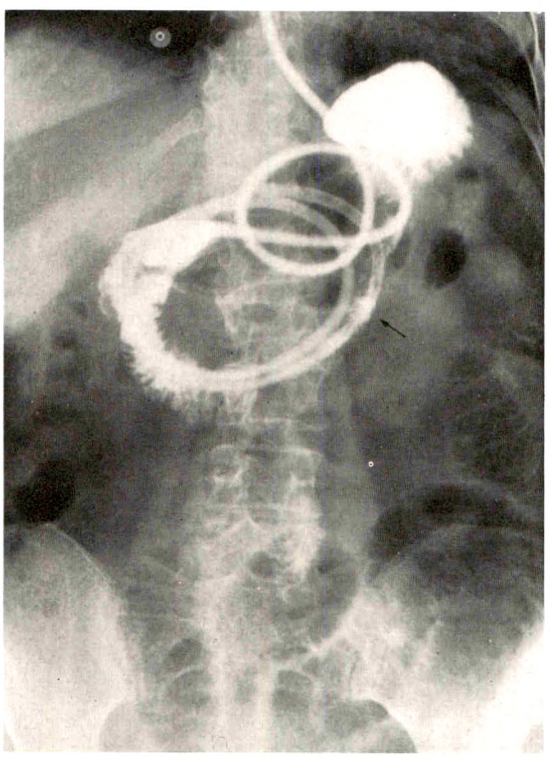


Fig. 3. Hypaque contrast medium swallow shows the Cantor tube passing into the duodenum and twice re-entering the stomach through the gastroenterostomy (arrow).

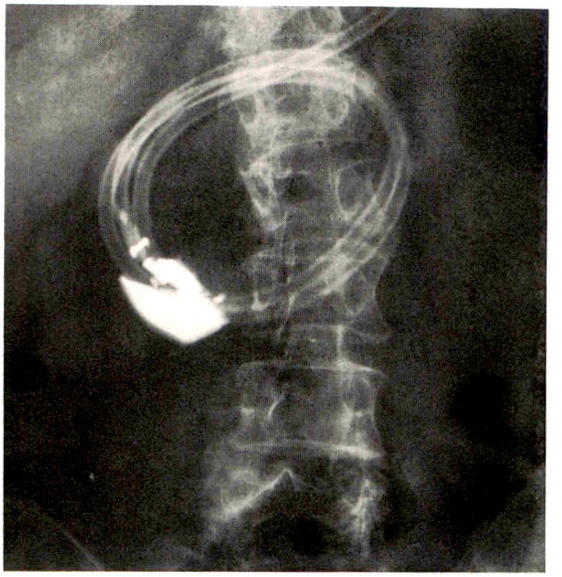


Fig. 4. Attempted withdrawal at the bedside resulted in tightening of the coiled loops.

SUMMARY

An unusual complication of passing the Cantor tube in a patient with a gastroenterostomy is presented, and the radiologist is alerted to its roentgenographic appearance.

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SMALL BOWEL TRANSIT TIME OF WATER SOLUBLE IODINATED CONTRAST MEDIUM AND BARIUM SULFATE IN CATS WITH SIMULATED SURGICAL ACUTE ABDOMEN*

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THE indications and contraindications for the use of water soluble iodine-containing contrast media or barium sulfate in patients in whom small bowel obstruction or adynamic ileus is suspected, have been reviewed repeatedly.[5,7] The subject is controversial as the opinions are divided; yet, the problem is important and common. In postoperative patients particularly, a distinction between adynamic ileus and mechanical obstruction on plain roentgenograms is often difficult, yet barium sulfate often requires prolonged studies.

The transit times of the water soluble contrast medium and barium sulfate through the small bowel have recently been compared in normal dogs.[4] Other studies have compared the effectiveness of these 2 contrast media in the study of experimental, mechanical obstruction in dogs.[6] Comparison of transit times of these media in similar experimental conditions to that of the surgical acute abdomen assumes great importance.

Transit times of bismuth meals in cats before operations and after various surgical conditions were studied more than 40 years ago.[1,8] In the present study, to enable the quantitation and reproduction of hypodynamic ileus simulating adynamic ileus in acute abdominal conditions, we have used a drug (probanthine). Clinically, this drug (probantheline bromide) is used to produce a hypotonic state of the small bowel.[2]

The transit times of each contrast medium from the small bowel to the colon were compared in cats: (a) used as normal controls; (b) given large doses of probanthine; (c) 24 hours after abdominal exploration and handling of the small bowel; and (d) in which small bowel obstruction or perforation, or both of these conditions, were surgically induced.

MATERIAL AND METHOD

Nineteen unanesthetized male and female cats, varying in weight from 4 to 6.5 pounds, were given hypaque, a water soluble, iodine-containing contrast medium (sodium diatrizoate 20 per cent) or U.S.P. barium sulfate, on separate occasions in similar experimental situations. Hypaque, 15 ml., or barium sulfate 30 ml., was introduced through a nasogastric tube into the stomach of the cat and probanthine administered when indicated.

The animals were fasted the night before each examination. Roentgenograms of the small bowel transit of contrast medium were obtained at intervals of from 15 minutes to 1 hour for the first 7 hours and when indicated on the following day. Food or liquid was withheld also during the day of the examination. The cats were immobilized between roentgenographic exposures during the first 7 hours.

The dose of probanthine administered intramuscularly to each cat was 1 mg. per 1½ pounds (0.73 kg.) of body weight. Dilatation of the pupils of the cat about 2 minutes later was an indication to us of effective pharmacologic action and was observed for as long as 7 hours.

Preoperative diabutal was given the animals which underwent abdominal exploration. The surgical procedure on each of 18 cats involved exteriorization and handling of the cecum and the entire small

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Supported in part by a grant from the Sterling-Winthrop Research Institute, Rensselaer, New York.

bowel. A metallic nerve clip was placed in lymph nodes adjacent to the terminal ileum. Mechanical obstruction of the terminal small bowel was accomplished with umbilical tape ties in 8 cats. When perforation was desired, the immediate proximal bowel was punctured with a sterile ear-marker on the antimesenteric half of the bowel in 4 of the cats with induced small bowel obstruction and in 3 without obstruction.

A mixture of barium sulfate and hypaque (15 ml. and 8.5 ml., respectively), with and without probanthine was given to 3 animals. The transit times of this admixture of the contrast media through the small bowel were also compared in the normal state and 24 hours after abdominal exploration.

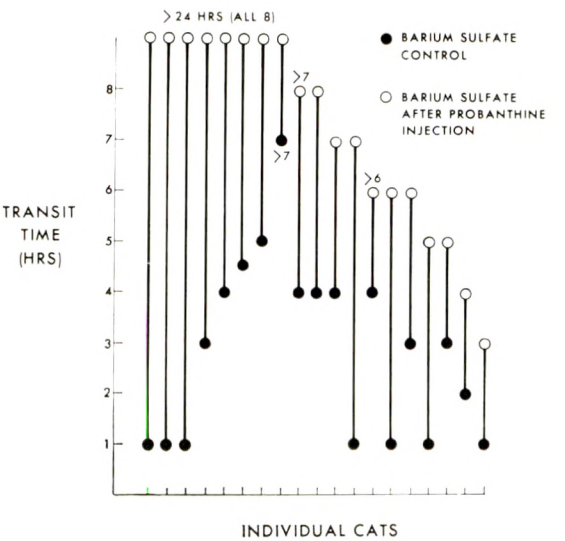


Fig. 2. Small bowel transit times of barium sulfate with probanthine induced adynamic ileus.

RESULTS

The results of the studies are summarized in Figures 1–5. Twofold or greater differences in small bowel transit time in the same cat were considered significant.

In 13 of 19 cats the small bowel transit time increased at least twofold with barium, compared with that from hypaque. In 2 of the 6 remaining cats, the increase in transit time with barium was of lesser magnitude.

The adynamic ileus induced with probanthine resulted in at least twofold increases in individual transit times in all but 2 cats given barium as the contrast medium and in all but 3 when the contrast medium was hypaque. In 8 of the 19 cats with the simulated adynamic ileus, compared small bowel transit times were at least twofold greater with barium than with hypaque. In 2 of the 3 cats in which an equal mixture of both contrast media was used, a twofold increase in transit time was noted with the simulated adynamic ileus.

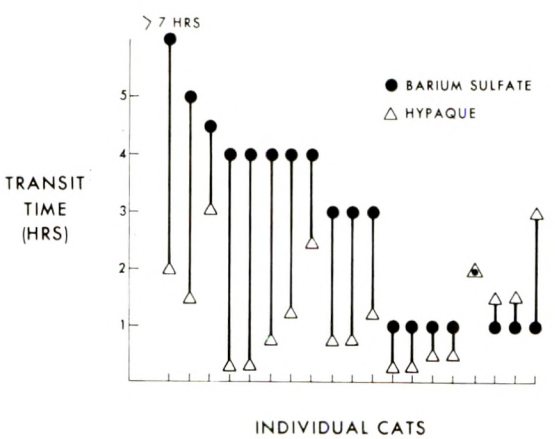


Fig. 1. Small bowel transit times of contrast media in intact cats.

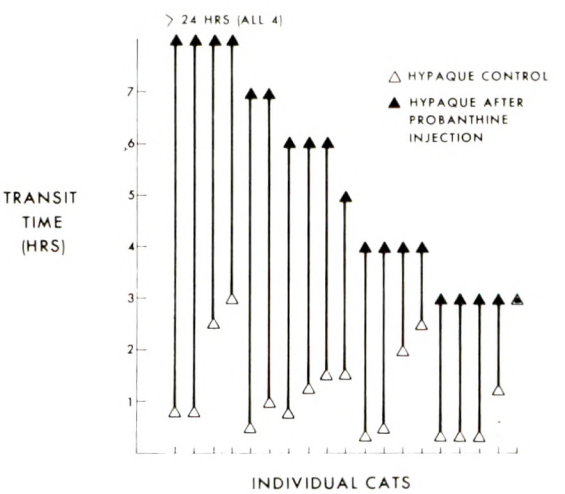


Fig. 3. Small bowel transit times of hypaque with probanthine induced adynamic ileus.

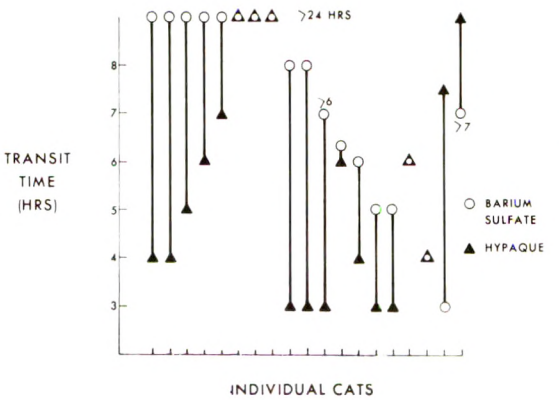


FIG. 4. Small bowel transit times of contrast media with probanthine induced adynamic ileus.

Twenty-four hours after abdominal exploration and handling of the bowel the small bowel transit time of barium sulfate in 7 cats was approximately the same as during the preoperative control study. In 4 of 8 cats examined with hypaque 24 hours after operation, the small bowel transit time of hypaque was prolonged at least twofold as compared with the preoperative control study. At 24 hours the postoperative small bowel transit times with hypaque were shorter than the postoperative transit times with barium.

In 1 of 3 cats receiving an admixture of both media the 24 hour postoperative small bowel transit time was prolonged at least twofold compared to the preoperative study.

In a small series of cats with induced obstruction of the small bowel, or perforation, or both, we observed that barium was the agent that defined the level of obstruction. When barium was administered to the 3 cats with perforation, a barium spill was observed in 1 of 2 cats whose distal small bowel was obstructed at 24 hours. This cat was killed 4 days later and a severe peritonitis was noted. The other cat was maintained on parenteral fluids until killed at 14 days; a severe peritonitis had developed in this cat also. The third cat with perforation but no distal obstruction was re-studied 1 month later. At that time the transit time of barium sulfate in that cat, both after administration of probanthine simulating adynamic ileus and without any drug, approximated the transit time during the preoperative control period. In this same cat at necropsy, a small amount of sterile serous ascites was seen, with adhesions at the healed site of perforation.

The greater the volume of administered contrast medium, the faster was the transit time. In all instances in which hypaque was administered, sufficient absorption from the gastrointestinal tract resulted in faint opacification of the urinary bladder of the cat.

DISCUSSION

Transit times vary notoriously, depending on the administered contrast medium and its volume, the age of the patients,[3] the clinical condition,[1] and the administered drugs, among other factors.

The transit time of each agent varied considerably among the group of cats, but, in individual cats, the transit time of a given contrast medium was fairly reproducible. Our reported results reflect at least a twofold change in compared transit times of individual cats.

Barium sulfate is an inert agent when confined to the lumen of the gastrointestinal tract. By contrast, hypaque, because of hyperosmolarity, readily draws body fluids to the gastrointestinal lumen and distends the bowel. The resultant increased volume of the gastrointestinal contents accounts for the rapid transit of the water soluble

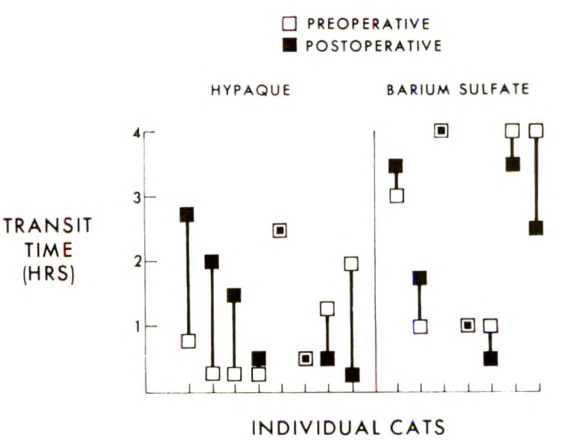


FIG. 5. Twenty-four hours after laparotomy with small bowel manipulation.

agent compared with that of barium. In this manner the water soluble contrast medium acts as a saline type of cathartic.[5] At the same time, hypaque is absorbed from the gastrointestinal tract of the cat sufficiently for faint opacification of the urinary bladder. Such urinary tract opacification is seen commonly in gastrointestinal studies of children and some adults after administration of hypaque.

Probanthine (probantheline bromide) in small doses acts as an anticholinergic.[2] In large doses it is a ganglion-blocking agent that results in hypotonicity primarily of the small bowel, and simulates adynamic ileus of acute abdominal conditions. This hypotonic action results in prolonged transit with each contrast medium. The hypotonic bowel responds only weakly to the effect of increased fluid within the gastrointestinal lumen and distention caused by hypaque.

Twenty-four hours after abdominal exploration, we observed no significant difference in transit time with barium sulfate. This observation supports the contention of Cannon and Murphy[1] that the transit time was normal in cats subjected to laparotomy. It does not refute the observations of Olivecrona,[8] however, that peristalsis of the intestine ceases for several hours after prolonged manipulation of the small bowel. We observed a delay in transit time in 4 of 8 cats in which hypaque was the contrast medium used.

In a smaller series, barium defined the site of bowel obstruction and in 1 instance it defined the perforation, whereas hypaque did not. When a perforation was present, barium produced severe peritonitis, whereas hypaque produced only small amounts of serous exudate.

SUMMARY

The transit time of barium sulfate and hypaque through the small bowel was compared in: (a) normal cats; (b) the same cats medicated with probanthine (a drug which produces a simulated adynamic ileus); (c) the same cats 24 hours after abdominal exploration; (d) the same cats with induced mechanical obstruction or perforation of the small bowel; and (e) a small group of cats in which both mechanical small bowel obstruction and perforation were surgically effected.

The small bowel transit time for hypaque was shorter than for barium in most instances, with or without adynamic ileus produced by probanthine. The transit time of barium sulfate 24 hours after abdominal exploration was unchanged compared with that in the same intact animal. Twenty-four hours after abdominal explorations, however, transit times with hypaque in half of the examined cats were longer than those of the control study in the same animal.

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SMALL INTESTINAL INTUSSUSCEPTION: AN EASILY MISUNDERSTOOD SIGN*

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THE roentgenologic diagnosis of intussusception of the small intestine of adults has been made fairly frequently for several decades. Descriptions of the characteristic roentgenogram always refer to the "coil spring" appearance of the lumen and the very narrow central channel (Fig. 1). However, if the intussusception already is established at the time of arrival of the contrast medium from above and if the segment is of sufficient length, only the narrow central channel will be visualized. This is most probable, of course, when the invagination is relatively long and fixed by swelling secondary to vascular congestion. On the other hand, the "coil spring" appearance is most likely when the invagination is relatively short and undergoing frequent reduction and recurrence, allowing contrast medium to coat all reflections of the bowel mucosa.

In the last few years 3 cases of established intussusception, in which only the narrow central channel was visible, have been encountered during small intestinal roentgenography. The first case caused consider-

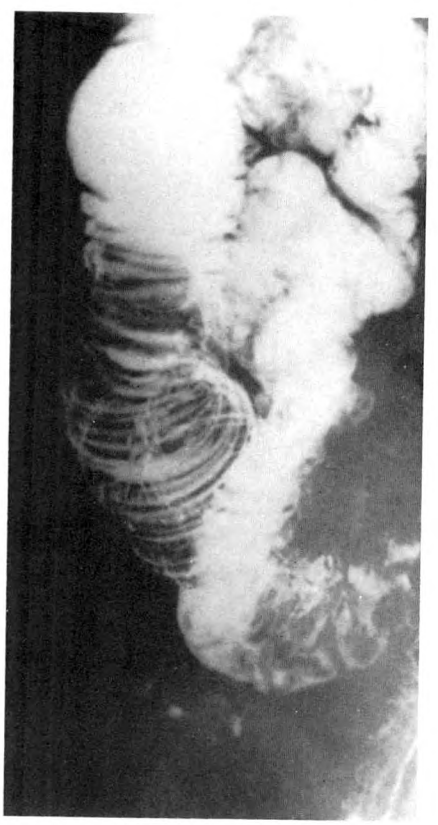


FIG. 1. Most common appearance of small intestine associated with intussusception—"coil spring" mucosal reflection and narrow central channel. In this case intussusception was due to polypoid metastatic melanoma.



FIG. 2. Case 1. Much less common but equally diagnostic appearance of intussusception. Narrowed segment extends from central axis of dilated proximal lumen which tapers sharply and symmetrically at point of narrowing. Narrowed channel (arrows) shows fine linear mucosal pattern.

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able discussion about the nature of the abnormality and its mechanism of production; hence this report. Two of these cases are briefly presented.

REPORT OF CASES

Case 1. A 20 year old woman had a history of nausea, vomiting, pain, and loss of weight to 65 pounds during a period of 3 years. A recent abdominal operation revealed a band which appeared to be obstructing the bowel at the ligament of Treitz. This was cut, but the symptoms persisted after operation. She was referred to the Mayo Clinic and roentgenologic examination of the small intestine revealed evidence of a high-grade obstruction of the jejunum by a long narrow segment (Fig. 2). This was thought at first to represent involvement by an extrinsic process, but in reality it illustrated a characteristic appearance of an established fixed intussusception, the narrow channel showing a fine linear mucosal pattern. Exactly the same appearance was present on a second examination. The patient was explored and the invaginated segment resected, since it could not be reduced. There was no mass leading the intussusceptum.

Case 11. A 46 year old man had experienced intermittent cramping abdominal pain for 2 months. Roentgenologic examination of the small intestine revealed a long narrow segment of proximal ileum, causing a high-grade obstruction. The narrow channel showed a fine linear mucosal pattern and a tapered junction with the proximal dilated lumen (Fig. 3). This appearance persisted for about 2 hours at which time reduction took place, allowing the identification of a 2 cm. broad-based polypoid tumor. This proved to be a hamartomatous polyp after resection.

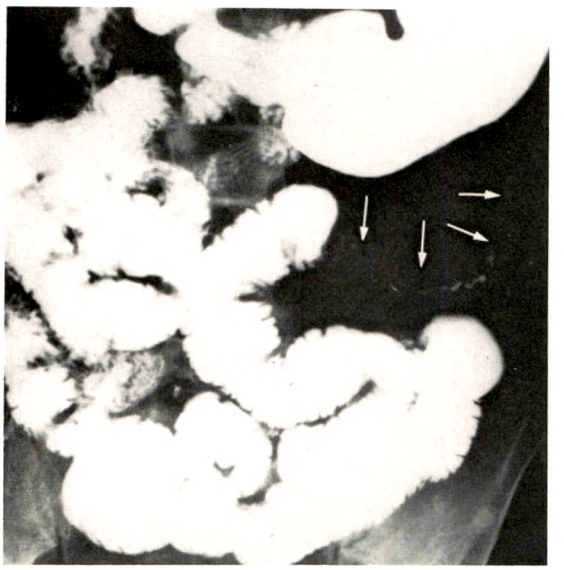


Fig. 3. Case 11. Another example of the appearance of established relatively fixed intussusception (arrows). This ultimately reduced, allowing identification of a 2 cm. polyp.

SUMMARY

The roentgenographic diagnosis of small intestinal intussusception is usually associated with a "coil spring" appearance plus a narrow central channel. However, if the invagination already is established and fixed on arrival of the contrast medium, only the central channel is visible, giving an almost equally characteristic appearance. This appearance, if persistent, may introduce a note of urgency since vascular insufficiency may be impending.

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POLYPOID LYMPHOID HYPERPLASIA OF THE TERMINAL ILEUM IN PATIENTS WITH FAMILIAL POLYPOSIS COLI AND WITH GARDNER'S SYNDROME*

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THE association of polypoid lymphoid hyperplasia of the terminal ileum with colonic adenomatous polyposis has received scant attention in the medical literature. Knowledge of this association is of some importance for it would save the patient from needless resection of the terminal ileum at the time of prophylactic colectomy. On macroscopic inspection, the lymphoid polyps are difficult to distinguish from the adenomatous polyps.

MATERIAL

(Succinct Case Reports)

Three patients, from different families, with polyposis of the colon are the subject of this report. Two patients (B.M. and C.M.H.) have Gardner's syndrome and the other (P.W.H.) has familial polyposis coli. All 3 patients have classic family histories and physical findings. Each underwent prophylactic colectomy and ileoproctostomy, with resection of short segments of terminal ileum. Two of these patients underwent the prophylactic surgery at the age of 25 years. The other patient (B.M.) underwent colectomy at the age of 17 years. A detailed history on patient B.M. was published by Duncan *et al.*[3] The preoperative barium enema examination on these patients showed the expected multiple polypoid lesions in the colon. In addition, similar, but generally smaller, defects were present in the terminal ileum (Fig. 1; and 2). In 1 patient (C.M.H), the polypoid lesions were still visible in the ileum on a postoperative barium enema roentgenogram (Fig. 3). During surgery, in each instance, multiple polypoid lesions were found in the terminal ileum.

On macroscopic inspection, the lesions in the colon and those in the terminal ileum appear very similar (Fig. 4). The lymphoid polyps are of the same size as

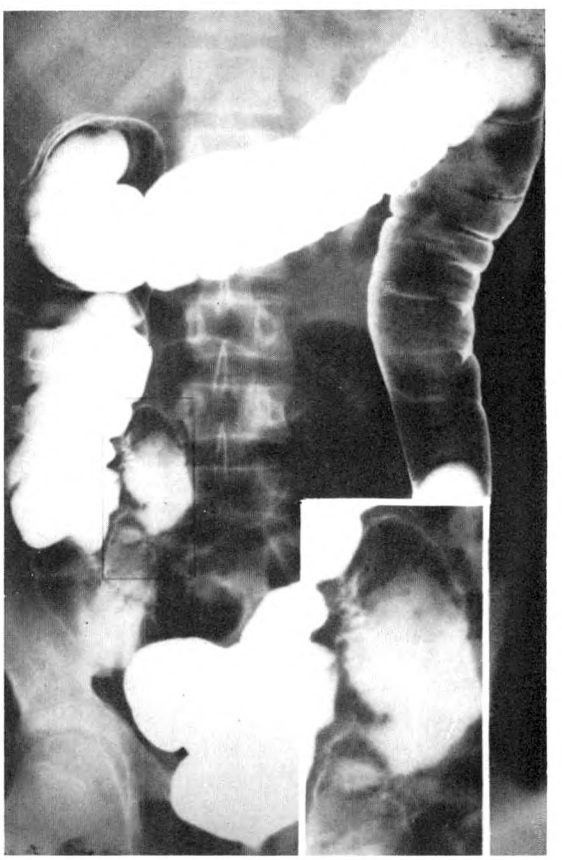


FIG. 1. Preoperative double contrast enema roentgenogram on Patient B.M. (Gardner's syndrome). The adenomatous polyps are best seen in the descending colon. Insert is magnification of terminal ileum. Multiple round filling defects are indistinguishable from the defects in the descending colon.

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the smaller adenomatous polyps of the colon. Histologic examination of the resected specimens shows that the polyps in the terminal ileum result from lymphoid hyperplasia, whereas the polyps in the colon are adenomatous in nature.

REVIEW OF THE LITERATURE

Borde and Gubler[1] were first to report the presence of polypoid hyperplasia of Peyers patches in the terminal ileum in a 14 year old boy who underwent a prophylactic colectomy and ileoproctostomy for familial colonic polyposis. Subsequently, Thomford and Greenberger[8] described lymphoid hyperplasia of the distal 5 feet of ileum in a 21 year old boy with Gardner's syndrome. Watne *et al.*[9] have reported that lymphoid hyperplasia in the terminal

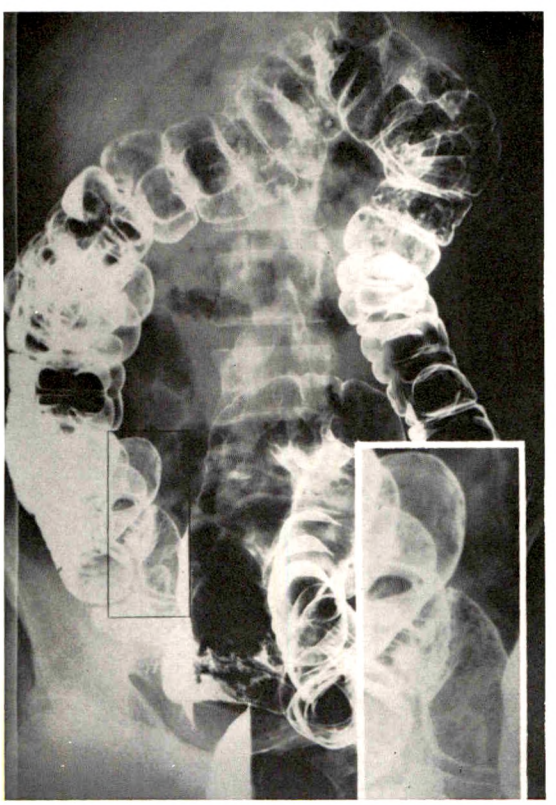


Fig. 2. Preoperative double contrast enema roentgenogram on Patient P.W.H. (familial polyposis coli). The small lucent filling defects in the terminal ileum are indistinguishable from the smaller of the colonic polyps. Insert is magnification of terminal ileum.



Fig. 3. Post ileoproctostomy double contrast enema roentgenogram on patient C.M.H. Small lucent defects are visible throughout the visualized small bowel. The defects appear mostly as localized widening of the small bowel mucosal folds.



Fig. 4. Photograph of resected specimen on Patient B.M. Small polyps are visible in the terminal ileum. They appear as localized widening of the mucosal folds. (From Duncan, B. R., Dohner, V. A. and Priest, J. H., *J. Pediat.*, with permission of the authors and editor.[3])

ileum was a frequent finding in their patients with Gardner's syndrome. Dorazio and Whelan[2] recently reported lymphoid hyperplasia in 2 patients with familial polyposis coli.

DISCUSSION

Isolated adenomatous polyps of the small bowel in patients with colonic polyposis have, on occasion, been observed.[7] The father of our Patient B.M. died at the age of 44 of malignant degeneration of a duodenal polyp, while none of his colonic polyps showed any evidence of malignancy. Patient B.M. himself had a duodenal polyp removed at the time of colectomy. We have found only one report about a patient with Gardner's syndrome where it is said that the polyposis (adenomatous?) extended into the terminal ileum.

The discovery of multiple polypoid lesions in the terminal ileum of patients with familial polyposis or Gardner's syndrome, either by barium enema examination or at the time of prophylactic colectomy, should not be assumed to represent extension of the adenomatous polyposis into the small bowel, and should of itself not constitute an inducement to extensive ileal resection. The true nature of these lesions should be verified by frozen section of a biopsy specimen.

Because lymphoid hyperplasia of the small bowel is at times associated with dysgammaglobulinemia,[4,5] a differential gamma globulin determination was obtained postoperatively on Patient B.M. The results of that test were normal.

SUMMARY

Multiple polypoid lesions of the terminal ileum observed by barium study and at surgery in patients with colonic polyposis will probably be lymphoid hyperplasia and not an extension of the adenomatous polyposis of the colon, even though their macroscopic appearance is similar. The nature of the ileal polyps should be verified by histology.

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RARE ILEOCECAL LESIONS*

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ARRIVING at a preoperative roentgen diagnosis of right lower quadrant problems is a challenge to any radiologist. When evaluating roentgenograms of the ileocecal area, we consider such usually known lesions as: carcinoma, amebiasis or tuberculosis of the cecum; abscess, mucocele or carcinoid of the appendix; and granulomatous ileitis or lymphosarcoma of the terminal ileum.[1] Felson and Wiot[6] recently reported roentgen findings of the following right lower quadrant unusual lesions: myxoglobulosis of the appendix, ileal prolapse, diverticulitis of the distal ileum, intussuscepting lymphoma and cecal endometriosis.

The purpose of this paper is to enlarge the list of differential roentgen possibilities in the ileocecal area.

CARCINOID OF THE ILEOCECAL VALVE

Arising from the chromaffin cells of the gastrointestinal mucosa, the carcinoid tumor is circumscribed but not encapsulated. It is invasive and metastasizes. The clinically well known carcinoid syndrome is produced by the serotonin released from the tumor.[2] Carcinoid has been found in the appendix, stomach, duodenum, small bowel, colon, rectum, root of the mesentery and in the ileocecal valve.[10]

REPORT OF A CASE

A 65 year old widow visited her physician after suffering lower abdominal cramps and diarrhea for a few weeks. A barium enema examination demonstrated a small polyp at the lateral wall of the cecum and an enlarged ileocecal valve (Fig. 1, *A* and *B*). This was pursued 5 months later when the patient was hospitalized because of 15 pound weight loss, weakness, fatigability, continuous cramps, bloating and loose bowel movements. There were no palpable masses in the abdomen. The patient was afebrile. Laboratory work showed: hemoglobin 13.0 gm. per 100 ml., hematocrit 43 per cent, and white blood cell count 6,700. The barium meal revealed dilatation only of the last few feet of ileum. The valvulae conniventes were stretched. There was barium in the cecum. Compression spot roentgenograms of the ileocecum demonstrated a "coil spring" appearance of a segment of bowel near the cecum consistent with an ileoileal intussusception (Fig. 1*C*). The previous barium enema roentgenograms were re-evaluated. The size of the ileocecal valve and the total lack of reflux of barium in the terminal ileum became significant. Combined with the low grade small bowel obstruction, the findings pointed toward a neoplastic process of the ileocecal valve.

The surgeon found a mass involving the ileocecal and retrocecal area. There were also multiple 1 cm. white seedings throughout the small bowel mesentery. A right hemicolectomy was performed.

The specimen consisted of the distal 12 cm. of ileum, the cecum and the proximal 15 cm. of the ascending colon. The diameter of the distal ileum was 7 cm., the wall was firm and the mucosa had a hemorrhagic appearance. A fungating 2×3×3.5 cm. mass occupied the ileocecal junction. The center of the mass had an ulceration. The tumor extended through the bowel wall and serosa. The cut surface was yellow-gray with a white appearing center.

The pathologic diagnosis was carcinoid of the ileocecal valve with metastases to regional lymph nodes and extension to the pericolonic fat and muscle layers of the appendix.

ROENTGEN MANIFESTATIONS

According to Hinkel,[9] 8 per cent of the ileocecal valves are posteriorly situated. These valves' protruding lips displace the barium and produce a spongy, ovoid, snowshoe or spindle-shaped filling defect. In the center there usually is a slit or modified star representing the lumen of the ileum. When

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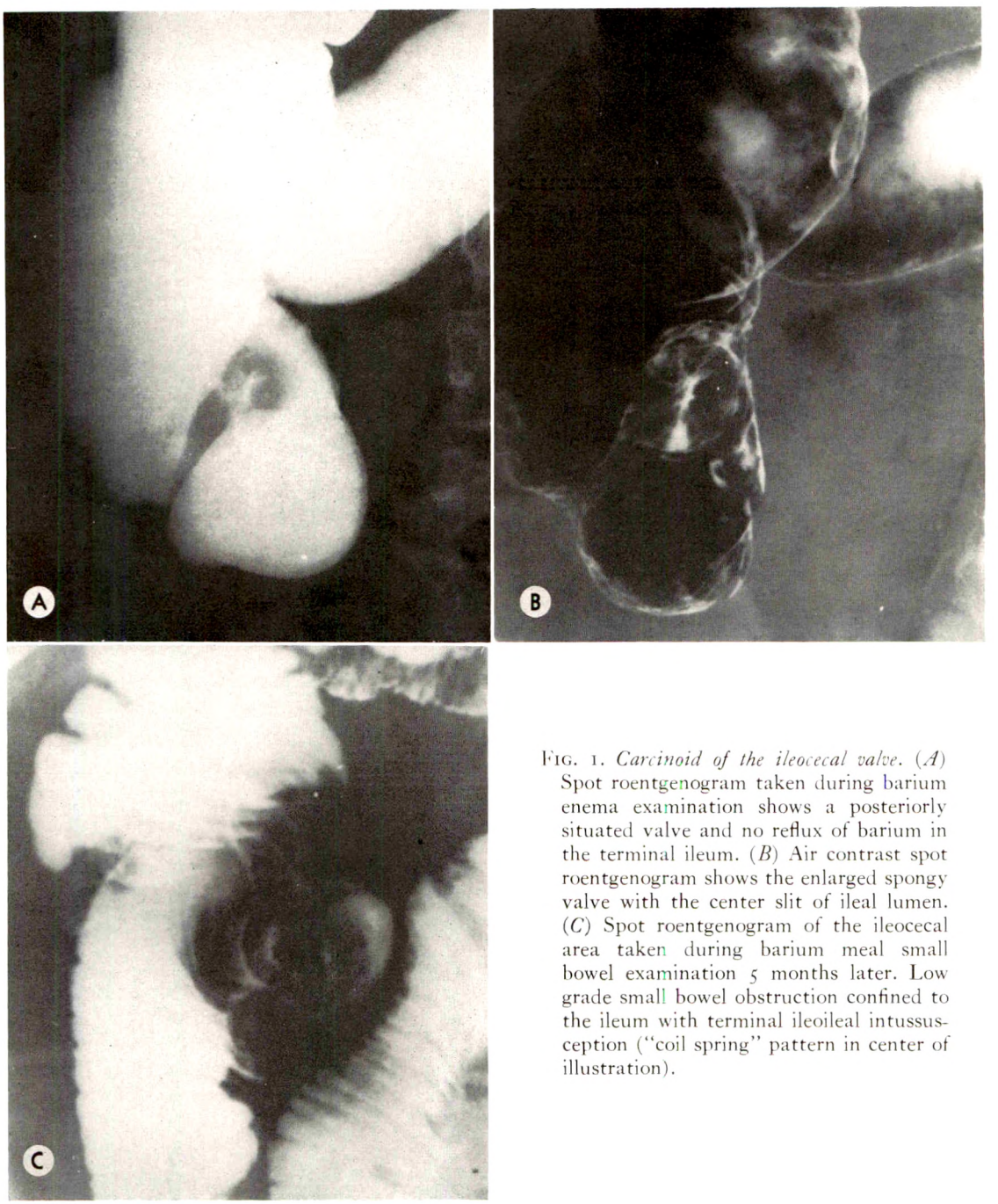


Fig. 1. *Carcinoid of the ileocecal valve.* (*A*) Spot roentgenogram taken during barium enema examination shows a posteriorly situated valve and no reflux of barium in the terminal ileum. (*B*) Air contrast spot roentgenogram shows the enlarged spongy valve with the center slit of ileal lumen. (*C*) Spot roentgenogram of the ileocecal area taken during barium meal small bowel examination 5 months later. Low grade small bowel obstruction confined to the ileum with terminal ileoileal intussusception ("coil spring" pattern in center of illustration).

a posteriorly situated ileocecal valve appears to be unusually large on compression spot roentgenograms (Fig. 1, *A* and *B*), the following possibilities are considered: edema of the lips, hypertrophy, fatty infiltration or tumor of the valve lips.

It is difficult to establish tumor infiltration of the ileocecal valve, unless the valve lips are obviously asymmetric or an ulceration is demonstrated. Absence of reflux of the barium in the terminal ileum during barium enema examination by itself cannot

be diagnostic of tumor because hypertrophy, edema or fatty infiltration will also prevent reflux.

The most important additional roentgen study is the barium meal small bowel examination. In not too advanced cases there will be roentgen signs of low grade bowel obstruction limited to the ileum. In advanced cases there is complete obstruction. Ileoileal intussusception (Fig. 1*C*), due to violent peristaltic activity against the tight ileocecal valve lips, is an indirect roentgen sign of the presence of tumor.

INTUSSUSCEPTING MECKEL'S DIVERTICULUM

Only a very small number of Meckel's diverticula give rise to symptoms and only a very few are diagnosed preoperatively. The symptoms are usually the result of complications, such as: peptic ulceration, hemorrhage, perforation, acute inflammation, volvulus, neoplasm or intussusception.[3] On the other hand, only 2 per cent of all small bowel intussusceptions are due to an inverted Meckel's diverticulum.[13]

REPORT OF A CASE

A 7 year old girl was admitted to the hospital with acute abdominal pain, nausea and vomiting. She developed the first abdominal cramps 24 hours after a picnic. Her abdomen was distended. The child was acutely ill, with a temperature of 99.2°F. and pulse of 110. The bowel sounds were hyperactive. The right lower abdomen was rigid. There was blood on the rectal examining glove.

Laboratory data were normal except for a white blood cell count of 20,900.

Multiple plain roentgenograms of the abdomen showed dilated small bowel loops arranged in a step-ladder pattern with air fluid levels. There was no gas in the colon. An emergency barium enema examination was then performed. A "coil spring" filling defect was demonstrated near the ileocecal valve with compression roentgenograms (Fig. 2, *A* and *B*). The postevacuation roentgenogram revealed extensive filling defects with a "coil spring" appearance occupying the entire ascending colon (Fig. 2*C*). At the end of the intussusceptedileum, toward the hepatic flexure, was a 4 cm. tumor-like round filling defect.

At surgery, a segment of 4 feet of ileum was reduced from the ascending colon and cecum. A hard mass was then felt in the lumen of the reduced bowel. A segmental resection was performed.

The open ileal segment revealed an inverted Meckel's diverticulum occupying the entire width of the bowel lumen (Fig. 2*D*).

ROENTGEN MANIFESTATIONS

An inverted Meckel's diverticulum protrudes into the small bowel lumen, resembles a tumor and obstructs like a tumor (Fig. 2*D*). The peristaltic activity of the ileum tries to advance the mass. As a result the ileum telescopes into itself. The resulting intussusception is commonly an ileoileal type.[7] A barium meal small bowel examination will show a filling defect in the ileum with moderately dilated small bowel loops proximal to the defect.[13]

The rare ileocolic intussusception of an inverted Meckel's diverticulum has different roentgen signs. When the intussusception enters the cecum and ascending colon, the distensibility of the colon permits the intussusception of numerous ileal loops. This produces a very extensive "coil spring" pattern. The inverted Meckel's diverticulum can be identified on the roentgenograms as a round filling defect sitting at the head of the intussuscepted ileal loops (Fig. 2*C*).

NEUROFIBROMA OF THE TERMINAL ILEUM

Gastrointestinal lesions in patients with von Recklinghausen's disease have been reported in the pharynx, tongue, gallbladder, small bowel, colon and rectum.[4] The lesions are silent clinically, until abdominal pain, intestinal obstruction or hemorrhage occurs.

REPORT OF A CASE

A 43 year old man with von Recklinghausen's disease since childhood was admitted to the hospital with right lower abdominal pain and palpable mass. Ten years prior to this admission, he underwent exploration for a right lower quadrant mass. Two small tumors were

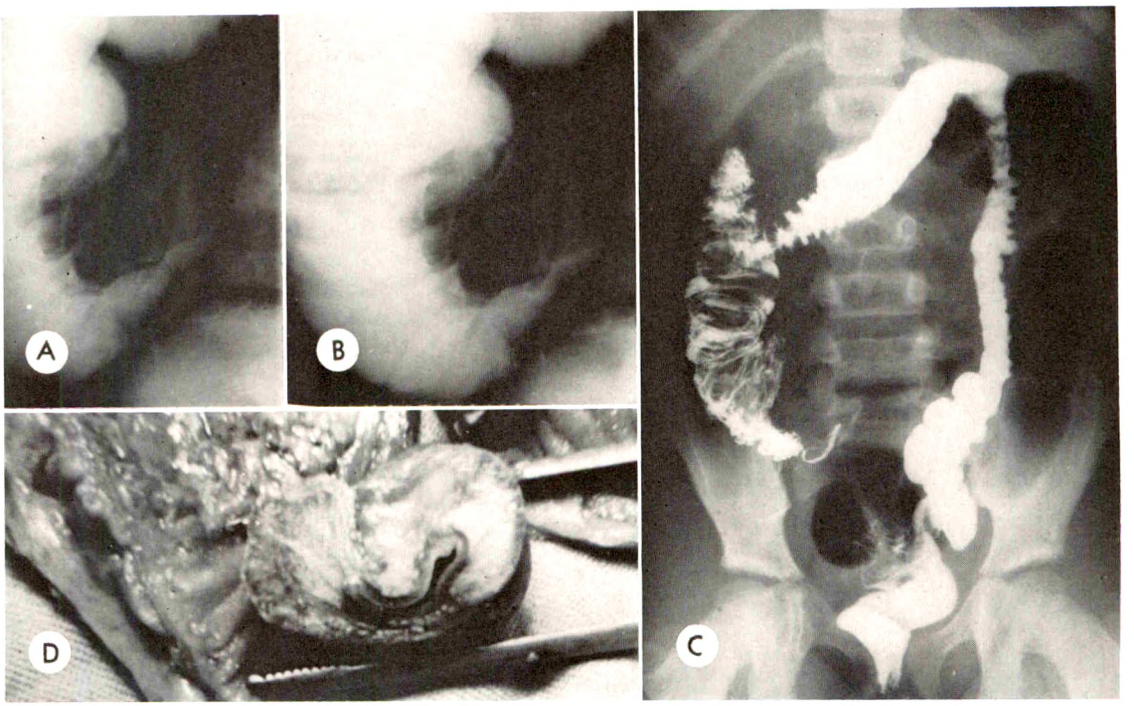


Fig. 2. *Intussuscepting Meckel's diverticulum.* (*A* and *B*) Spot roentgenograms of the cecum taken during barium enema examination. There is a "coil spring" filling defect in the ileocecal valve area. (*C*) The post-evacuation roentgenogram shows multiple ileal loops intussuscepted in the ascending colon. There is a 4 cm. round filling defect at the head of the intussusception. (*D*) Surgical specimen. The lumen of the ileum reveals a tumor-like inverted Meckel's diverticulum.

removed from the ileum. One was reported to represent a fibroma, the other a leiomyoma. Two months later he was readmitted with small bowel obstruction and an incisional hernia. Necrotic bowel loops were resected and an entero-enterostomy was performed. Four years later the patient developed a partial small bowel obstruction and hepatitis. Since then, and for the past 6 years, he has had chronic recurrent abdominal pain. One month prior to this latest admission, he developed fever, chills, nausea and vomiting. He improved on antibiotics but a right lower abdominal mass persisted. He lost 10 pounds in 2 months.

The physical examination showed multiple neurofibromatous nodules over the entire body, particularly the thorax. There was a palpable lower abdominal mass. Laboratory data were normal except for a white blood cell count of 11,750. The barium enema examination showed an incomplete filling of the cecum which was displaced laterally. There was no reflux in the terminal ileum. The barium meal small bowel examination (Fig. 3) revealed a space in the right lower quadrant occupied by a flattened ileal loop and a fistulous tract. Three nodular pressure defects were identified in the same area. The defects appeared to be extramucosal and caused narrowing of the ileal lumen.

At surgery, the loops of ileum were tightly adherent to each other and to a large mass of firm, pale-yellow tissue embedded in the mesentery. A right hemicolectomy was performed.

The surgical specimen showed multiple tumor nodules adherent to the serosal surface of the ileum. They protruded into the intestinal lumen and were seen bulging under the mucosa. The largest tumor measured 4 cm. in diameter. The cut surface was deep pink, hemorrhagic and very firm. The pathologic diagnosis was multiple neurofibromata of the terminal ileum.

Roentgen Manifestations

The intestinal neurofibroma originates from the chromaffin cells of the enteric ganglionated plexuses.[11] The exact location of these plexuses is important to explain the roentgen findings. The plexus of Auerbach is situated between the longitudinal

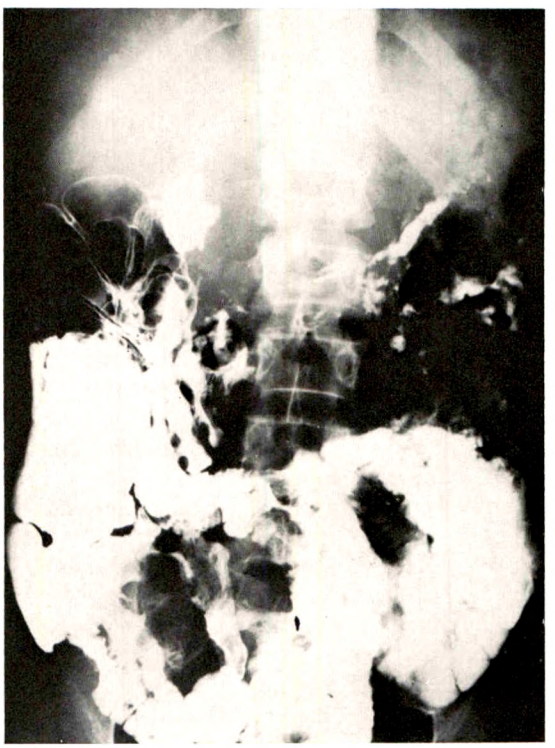


Fig. 3. *Neurofibroma of the terminal ileum.* Two hour roentgenogram after a barium meal. There is a mass in the ileocecal area. Three extramucosal filling defects are seen on a loop of terminal ileum. Note the cutaneous neurofibromata seen in the right upper section of the roentgenogram.

and circular layers of the muscularis and the plexus of Meissner lies in the submucosa. The nerves reach the intestinal wall through the mesentery. Consequently, small tumors originating from the chromaffin cells will be situated in the muscularis, submucosa or on the serosal surface.

Roentgenographically, the neurofibromata are seen as typical extramucosal, intramural defects, best demonstrated with compression spot roentgenograms. The neurofibromata situated on the serosal surface may coalesce and are seen as masses spreading or flattening the loops of small bowel (Fig. 3). These tumors may cause small bowel obstruction.

ADENOCARCINOMA OF THE APPENDIX

A colonic type of carcinoma is the rarest type of malignancy found in the appendix. The diagnosis is usually not made preoperatively.[8] The rapid involvement of the cecum makes even the exact operative diagnosis difficult.

REPORT OF A CASE

A 26 year old woman was admitted to the hospital with a 6 week history of back pain, accompanied in the last 2 weeks with sharp epigastric pain. The last few days she had nausea and vomiting. A hard movable 5×5 cm. mass was found in the right lower abdomen. The liver was firm and enlarged. The patient became febrile in the hospital. She developed ecchymoses of the legs and had dark brown guaiac positive stools. The laboratory data were: white blood cell count 13,700, hemoglobin 14.2 gm. per 100 ml., hematocrit 44 per cent, blood urea nitrogen 19 mg. per cent, alkaline phosphatase 33.1 SU, bilirubin 3.0 mg. per cent, transaminase SGOT 246 units, transaminase SGPT 142 units. The blood coagulation tests were markedly abnormal. The technetium sulfide liver scan showed a mottled uptake pattern. The barium meal revealed a very slow transit time. The barium advanced to the distal jejunum at 6 hours and into the colon at 24 hours. A large liver was identified and there was extrinsic pressure on the third portion of the duodenum. The barium enema (Fig. 4) showed extrinsic pressure on the tip and medial aspect of the cecum. The refluxed barium showed a long narrow terminal ileal segment.

A hard, knobby mass was found in the right lower abdomen at surgery. The mass arose from the ileocecal area and extended across the cecal and small bowel mesentery. There were numerous hard lymph nodes around the superior mesenteric artery and across the aorta. An ileotransverse colostomy was performed. The patient's condition deteriorated rapidly and she died 7 days after surgery. The autopsy showed an adenocarcinoma of the appendix with metastases to the regional, mesenteric, peripancreatic, paraaortic, omental and mediastinal lymph nodes, and to the liver, cecum, jejunum and vertebral column.

ROENTGEN MANIFESTATIONS

An adenocarcinoma of the appendix can be a surprise finding in a resected appendix. In these instances, the neoplasm is confined only to the mucosa and the prognosis is good. Once beyond the mucosal stage, the adenocarcinoma of the appendix is very

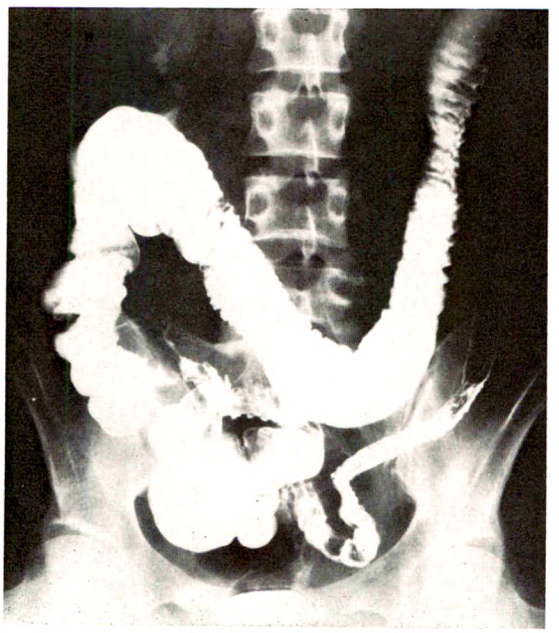


Fig. 4. *Adenocarcinoma of the appendix.* Postevacuation roentgenogram from a barium enema examination. There is extrinsic pressure on the cecum and narrowing of the terminal ileum projected across the right sacroiliac area. Other roentgen findings not shown here: hepatomegaly, extrinsic pressure on the third portion of the duodenum and a slow transit time of barium through the small bowel.

invasive locally and spreads rapidly by blood and lymph channels.[8]

The roentgen manifestations in advanced cases consist of mass lesions displacing the barium filled cecum and terminal ileum. The presence of widespread metastases throughout the abdomen can be deduced from the following roentgen findings: enlarged liver, extrinsic pressure on the duodenum, small bowel or colon and a slow transit of the barium through the small bowel. The latter can be explained by impairment of blood supply to the small bowel as a result of the compression of the superior mesenteric artery by enlarged lymph nodes.

METASTATIC ADENOCARCINOMA OF THE TERMINAL ILEUM

Metastatic carcinoma of the small bowel is an infrequent occurrence, particularly when it is not part of a generalized abdominal carcinomatosis. Reported primary lesions are: carcinoma of the cervix, colon, lung, stomach, kidney, breast, uterus and also melanoma.[5] Clinically, small bowel metastases manifest themselves by producing hemorrhage, perforation, partial or complete bowel obstruction.

REPORT OF A CASE

A 53 year old woman was admitted to the hospital with constipation alternating with nausea and diarrhea for 2 years, becoming progressively worse unless she limited herself to a diet of only liquids and ground food. Five years prior to her admission she had radium treatment followed by a hysterectomy for an endometrial carcinoma. The physical examination revealed a healthy appearing patient with a soft abdomen and no palpable masses. All the laboratory tests were within normal limits. A barium enema examination showed a rectosigmoid stricture 25 cm. in length. However, there was no obstruction to the retrograde flow of barium or to the evacuation. The stricture was attributed to the previous radiation treatment. The barium meal small bowel examination revealed dilatation of the last few feet of ileum (Fig. 5*A*). The loops of ileum had prominent valvulae conniventes. The barium entered the cecum without delay. The findings were interpreted as representing a partial ileal obstruction. The fluoroscopic search for the cause of the obstruction demonstrated a 3 cm. narrow segment in the terminal ileum only a few centimeters proximal to the ileocecal valve (Fig. 5*B*). The ileum distal to the narrow segment was of normal caliber. The ileal defect had an extramucosal appearance.

The abdomen was remarkably clean and easy to examine during laparotomy. There was no evidence of recurrent tumor in the pelvis. The stenotic rectosigmoid junction was pliable and it was not the site of tumor or of an inflammatory process. A mass was found in the terminal ileum. There was knuckling of the bowel at this point causing incomplete small bowel obstruction. Three enlarged lymph nodes were found at the base of the ileal mesentery and another 1 cm. tumor was found in the jejunum. A right hemicolectomy and a segmental resection of the jejunal node were performed.

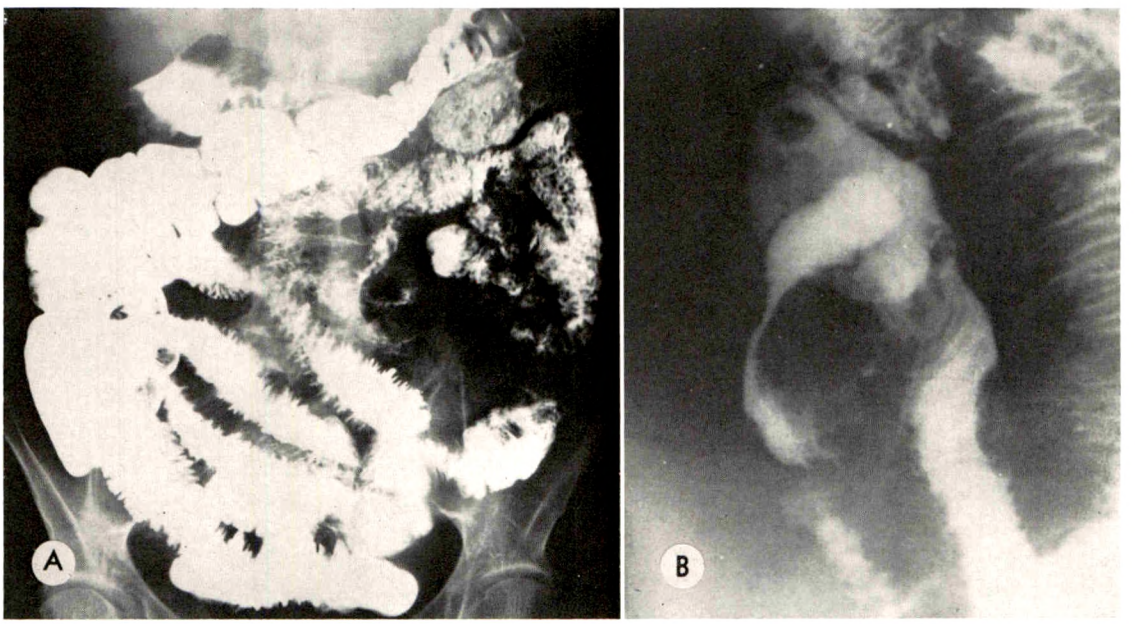


Fig. 5. *Metastatic adenocarcinoma of the terminal ileum.* (*A*) Two hour roentgenogram after a barium meal examination. There is dilatation of the ileum but no complete obstruction of the passage of barium into the cecum. (*B*) Compression roentgenogram taken during fluoroscopic search for the cause of the partial obstruction. A 3 cm. extramucosal lesion of the terminal ileum is demonstrated.

The hemicolectomy specimen showed a 3 cm. fungating and ulcerated mass of the ileum about 4 cm. from the ileocecal valve. The entire thickness of the ileum was involved. Two 0.5 cm. polypoid structures were found in the mucosa of the cecum. The lesions of the ileum, cecum, jejunum and 2 of 3 mesenteric lymph nodes showed metastatic adenocarcinoma from the endometrium.

ROENTGEN MANIFESTATIONS

Marshak *et al.*[12] divide small bowel metastases into 3 groups: (a) lesions involving the small bowel wall only; (b) lesions involving the mesentery only; and (c) lesions involving the bowel wall and the mesentery. The bowel metastatic lesions can be isolated or multiple. They have a very specific roentgen pattern. The defect on the barium filled small bowel loop has a definite extramucosal character (Fig. 5*B*). When the lesion enlarges significantly, it protrudes into the lumen and causes small bowel obstruction. The obstruction is incomplete in early cases and complete in advanced cases.

The submucosal location of metastasis to the small bowel is determined by the location of vessels and lymphatics in the bowel wall. The vascular plexus of Heller is located in the submucosa and the larger lymph vessels form a meshed plexus also placed in the submucosa.

TUMOR-LIKE INTESTINAL TUBERCULOSIS

Primary intestinal tuberculosis, seen in the absence of pulmonary lesions, is very rare, particularly in the United States. Eighty-five per cent of the primary intestinal lesions are in the ileocecal area.[3] Other reported intestinal sites are: the stomach, the mesentery and the retroperitoneal lymph nodes.[14] The last two areas of involvement can produce lesions resembling a neoplastic process.

REPORT OF A CASE

A 37 year old woman was admitted to the hospital with mild epigastric pain, frequent nausea and anorexia. She had amenorrhea since her last delivery which was 2 years prior to the

admission. There was a 17 pound weight loss in the past 6 months. Periods of constipation alternated with periods of soft bowel movements. The physical examination revealed epigastric tenderness and a suprapubic mass. The pelvic examination revealed an ill-defined mass about the uterus. There was a low grade fever, from 99° to 101° F. Laboratory data: white blood cell count 11,500, hematocrit 36 per cent, sedimentation rate 21, albumin 4.2 mg. per cent, globulin 4.5 mg. per cent, with a reversal of the albumin/globulin ratio. The old tuberculin test was positive with a dilution of 1:10,000. The chest roentgenogram was normal.

The barium enema examination showed a normal colon, especially the cecum. The refluxed barium in the terminal ileum revealed the terminal ileum displaced by two masses located lateral to the cecum (Fig. 6*A*). The postevacuation roentgenogram (Fig. 6*B*) showed considerably more reflux of barium in ileal loops, including loops in the mid abdomen, displaced by masses of 4 to 5 cm. size. A barium meal small bowel examination duplicated the barium enema findings. Compression roentgenograms (Fig. 6, *C* and *D*) showed ileal loops almost encircling multiple masses. The ileal loops were fixed.

The entire abdomen appeared at laparotomy to be matted with lesions grossly resembling disseminated carcinoma. The omentum was involved with coalescent masses. The parietal peritoneum was studded with small friable 0.5 to 2 cm. lesions. There was about 300 cc. of straw-colored fluid in the abdomen. The frozen sections showed a granulomatous inflammatory pattern, but no evidence of a tumor. The permanent sections showed a granulomatous process characteristic of tuberculosis. No acid-fast organisms were identified.

Postoperatively, the patient developed tuberculous peritonitis and was put on antituber-

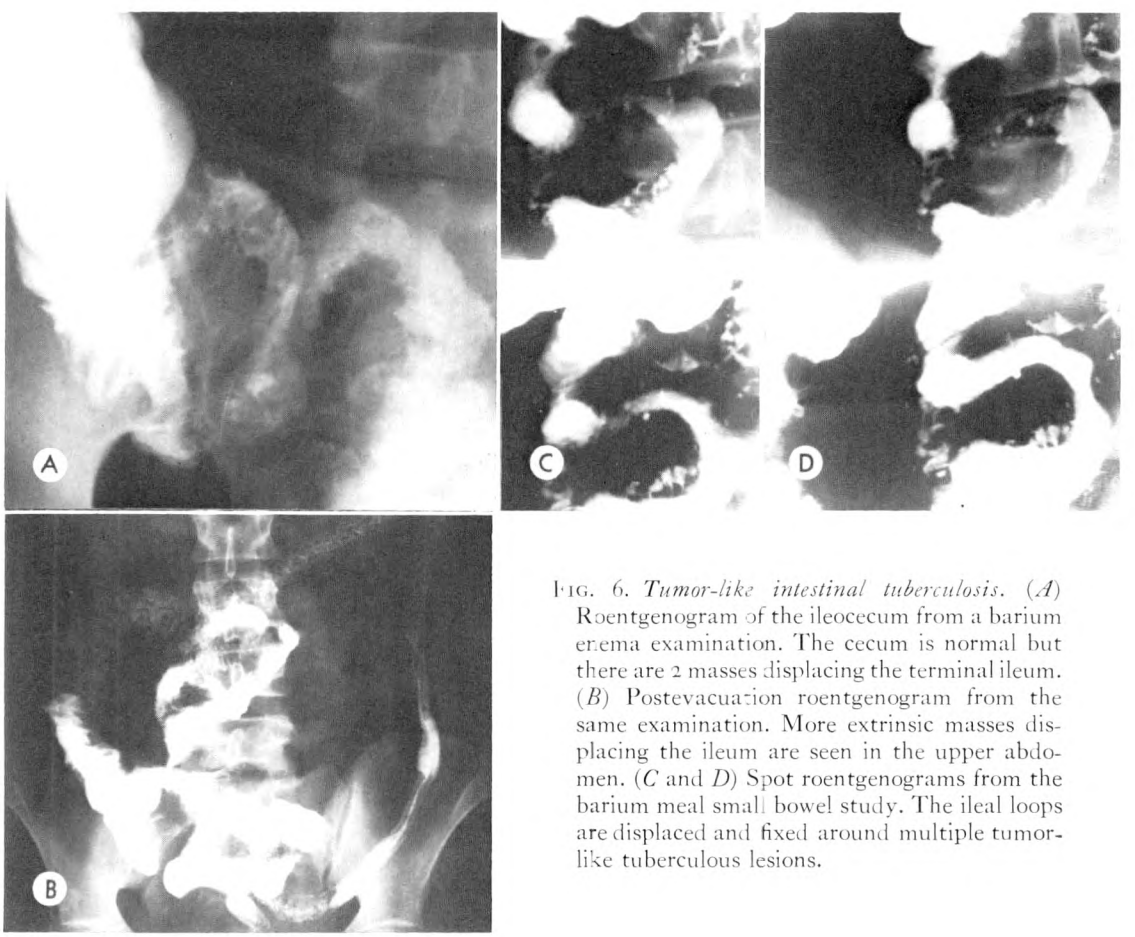


Fig. 6. *Tumor-like intestinal tuberculosis.* (*A*) Roentgenogram of the ileocecum from a barium enema examination. The cecum is normal but there are 2 masses displacing the terminal ileum. (*B*) Postevacuation roentgenogram from the same examination. More extrinsic masses displacing the ileum are seen in the upper abdomen. (*C* and *D*) Spot roentgenograms from the barium meal small bowel study. The ileal loops are displaced and fixed around multiple tumor-like tuberculous lesions.

culosis drugs. Six months later she developed pulmonary tuberculosis.

ROENTGEN MANIFESTATIONS

In the evolutive type of intestinal tuberculous infection, the acid-fast bacilli pass through the intestinal mucosa, invade the mucosa and produce edema, infiltration, ulcerations and ultimately fibrosis, stenosis and tissue hyperplasia.[3] The roentgen features of this form are well known: disturbed bowel motility, ulcerations, and in the late stages, the purse-shaped cecum, the straightening of the ileocecal junction and small bowel stenosis.

In the primary intestinal infection, very similar to the primary pulmonary tuberculous infection, the bacilli pass through the mucosa without greatly damaging it. The bacilli settle in the mesenteric lymph nodes. This results ultimately in the roentgenographically identifiable calcified mesenteric lymph nodes.

In very rare occasions, the mesenteric or the retroperitoneal lymph nodes[14] develop a full blown active tuberculous process, very similar to mediastinal lymph node tuberculosis. The important roentgen feature of this rare form of intestinal tuberculosis is multiple tumor-like masses in the abdomen displacing the barium filled small bowel loops (Fig. 6, *A–D*).

CONCLUSION

A variety of 6 rare ileocecal lesions is reported and their roentgen manifestations are briefly discussed.

The awareness of the existence of such lesions in the right lower abdomen could help establish more preoperative roentgen diagnoses.

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FLUID IN THE RIGHT FLANK AS A ROENTGENOGRAPHIC SIGN OF ACUTE APPENDICITIS*

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THE roentgen findings sometimes associated with acute appendicitis include: sentinel loop ileus in the right lower quadrant; air fluid levels in the cecum; obliteration of the right psoas shadow; abscess gas bubbles in the right lower quadrant; right lower quadrant soft tissue masses; coproliths in the right lower quadrant; gas in the appendix; and fluid in the peritoneal cavity indicating generalized peritonitis.[1,4]

Attention is called to the finding of a localized collection of fluid in the flank, which, in the author's experience, has been the most frequent and reliable roentgen finding in acute appendicitis. The finding is not a new one, but has not been emphasized in the literature. McCort[3] mentions fluid interposed between the colon and flank stripe in his discussion of perforation of the appendix, with gas outside the alimentary tract. Frimann-Dahl[2] has described fluid interposed between the colon contents and both flank stripes in his description of ascites, and in generalized peritonitis.

MATERIAL

In St. Joseph Infirmary, which is a general hospital, over the past 2 years more than 400 patients had appendicitis found at surgery. Of this number 28 patients had roentgenograms of the abdomen; these were reviewed for evidence of fluid in the right flank displacing the colon. The ages of the patients ranged from 6 to 79 years.

A survey was also made of 100 random, plain film abdominal roentgenograms, obtained in patients without symptoms of appendiceal disease, and a measurement made as to the distance of colonic contents from the flank stripe in the right lower quadrant.

RESULTS

Of the 28 patients having abdominal roentgenograms and later the finding of appendicitis at surgery, 15 cases showed the roentgen sign of fluid density interposed between the colonic contents and the right flank stripe. Six of these 15 patients also showed fluid levels in the cecum or ascending colon, 2 had coproliths in the right lower quadrant and 1 each had gas in the appendix, abscess gas and a right lower quadrant mass. At surgery 9 of these cases had gangrenous appendices with perforation, 2 had gangrenous appendices without perforation and 3 cases showed acute appendicitis without gangrenous change.

In 5 cases the findings were equivocal in that colonic contents could not be identified with certainty and a measurement could not be made. One of these patients showed an air fluid level in the cecum. The others had no definite roentgen findings of appendicitis. At surgery 2 of the cases had gangrenous appendices with perforation. The other showed acute appendicitis without gangrene or perforation.

In the 8 patients showing no displacement of colonic contents from the flank stripe, 1 had a fluid level in the cecum and 1 a coprolith in the right lower quadrant. The others had no definite roentgen findings of appendicitis. At surgery 3 of these patients had perforated appendices and 2 others had gangrenous appendices. One had acute appendicitis and the remaining 2

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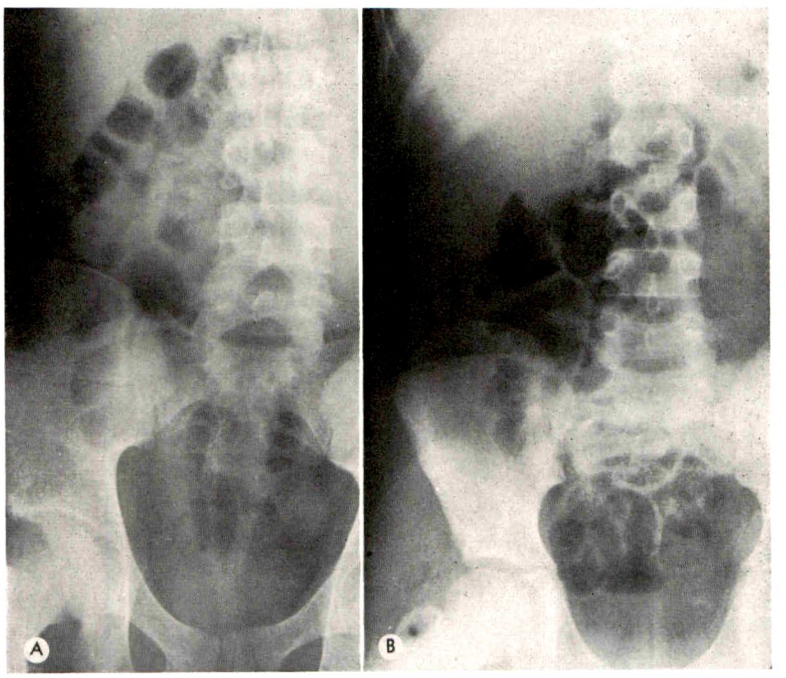


Fig. 1. (*A*) A 17 year old male with vague abdominal pains. The plain film roentgenogram of the abdomen shows displacement of the air filled ascending colon from the flank stripe on the right for a distance of 6 to 12 mm., depending on the point of measurement. Intravenous pyelograms and colon studies were negative. At surgery a gangrenous appendix with rupture was found.

(*B*) This patient is an 8 year old male. A 15 mm. displacement of the colon from the flank stripe is shown on the plain film roentgenogram of the abdomen. Acute appendicitis without perforation was found at surgery.

showed only subacute changes in the appendix with focal ulceration of the mucosa but no marked inflammatory reaction.

In the 100 random, plain film abdominal roentgenograms surveyed for a control, colonic contents could be identified with certainty in almost all cases and a measurement made as to the distance from the flank fat to colonic contents. This distance is usually 2 to 3 mm. and does not exceed 5 mm. in normal subjects.

DISCUSSION

A localized fluid collection in the right lower quadrant interposed between the colonic contents and the lucent flank stripe produces a smooth, fairly well defined margin against the extraperitoneal fat in the flank. Medially the fluid density merges with the bowel wall giving an irregular and often lobular appearance against the fecal or gas shadows in the colon (Fig. 1, *A* and *B*). The appearance is similar to that seen in both flanks in patients with ascites. The finding is best shown in roentgenograms made in the anteroposterior projection with the patient supine. A displacement of more than 5 mm., anywhere along the right flank stripe, is considered significant. On occasion, fluid in the colon may simulate the finding. A repeat study after a short interval will usually allow definite identification of the colonic contents in these cases.

Most patients who are operated for acute appendicitis do not have roentgen studies of the abdomen. The diagnosis is usually made on the basis of history, physical and laboratory findings. Abdominal roentgenograms may be obtained when the symp-

toms are mild or atypical or in cases where the symptoms are very severe and differentiation from other acute abdominal processes is difficult.

The present study is weighted by the fact that abdominal plain film roentgenograms were obtained more frequently in the more severe cases so that the per cent of patients having gangrenous or ruptured appendix was higher than is usually found in appendicitis. We are planning a future study to include abdominal plain film roentgenograms on all patients in whom appendicitis is suspected, to determine the true frequency of the finding in appendicitis.

SUMMARY

The roentgen finding of fluid interposed between colonic contents and the fat flank stripe in the right lower quadrant is emphasized as a frequent and reliable indication of appendicitis.

This finding was present in over one-half of the cases later coming to surgery with acute appendicitis.

The next most frequent finding was that of fluid levels in the cecum or ascending colon.

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ROENTGENOLOGIC FEATURES OF REGIONAL ENTERITIS OF THE UPPER GASTROINTESTINAL TRACT*

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THE inflammatory lesions of regional enteritis are not confined only to the terminal portion of the ileum but are also occasionally found within the esophagus, stomach, and duodenum.[1,3–6] Difficulty in diagnosis may be encountered because the radiologist generally expects this disease to be located in the more distal site. If the correct diagnosis is not established, the patient may be subjected to unnecessary exploratory surgery.

This paper reviews the roentgenologic features of upper gastrointestinal involvement with regional enteritis, as observed in a series of patients with such features seen at the Mayo Clinic.

MATERIAL AND METHOD

The histories of 383 patients with regional enteritis seen at the Mayo Clinic between 1964 and 1968 were reviewed. Of the 383 patients, 7 had regional enteritis in the duodenum. Four of the 7 also had stomach involvement. Since 1968, we have seen 4 more patients with regional enteritis and upper gastrointestinal involvement: 1 had involvement of the esophagus, 2 of the stomach, and 1 of the stomach and duodenum. In our series, 10 patients underwent surgery, and 1 patient had esophagoscopy. In 6 patients, upper gastrointestinal involvement was diagnosed after histologic examination of a surgical biopsy specimen; in the other 5, diagnosis was made after direct inspection of the mucosa of the affected organ, which revealed characteristic anatomic changes. Roentgenograms of the upper gastrointestinal tract were available in each case, and the roentgenologic abnormalities corresponded to the affected regions that were subsequently inspected or biopsied.

RESULTS

Barium study of the patient with esophageal involvement by regional enteritis revealed a large penetrating ulcer crater in the lower mid-esophagus (Fig. 1). The appearance of the stomach of 1 patient with gastric involvement by regional enteritis was normal on barium examination, but the examination had been done 2 months before surgery. Barium examination of the other 6 patients who had gastric involvement showed narrowing of the distal portion of the antrum and the pylorus, with loss of the normal rugal pattern in this region in 5 patients, and nodularity in 1 patient (Fig. 2, *A* and *B*). One of the patients was observed at fluoroscopy by us (D.A.L. and H.C.C.) and practically no motor activity in the stomach, resulting in a large amount of retained fluid, was seen (Fig. 2*C*).

The appearance of regional enteritis in the duodenum had characteristics similar to those seen in the more distal portion of the small bowel. These varied from slight narrowing of the affected segment of duodenum, with thickening of its mucosa (Fig. 3*A*), to a deformity and stenosis of a degree sufficient to cause obstruction, with associated gastric dilatation (Fig. 3*B*). In 1 patient the narrowed segment of duodenum caused dilatation of the more proximal portion of the duodenum and was associated

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FIG. 1. A 46 year old woman. Large ulcer situated in lower mid-esophagus. At esophagoscopy, a biopsy specimen was obtained from the ulcer. There were characteristic colon changes of regional enteritis on barium examination.

with reflux of barium into the pancreatic and biliary ducts (Fig. 3*C*).

When both stomach and duodenum were involved, the narrowing extended from the antrum to the proximal portion of the duodenum, so that the normal anatomic landmarks of the antrum, pyloric canal, and duodenal cap were lost, these areas forming a continuous tapering segment (Fig. 4, *A* and *B*).

In 10 patients in our series, the terminal portion of the ileum was also involved by regional enteritis, and in 7 of these, part of the colon was involved in addition. In 1, the colon (but not the terminal ileum) was also involved. In 6 of the patients, the characteristic involvement of the distal portion of the ileum or the colon was seen on barium study. Three patients had had the involved segments removed previously. In 2 more, the diagnosis had not been established: in 1, because barium study had revealed no abnormality, and in the other, because the distal portion of the bowel was not examined roentgenologically.

The patient with esophageal involvement had a history of painful dysphagia relieved by steroid medication and aggravated when the use of the steroids was discontinued. Clinical manifestations in the other patients were remarkably similar. Seven of the 8 patients with duodenal involvement lost weight and had epigastric pain; the pain was only moderately relieved by antacids. The eighth patient had frequent postprandial vomiting, as had 6 of the other patients. In the 5 patients with gastric as well as duodenal involvement, anorexia or easy fatigability were additional symptoms. Neither of the 2 patients with gastric involvement who did not have duodenal involvement had vomiting or significant epigastric pain: 1 patient had anorexia, whereas the other had mild epigastric distress.

Seven patients had abdominal cramps and diarrhea as well as the symptoms from upper gastrointestinal involvement; 1 patient had had abdominal cramps and diarrhea, but these had been in remission for the previous 3 years. The remaining 3 patients gave no history of diarrhea or cramps. Nevertheless, in 1 of these patients, a small bowel examination was done, and the distal ileal involvement was seen (Fig. 2*B*). In the other 2 patients, regional enteritis was not suspected, and further barium studies were not obtained. The correct diagnosis was not made until surgery.

DISCUSSION

The esophageal ulcer in the patient with regional enteritis that involves the colon was considered the result of esophageal involvement by regional enteritis because there were no other predisposing causes. In this case, esophagoscopy had not been performed prior to the discovery of the ulcer; Barret's ulcer was excluded as the result of

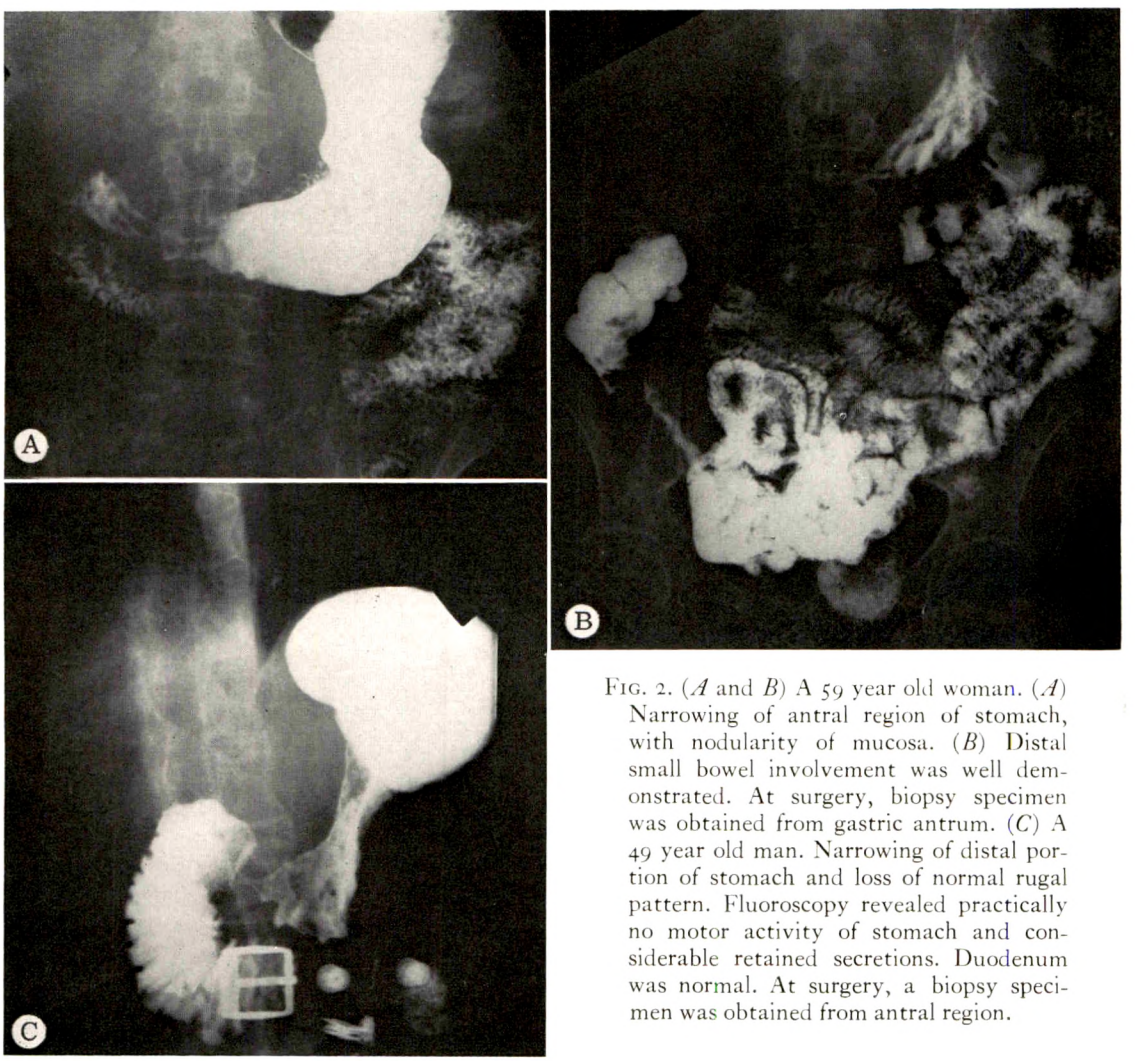


Fig. 2. (*A* and *B*) A 59 year old woman. (*A*) Narrowing of antral region of stomach, with nodularity of mucosa. (*B*) Distal small bowel involvement was well demonstrated. At surgery, biopsy specimen was obtained from gastric antrum. (*C*) A 49 year old man. Narrowing of distal portion of stomach and loss of normal rugal pattern. Fluoroscopy revealed practically no motor activity of stomach and considerable retained secretions. Duodenum was normal. At surgery, a biopsy specimen was obtained from antral region.

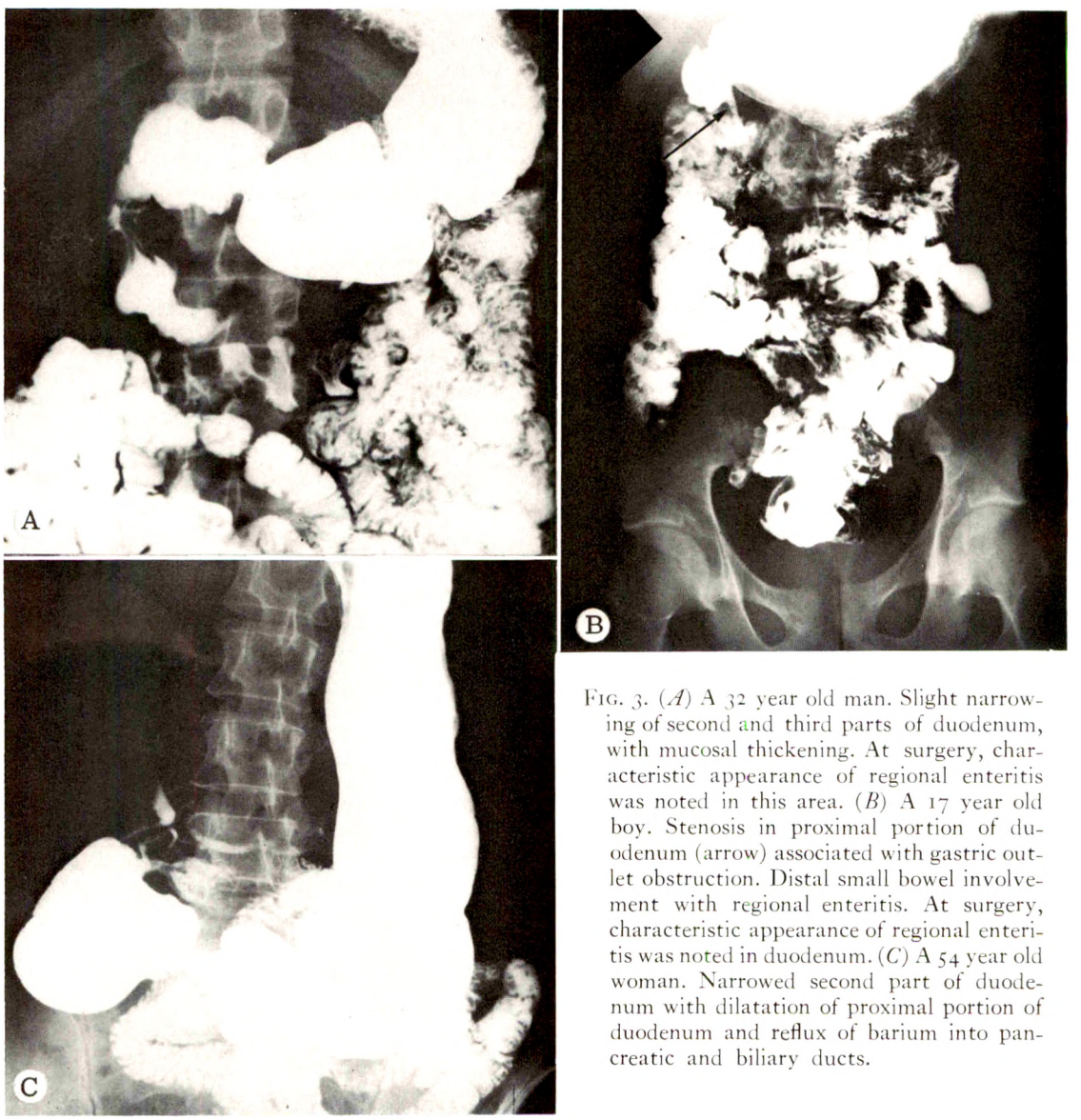


FIG. 3. (*A*) A 32 year old man. Slight narrowing of second and third parts of duodenum, with mucosal thickening. At surgery, characteristic appearance of regional enteritis was noted in this area. (*B*) A 17 year old boy. Stenosis in proximal portion of duodenum (arrow) associated with gastric outlet obstruction. Distal small bowel involvement with regional enteritis. At surgery, characteristic appearance of regional enteritis was noted in duodenum. (*C*) A 54 year old woman. Narrowed second part of duodenum with dilatation of proximal portion of duodenum and reflux of barium into pancreatic and biliary ducts.

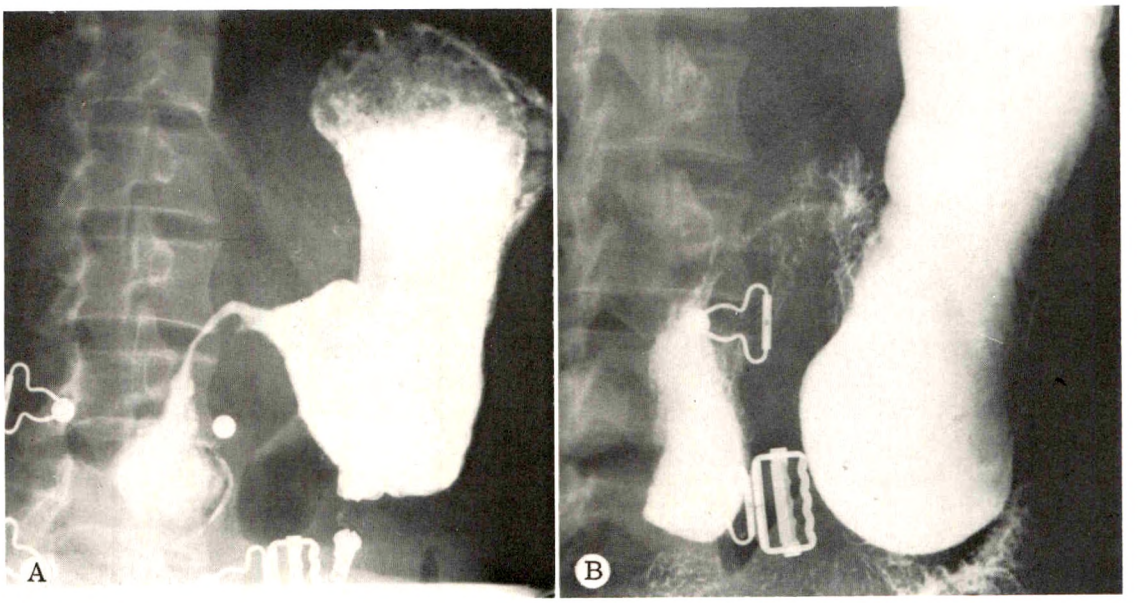


Fig. 4. (*A*) A 28 year old man. Narrowed antrum, pylorus, and proximal portion of duodenum. Normal anatomic landmarks in this area have been lost. At gastroscopy, a biopsy specimen was obtained from antral region. (*B*) A 26 year old man. Narrowed distal portion of stomach and adjacent duodenum similar in appearance to that in *A*. At surgery, characteristic appearance of regional enteritis was noted in antrum and duodenum.

histologic examination of a biopsy specimen from the ulcer, the biopsy revealing inflammatory esophageal mucosa; and the ulcer was probably not induced by steroids because it failed to heal when the use of these medications was discontinued and healed when the dosage of steroids was increased and continued. A case of regional enteritis of the colon and esophageal ulceration identical to this has been reported by Gelfand and Krone.[3]

When inflammatory changes are demonstrated roentgenologically in the distal portion of the stomach or in the duodenum, the diagnosis of regional enteritis should be included with those of lymphoma, carcinoma, and eosinophilic gastritis. A known history of regional enteritis or the demonstration of the characteristic features of this disease in a more distal segment of bowel should account for these proximal inflammatory changes. Because of the known presence of regional enteritis in 8 patients in our series, gastric or duodenal involvement was correctly diagnosed before surgery. In 2 of the patients in our series, diagnosis was not made until exploratory laparotomy because neither patient had symptoms of abdominal cramps or diarrhea and 1 of these patients had distal ileal involvement that was not seen on the barium study; in the other patient, neither small bowel nor colon examination was done. This patient had gastric involvement but no duodenal involvement, and the roentgenologic appearance of the stomach was similar to that of involvement by scirrhous carcinoma (Fig. 2*C*).

Difficulty in diagnosis might be encountered when there are few or no symptoms resulting from distal small bowel involvement, particularly in the patients with gastric involvement who do not have duodenal involvement. Correct diagnosis, however, can be established if a careful roentgenologic examination of the small bowel or colon is undertaken. In all patients in our series, there was (or had been)

involvement of the distal portion of the small bowel or colon by regional enteritis.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes scirrhous carcinoma of the stomach, lymphoma, and eosinophilic gastritis. Because gastric carcinomas rarely cross the pylorus, this diagnosis generally can be excluded when the duodenum as well as the stomach is involved with regional enteritis. In the 2 patients with gastric involvement who did not have duodenal involvement, the antrum of the stomach was more pliable on palpation than might be expected in scirrhous carcinoma. Examination of the small bowel and colon established the diagnosis of previously unsuspected regional enteritis in 1 of these cases, and probably would have done so in the other case, had it been undertaken. Lymphoma may be indistinguishable from regional enteritis involving the small bowel, but such a lesion is likely to cause prominence rather than loss of the rugal pattern in the stomach. Eosinophilic gastritis results in pyloric narrowing, but will be associated with peripheral eosinophilia invariably and with a history of allergy half of the time.[2]

SUMMARY

The roentgenologic features in 1 case of regional enteritis with involvement of the esophagus and in 10 cases of regional enteritis with gastric or duodenal involvement are described. Correct diagnosis was established prior to surgery or biopsy in 9 of the cases. In the other 2 cases, diagnosis was not established prior to surgery because the distal portion of the small bowel was normal roentgenologically in 1 case and was not examined in the other. The colon was not examined in either case. Difficulty in diagnosis may be encountered if the stomach, but not the duodenum, is involved with regional enteritis because the roentgenologic findings may resemble those of scirrhous carcinoma. Careful roentgenologic examination of the small bowel and colon will almost always reveal an area with characteristic changes of regional enteritis, and thus will account for the gastric or duodenal abnormalities.

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CROHN'S DISEASE WITH PREDOMINANT INVOLVEMENT OF THE APPENDIX*

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THE frequency of appendiceal involvement in Crohn's disease is difficult to determine. In their book, Crohn and Yarmis[1] indicate that the appendix had been removed in over 25 per cent of patients subsequently diagnosed as having regional "ileitis," but they also state that "the appendix in the resected specimens of combined ileocolitis does not participate in the peculiar and characteristic granulomatous inflammation." In one of their case illustrations, however, the appendix is graphically represented as being incorporated in the zone of disease.

In another large series,[9] where 377 specimens of ileum and colon involved with Crohn's disease were examined, the appendix was not specifically mentioned in the tabulation of disease distribution. The appendix was also excluded from involvement, according to the distribution diagrams, in a study of an additional 30 cases, all with colon disease.[7] However, in a different group of 75 patients with disease in the large bowel, 5 cases did involve the appendix, 2 of which were not in continuity with other areas of abnormality;[6] and 24 per cent of 120 specimens with Crohn's disease showed gross involvement of the appendix according to yet another pathologic review.[10]

There have been isolated reports of disease primarily involving the appendix,[2,3] although a claim has been made that confinement to even the cecum is rare.[12] Entities called fibroplastic appendicitis and appendicular granulatoma have also been described. Their histologic features have been variably interpreted as being compatible with Crohn's disease,[5,8] the chronic result of a prior appendicitis, possibly with a contained perforation,[4,13] or a granulomatous infection.[11]

REPORT OF CASES

CASE 1. A 27 year old male dentist was admitted to the University of Iowa Hospitals with a 4 day history of periumbilical pain with cramping, a 4×5 cm. tender mass in the right lower quadrant of the abdomen, a temperature of 102° F., and a white blood cell count of 23,000, with a shift to the left.

On careful questioning he admitted having "stomach" problems since the age of 11 years, characterized by 1 to 4 day periods of cramping abdominal pain with alternating constipation and diarrhea, but without mucus or blood in the stool. In addition, a perianal abscess was drained several years ago. After a roentgenographic examination of his gastrointestinal tract 3 years previously, he was told that he had an "irritable colon."

His temperature returned to normal and the white blood cell count was 12,000, 72 hours after a course of tetracycline therapy had been instituted. The right lower quadrant mass, however, did not change in size, nor did the tenderness and cramping pain subside.

The patient's abdomen was explored 10 days after admission at which time a very firm mass, involving the ileocecal region, was encountered. Because a neoplasm could not be excluded a resection of the right colon and distal ileum was performed. The pathologic examination was reported as compatible with regional enteritis, primarily involving the appendix but also a small area of cecum and ileum. Microscopic fistulae were identified in the appendix, and nonspecific lymph follicle hyperplasia was verified in the ileum.

The postoperative course was complicated by temperature spikes to 104° F. beginning the 5th day, a rising white blood cell count to 21,000, and symptoms of small bowel obstruction. A sterile hematoma of 200 cc. was evacuated from the right lower quadrant of the abdomen on the 9th postoperative day. Concurrently a course of azulfidine therapy was begun. On the 37th postoperative day all wounds had healed and the patient was discharged (Fig. 1, *A* and *B*).

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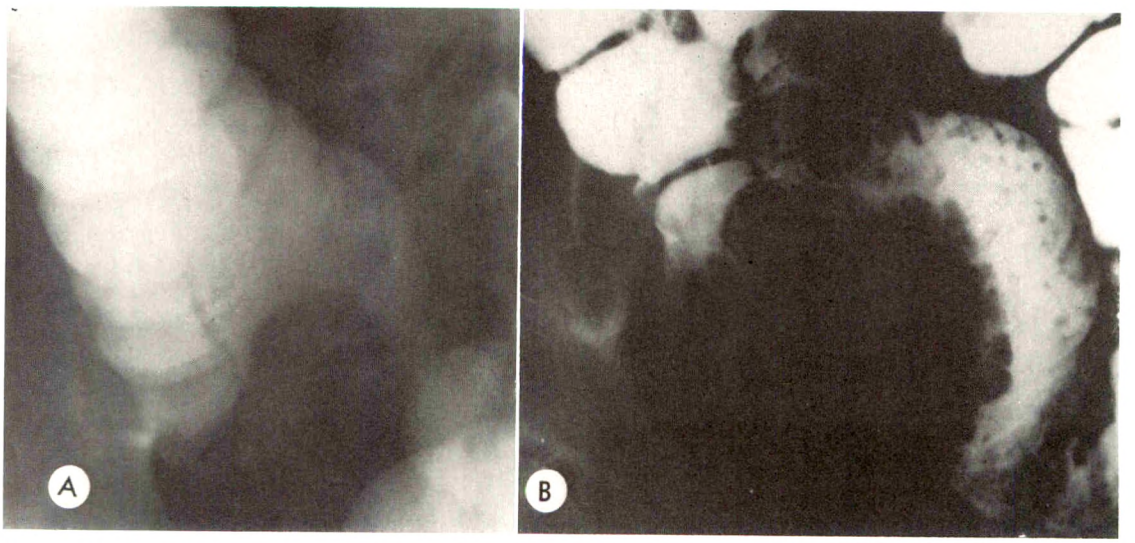


Fig. 1. Case I. (*A*) An extramucosal mass caused an impression defect upon the tip of the cecum. The remainder of the colon was normal. (*B*) The distal 10 cm. of ileum was displaced medially and superiorly by the mass arising from the region of the appendix. Although there was no evidence of ulceration or stricture, some enlarged folds near the ileocecal valve and more proximal 2 to 3 mm. filling defects, consistent with lymph follicle enlargement, could be seen. No abnormalities were detected in the more proximal portions of the upper gastrointestinal tract.

Case II. A 37 year old woman was admitted to the University of Iowa Hospitals with a 6 day history of cramping abdominal pain and distention but without malaise or temperature elevation. During the prior 8 months she had other episodes of this cramping pain and had stopped eating high residue foods.

On physical examination a 3×4 cm., tender, movable mass was palpated in the right lower quadrant of the abdomen, and hyperactive bowel sounds were heard. Her temperature was 101° F., and the white blood cell count was 8,000, without a shift.

Despite 5 days of nasogastric suction, intravenous fluids and ampicillin, the patient's abdomen remained distended and tender in the right lower quadrant. Her fever also persisted at 100° to 101° F.

At a laparotomy performed 7 days after admission, an inflammatory mass composed of cecum, terminal ileum and appendix was encountered. The distal 5 cm. of ileum was thickened with "fat wrapping" and many large lymph nodes were present in the mesentery; the remaining small bowel and colon were normal. As the appendix was mobilized, a 2 mm. perforation of the ileum was encountered. This fistula was closed and the appendix removed. On pathologic examination the mucosa of the appendix was normal, but the wall was infiltrated with chronic inflammatory cells as well as noncaseating granulomata. The interpretation was Crohn's disease.

Her postoperative course was uncomplicated. She was discharged on the 8th day after a course of therapy consisting of azulfidine and prednisone had been instituted (Fig. 2, *A* and *B*).

Discussion

The roentgenographic features of the cases presented are by no means specific for Crohn's disease. In each instance our interpretation favored the diagnosis of appendiceal abscess. Indeed, any mass lesion arising from the region of the appendix, be it mucocele, neoplasm, or localized inflammation, could produce at least a similar appearance. Although the lymph follicle enlargement seen in the distal ileum of Case I and the fold thickening in Case II did tend to indicate an inflammatory origin for the mass, the medial and superior displacement of the distal ileum influenced us against the diagnosis of Crohn's disease, in which the involved ileum tends to be retracted inferiorly and applied towards the cecum.

Crohn's disease predominantly involving the appendix is unusual. Despite the roentgenographic features more suggestive of appendiceal abscess, a clinical course

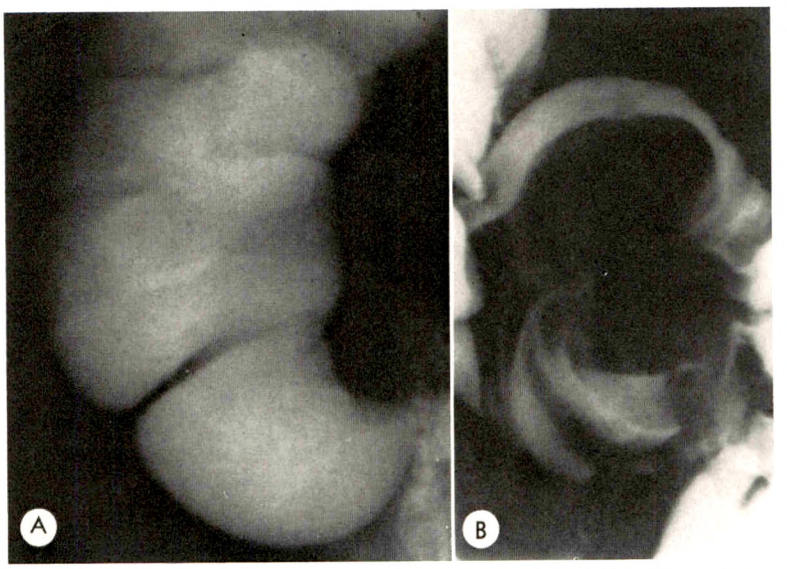


Fig. 2. Case 11. (*A*) The colon examination was normal except for an area of diminished distensibility along the medial aspect of the cecal tip, suggesting a mass. (*B*) The upper gastrointestinal examination also failed to reveal any proximal abnormalities. However, the distal 7 cm. of ileum as well as the tip of the cecum were displaced by the appendiceal mass, which was best defined on this delayed fluoroscopic spot roentgenogram. An area of persistent narrowing approximately 5 cm. proximal to the ileocecal valve probably contributed to the patient's symptom of cramping. Although the mucosal folds of the displaced ileum and cecum were enlarged, no penetrating ulcers of fistulae could be demonstrated.

which is atypical or having specific features suggestive of Crohn's disease (such as the perianal abscess in Case 1) should lead one to consider this entity.

SUMMARY AND CONCLUSIONS

Two cases are reported in which Crohn's disease predominantly involved the appendix.

The roentgenographic findings were nonspecific, resembling those seen in appendiceal abscess or other mass lesions arising from the area.

Pertinent clinical data, however, should lead one to consider this entity as a diagnostic possibility.

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ROENTGEN STUDIES IN EXPERIMENTAL COLONIC OBSTRUCTION

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PREVIOUS studies of the appearance of the colon in colonic obstruction have been based on retrospective analyses of roentgenograms of cases with colonic obstruction.[2,6,11,13] In a study on colonic ileus and its differential roentgen diagnosis by Bryk and Soong,[2] the altered appearance of the dilated colon and its contents in colonic obstruction was emphasized. A number of roentgen features were noted such as fluid filled colon, mottled fluid content, ragged inner colonic contours, thickened septa and accentuated haustrations with increased number of septa.

No experimental studies were available to explain the development and the significance of these findings.

Clinical reports of elevated intraluminal pressure in the obstructed colon[4,16,18] were available, but no attempt has been made to correlate these with the roentgen findings either on an experimental or clinical basis.

Studies in experimentally produced colon obstruction were therefore instituted in an attempt to understand more clearly the changes in the obstructed colon as visualized on the plain roentgenogram.

Only a few studies of experimentally produced colonic obstruction have been published. Glotzer and Pihl[7] obstructed the colon of dogs and demonstrated the development of colitis proximal to the obstructed segment. Hopkinson and Schenk[10] produced colonic obstruction in dogs for the purpose of studying the effect of hyperbaric oxygen on experimental intestinal obstruction. Dennis *et al.*[5] obstructed the cecal appendage of rabbits for the purpose of studying the etiology of acute appendicitis.

MATERIAL AND METHOD

The experimental studies were done on the laboratory animals which were most readily available—the dog and the rabbit. These were used despite the fact that there is a considerable difference in the anatomy of their colons as compared to that of the human.

Six dogs were studied. The distal colon was obstructed by a length of surgical tape passed around the bowel and tightened to obliterate the luminal opening. The tape was sutured to itself and the bowel was enfolded over it. In all the dogs studied, roentgenographic evidence of obstruction developed. This improved spontaneously over a period of a few weeks due to spontaneous recanalization of the colon at the site of the ligature. This finding has been previously reported by Hopkinson and Schenk.[10] In 2 of these dogs, 38 and 63 days respectively following the first obstruction, the colon was re-obstructed in the same fashion as in the first obstruction.

In 3 of the 4 other dogs studied, catheters were introduced into the colon proximal to the obstruction. The proximal ends of the catheters were attached to McQuigg adaptors and buried subcutaneously for the purpose of measuring intraluminal pressure and correlating it with the roentgenograms.

Following obstruction all the dogs were fed their normal diet.

Abdominal roentgenograms consisting of supine, prone and erect films were obtained at intervals until the animal expired or the

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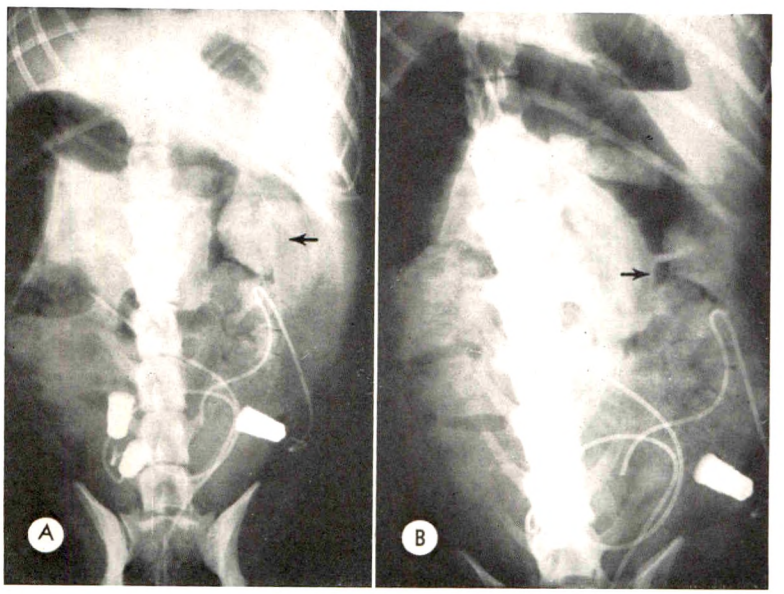


FIG. 1. Dog E. Colon obstruction of 4 days' duration. Intraluminal pressure 6.0 cm. of water. (*A*) Supine roentgenogram. (*B*) Erect roentgenogram. Note the 3 catheters with McQuigg adaptors, one in the cecum, another in the descending colon and the third in the rectum. The colon is slightly dilated and gas distended. There are no fluid levels in the erect roentgenogram. There is fecal retention in the left colon with particulate fecal matter outlined by the gaseous distention (arrows).

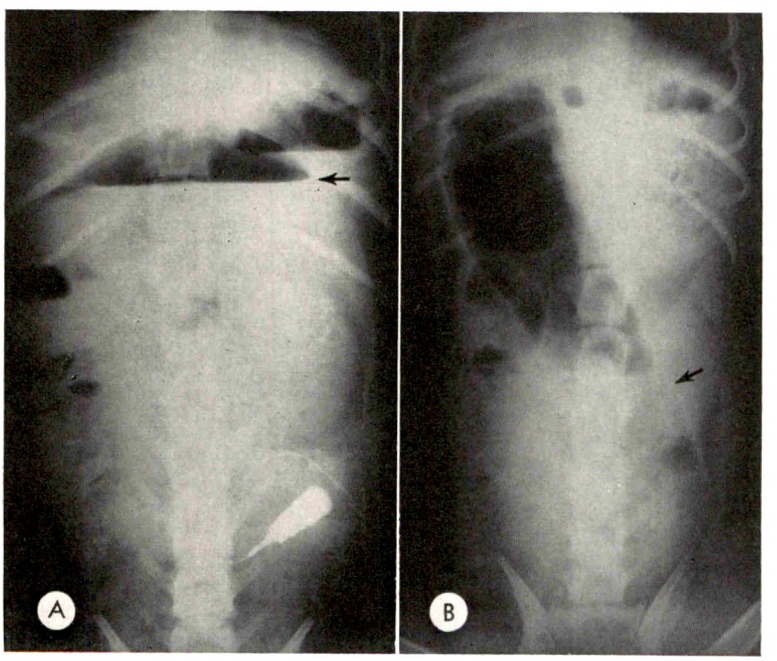


FIG. 2. Dog F. (*A* and *B*) Erect roentgenograms. (*A*) Colon obstruction of 11 days' duration. Intraluminal pressure 41 cm. of water. Note the catheter with the McQuigg adaptor situated in the left colon. The colon is predominantly fluid filled with a long air fluid level present in the transverse colon (arrow). The fluid is roentgenographically homogeneous. There is only minimal small bowel distention indicating competence of the ileocecal junction. (*B*) Eleven days later, 22 days after obstruction. There is evidence of partial relief of the obstruction. There is more gas and less fluid in the distended colon. The retained fluid has developed a mottled coarsely granular appearance (arrow). The catheter previously present in the descending colon was dislodged. The dog went on to complete resolution of the obstruction and normal roentgenographic appearance 7 days after this study.

obstruction resolved spontaneously. When intraluminal pressures were measured, roentgenograms were obtained at the same time. The pressures were measured by re-opening the incision and puncturing the McQuigg adaptors with a needle. The adaptors were flushed with saline and connected to a saline filled water manometer. The pressures were recorded in cm. of water. The McQuigg adaptors were pulled out by the animals or spontaneously ejected after a few days so that the longest time interval after obstruction for which pressures were obtained was 11 days.

Attempts to obstruct the rabbit colon by surgical tape were unsuccessful because the tape cut through the thin walled bowel with leakage of fecal content and death from peritonitis. Obstruction was therefore produced in the distal colon by transecting the colon and suturing the transected ends.

Five rabbits were thus obstructed and followed by serial roentgenograms until they expired. Seven other rabbits were obstructed and followed for a specified time interval. After abdominal roentgenograms, intraluminal pressures were determined at laparotomy and the animals sacrificed. The intraluminal pressures were measured by puncturing the exposed colon with a needle which was connected to a saline filled water manometer. The pressures were recorded in cm. of water. In a few of these rabbits, specimens were obtained for roentgenographic study of the inflated bowel and for histologic study.

In 2 rabbits, the cecum was obstructed proximal to the ileocecal junction producing a closed loop type of obstruction. These were followed for 6 and 8 days respectively, roentgenograms made and intraluminal pressures measured.

For comparison with the colon of the obstructed rabbits, air enema examinations were performed on 4 normal rabbits. Specimens of the cecum and ascending colon were obtained and these were washed, inflated and roentgenographed for comparison with the specimens from the obstructed colons. One specimen for histologic study was also obtained.

RESULTS

A. COLON OBSTRUCTION IN THE DOG

1. *Clinical aspects.* Obstruction of the colon produced clinical abdominal distention which increased during the first week. The animals continued to eat their normal diet. Maximum clinical and roentgenographic distention occurred at about 10 days after which the animals began to improve due to recanalization of the colon. One dog that was re-obstructed 38 days following the first obstruction went on to colonic perforation and death 5 days after the second obstruction.

2. *Roentgen findings.* Following obstruction the colon became diffusely distended. There was usually only minimal distention of the small bowel indicating the presence of a fairly competent ileocecal junction. The colon of the dog is tubular with no haustrations,[14] so that the distended colon appeared sausage shaped following the periphery of the abdomen.

The intraluminal contents varied with the degree of distention. With minimal distention, early in the course of obstruction, there was no fluid. The retained fecal content was visualized with the particulate fecal matter outlined by gas in the distended colon (Fig. 1, *A* and *B*). At the time of maximum obstruction and distention, however, there was considerable fluid in the colon. The fluid was fairly homogeneous as seen roentgenographically (Fig. 2*A*).

TABLE I

COLON OBSTRUCTION IN DOGS

Dog	Time Interval from Colon Obstruction (da.)	Average Intraluminal Pressure (cm. water)
D	3	5.8
E	4	6.0
F	4	11.0
	11	41.0

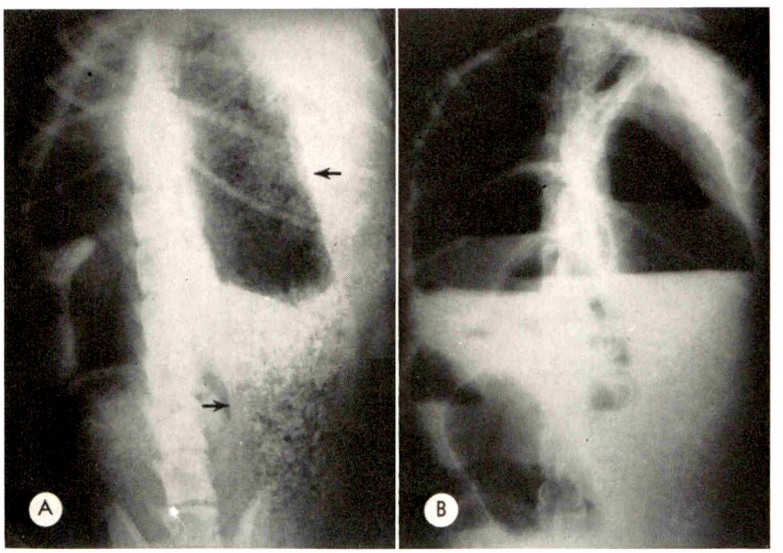


Fig. 3. Dog B. (*A* and *B*) Erect roentgenograms. (*A*) Colon obstruction of 38 days' duration. Clinically and roentgenographically there was evidence of partial spontaneous relief of the obstruction. The colon is predominantly gas distended. The retained fecal content has a mottled coarsely granular appearance (arrows). (*B*) Four days after re-obstruction and 1 day before spontaneous perforation and death. The colonic gaseous distention has increased and there is a large amount of retained fluid with air fluid levels. Note that the colonic contents have changed from the coarsely mottled fecal material present prior to re-obstruction to a predominantly homogeneous fluid. There is also moderate small bowel dilatation with multiple air fluid levels.

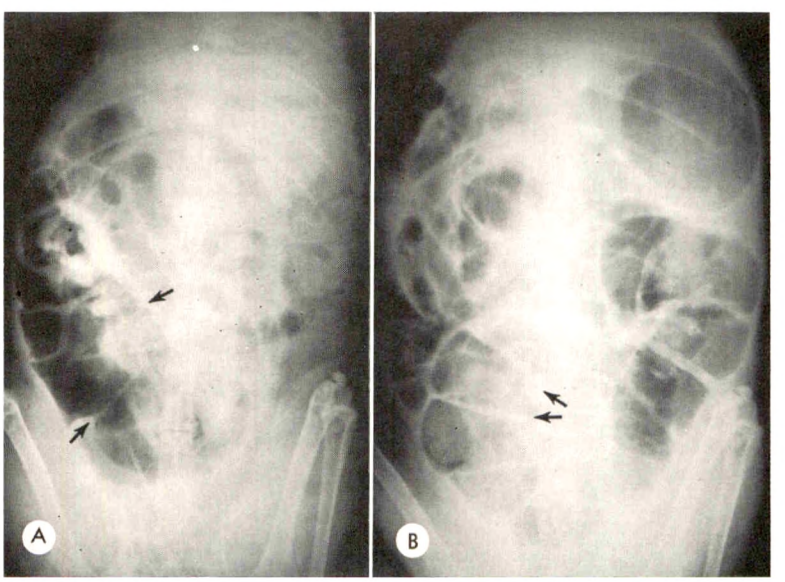


Fig. 4. Rabbit A. (*A* and *B*) Prone roentgenograms. (*A*) Colon obstruction of 4 days' duration. There is minimal intestinal distention. The predominant distention is that of the cecum in the right side of the abdomen. Note the cecal septa (arrows) completely crossing the lumen. These are the roentgenographic representation of the rabbit cecal spiral valve. (*B*) Ten days later, obstruction of 14 days' duration. The intestinal distention has increased markedly and involves the colon, small bowel and stomach. Note the increased thickness of the cecal septa as compared to the previous study (arrows).

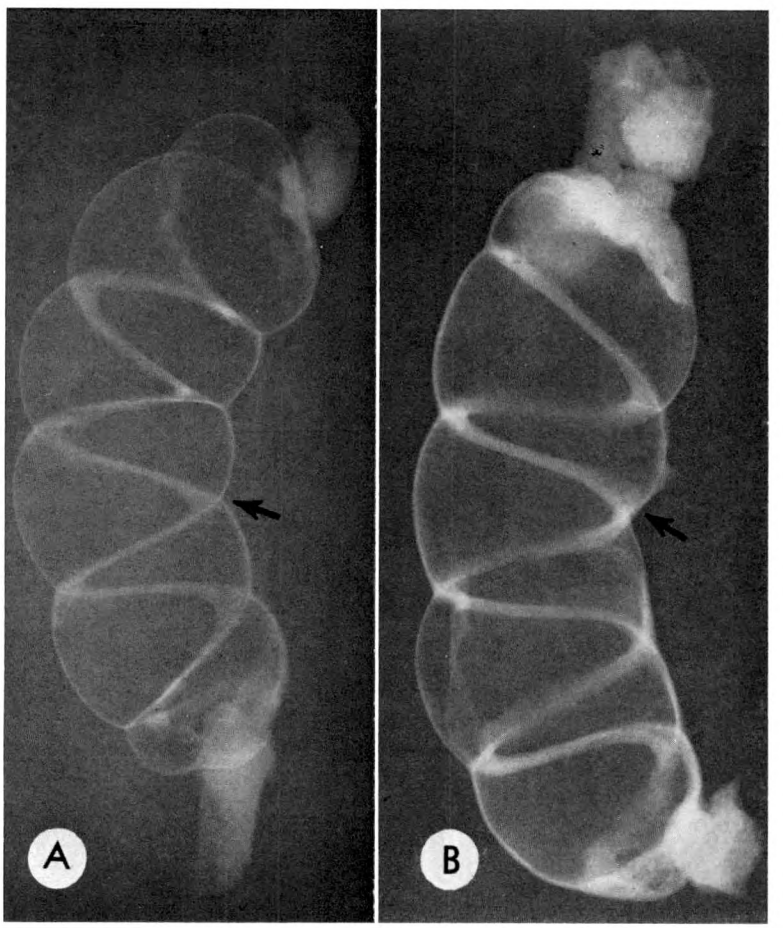


FIG. 5. (*A*) Excised inflated cecum of a nonobstructed rabbit. Note the spiral valve crossing the cecal lumen. The average thickness of those septa seen sharply in profile is 1.5 mm. (arrow). (*B*) Excised inflated cecum of Rabbit J, 14 days after colon obstruction. Note the increased thickness of the cecal septa with an average thickness of 3 mm. (arrow).

With spontaneous improvement, the volume of fluid decreased and the fluid developed a mottled coarsely granular roentgen appearance (Fig. 2*B*). As obstruction resolved completely, fluid was no longer present and the fecal content again appeared solid. In 1 of the cases that was re-obstructed 38 days following the first obstruction and went on to perforation, the intraluminal content changed from coarsely mottled fecal material to predominantly homogeneous fluid (Fig. 3, *A* and *B*).

3. *Intraluminal pressures.* These were studied in 3 dogs (Table 1). There was a rough correlation between the intraluminal pressure, the duration and degreee of distention, and the roentgen appearance of the intraluminal contents. In Dogs D and E there was minimal gaseous distention without any fluid retention. The fecal content was normal in appearance. This was associated with low pressures of 5.8 and 6 cm. of water, respectively (Fig. 1, *A* and *B*). In Dog F, there was slightly more prominent distention with retained fluid at 4 days associated with a pressure of 11 cm. of water. At 11 days when distention was most marked, the colon was predominantly fluid filled and the pressure was 41 cm. of water (Fig. 2*A*). Unfortunately, after this measurement the catheter with the McQuigg adaptor became dislodged so that no

additional measurements were recorded. The animal, however, improved with an associated change in the intraluminal contents to mottled semisolid (Fig. 2*B*) and then normal, as noted in the other amimals previously described.

B. COLON OBSTRUCTION IN THE RABBIT

1. *Clinical aspects.* Following successful obstruction, the rabbits developed progressive abdominal distention. No vomiting or diarrhea was noted. The survival time following obstruction in the 5 animals, followed until death, varied from 10 to 16 days. Autopsy at the time of death did not reveal any cases of colonic perforation so that death was apparently due to the toxic effects of colonic obstruction.

2. *Roentgen findings.* The viscus which became most markedly distended after obstruction was the wide thin walled cecum. This occupied a major portion of the peritoneal cavity. The distended cecum was divided by multiple transverse septa which are the roentgenographic representation of the spiral valve.[1] The small closely packed haustrations in the dilated ascending colon with thin septa which did not completely cross the colonic lumen were usually also observed. There was associated diffuse small bowel distention which frequently obscured portions of the distended colon. The presence of small bowel distention indicated incompetence of the ileocecal junction.

(A) Septal thickening. With colonic obstruction there was evidence of thickening of the cecal septa. This was indicated: (a) by progressive increase in thickness of the cecal septa as the obstructed rabbits were serially roentgenographed (Fig. 4, *A* and *B*); (b) by comparing the excised and inflated cecum of the obstructed rabbits with that of the normal rabbit. In the latter case, the septa in the obstructed cecum measured 3 mm. in thickness while those of the normal cecum measured 1.5 mm. in thickness (Fig. 5, *A* and *B*); (c) by histologic study of the excised obstructed cecum compared to the normal nonobstructed cecum (Fig. 6, *A* and *B*). This showed septal thickening in the obstructed cecum caused predominantly by submucosal edema. The muscular layer which extends for a short distance into the base of the septa was also slightly hypertrophied. The wall of the obstructed cecum was similarly thickened by a combination of submucosal edema and muscular hypertrophy.

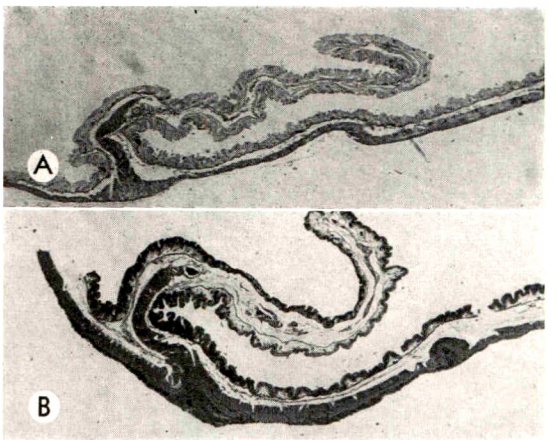


FIG. 6. Photomicrographs of longitudinal sections of the rabbit cecum. (*A*) Nonobstructed rabbit. (*B*) Rabbit J, 14 days after colonic obstruction. Note the thickening of the cecal septum and wall in the specimen from the obstructed rabbit. The septal thickening is due predominantly to submucosal edema and minimally to hypertrophy of the muscle which extends for a short distance into its base. The cecal wall is thickened as a result of both submucosal edema and muscular hypertrophy.

The thickening of the cecal septa was most obvious in the *in vitro* studies of the inflated bowel and in the histologic specimens. Evaluation was more difficult *in vivo* because of the change in orientation of the distended cecum from examination to examination due to the fact that the rabbit cecum is on a mesentery and has considerable mobility.[1] This caused the septa to appear thickened because of differences in their orientation to the roentgen beam. This phenomenon was shown to occur in the inflated *in vitro* specimen whose axis was not perpendicular to the roentgen beam. Change in position of the cecum also caused differences due to the effect on roentgen magnification. Another important

factor was the considerable thickening produced *in vivo* by adherent secretions and fecal matter.

Comparisons were therefore made in the same projection, with portions of the cecum similarly oriented and between septa that were sharply defined and smooth indicating that they were parallel to the axis of the roentgen beam and not coated with fecal matter.

(B) Accentuated haustrations and increased number of septa. This finding was demonstrated in the ascending colon. Following obstruction the haustrations became more prominent associated with an increase in the number of septa in any specified length of the colon. This was best demonstrated in the *in vitro* study when the excised and inflated ascending colon of the obstructed rabbit was compared with that of the normal rabbit (Fig. 7, *A* and *B*). *In vivo* demonstration was more difficult because the small ascending colon was obscured by the large distended redundant cecum and because the axis of the ascending colon was frequently not perpendicular to the roentgen beam. This finding was therefore seen *in vivo* in only 4 of the 12 cases studied (Fig. 8, *A* and *B*).

Histologic study of the excised obstructed colon as compared to the nonobstructed colon demonstrated thickening of the mus-

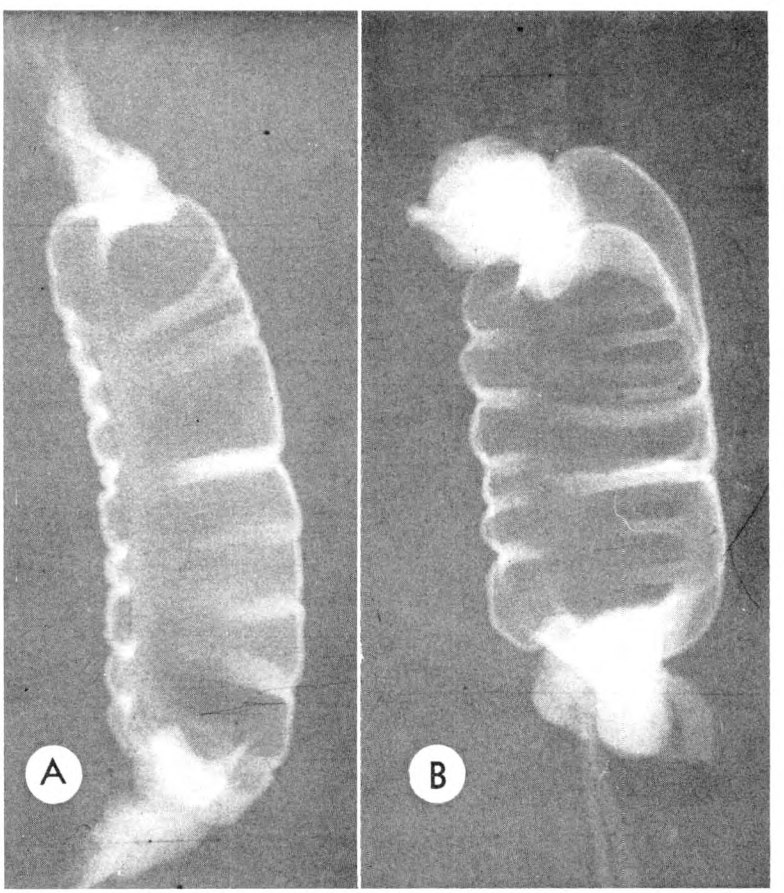


FIG. 7. (*A*) Excised inflated ascending colon of a nonobstructed rabbit. Note the multiple small haustrations along the colonic contours due to the teniae coli present in this portion of the rabbit colon. They produce the internal infoldings or septa which do not completely cross the colonic lumen. (*B*) Excised inflated ascending colon of Rabbit M. Colonic obstruction of 4 days' duration. Note the greater prominence of the haustrations associated with an increase in the number of septa per unit length of colon.

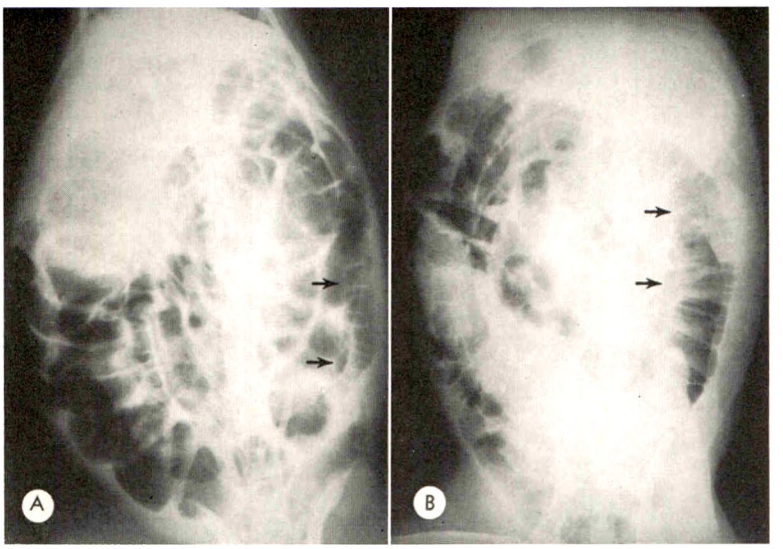


Fig. 8. (*A*) Air enema examination on a nonobstructed rabbit. Prone roentgenogram. There is diffuse gaseous distention of the colon and small bowel. Note the distended ascending colon along the left side of the abdomen (arrows). The haustrations are poorly defined but the septa are well seen. (*B*) Rabbit B. Prone roentgenogram. Colonic obstruction of 7 days' duration. Note the distended ascending colon along the left side of the abdomen (arrows). There is increased prominence of the haustrations and the septa are increased in number and prominence per unit length of bowel.

cular layer presumably due to muscular hypertrophy.

(c) Ragged inner colonic contour. This was shown to be due to fecal matter adherent to the inner colonic lining. In 2 cases showing this finding on the abdominal roentgenograms, the cecum was immediately removed, the fecal matter washed out and the bowel reinflated. The inner wall was no longer ragged indicating that the adherent fecal matter was the cause of this finding (Fig. 9, *A* and *B*). Fecal matter adherent to the cecal septa also produced apparent septal thickening but this could be recognized by the irregularity of these septa, which was similar to the ragged contour of the inner colonic wall.

A ragged inner colonic contour was usually seen when there was minimal fluid in the colon.

(d) Intraluminal contents. This varied with the degree of distention and the duration of obstruction. With minimal distention there was usually no fluid but the retained fecal matter was outlined by the gas content. With greater distention due to more severe or prolonged obstruction, fluid was seen which was roentgenographically homogeneous or slightly mottled. Since none of the rabbits improved spontaneously, the mottled fecal fluid seen in the dog with resolving obstruction was not observed.

3. *Intraluminal pressure studies.* Intraluminal pressures were measured in 7 rabbits after distal colon obstruction at intervals varying from 4 days to 14 days following obstruction. Pressures were also measured in 2 rabbits at 8 and 6 days in whom a closed loop obstruction of the cecum had been produced (Table II).

In the 7 rabbits with colon obstruction, the average pressures varied from 5 to 9.4 cm. of water. Roentgenographically, there was associated small bowel distention indicating incompetence of the ileocecal junction. The pressures observed correlated roughly with the degree of distention observed roentgenographically but not with the duration of the obstruction (Table II). There was associated minimal fluid retention. In the 2 cases with closed loop cecal

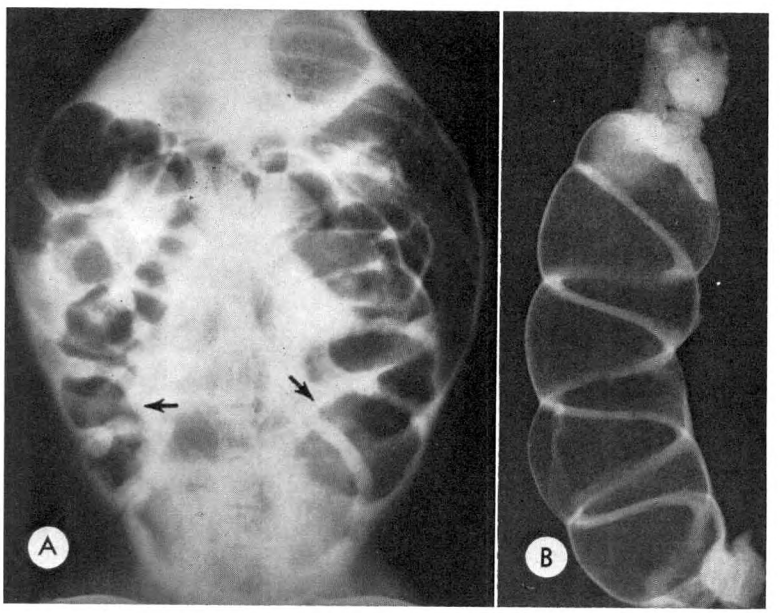


FIG. 9. (*A*) Rabbit L. Prone roentgenogram. Colonic distention of 14 days' duration. Intraluminal pressure of 8.1 cm. of water. The cecum is markedly distended. Note the ragged inner contour of the gas distended cecum (arrows). The cecal septa appear thickened and have a similar ragged contour. (*B*) Rabbit L. Excised cecum removed immediately after the roentgenographic study. The cecum was washed out and reinflated. The inner contours are now smooth indicating that the ragged contours noted *in vivo* were due to fecal matter adherent to the inner colonic lining.

obstruction there was greater fluid retention associated with significantly higher intraluminal pressures of 16 and 15.2 cm. of water, respectively (Fig. 10).

DISCUSSION

This study helps clarify some of the roentgen findings previously noted in human colonic obstruction.

The roentgen appearance of the intraluminal contents of the obstructed human colon varies. The various findings noted are: predominant gaseous distention with minimal fluid; predominant fluid filled colon; roentgenographically homogeneous fluid; roentgenographically mottled fluid; and particulate fecal matter.

The present study indicates that gaseous distention with particulate fecal matter is indicative of early obstruction or mild obstruction with low intraluminal pressures, as noted in the dog experiments. The fluid filled colon, in contrast, indicates acute severe obstruction with high intraluminal pressures. Both the dog and the rabbit studies showed a rough correlation between the volume of fluid and the intraluminal pressure.

Mottled coarsely granular fluid content has been roentgenographically observed in some cases of human colon obstruction.[2] This experimental study in dogs indicates that this pattern develops during improvement in the colonic obstruction. It is presumably due to improved ability of the colon to absorb fluid so that the fecal content became more particulate, with gas finely mixed with the fecal particles. In humans it probably implies a subacute or chronic type of obstruction with episodes of improvement. Clinical studies have indicated that in human colonic obstruction there frequently are episodes of improvement due to partial relief of the obstruction.[17]

The intraluminal pressure studies also show a rough correlation between the degree of distention and the intraluminal

pressure. This agrees with the clinical study correlating cecal size and the probability of impending perforation, which showed that perforation was related to the size of the distended cecum.[12] The degree of colonic distention and thus the intraluminal pressure was also shown to be related to the competence of the ileocecal junction. In the dog with little small bowel distention, high pressures developed in the colon, while the colonic pressures in the rabbits, all of whom showed small bowel distention, were low. Production of a closed loop obstruction in the rabbit by occluding the blind cecum resulted in the development of intraluminal pressures higher than those seen in rabbit colonic obstruction. Spontaneous colon perforation was produced only in the dog and not in the rabbit in whom the pressures were low and death was apparently due to the toxic effects of the obstruction.

The intraluminal pressures experimentally produced and measured in this study are comparable to those measured at surgery in clinical colonic obstruction. They varied from 5.8 to 41 cm. of water in the dog and from 5 to 16 cm. of water in the rabbit. Reported pressures in human colonic obstruction have varied from 1 to 52 cm. of water.[4,16,18] In one study, the average of 34 determinations was 14 cm. of water with the critical pressure for perforation felt to be 20 cm. of water.[4]

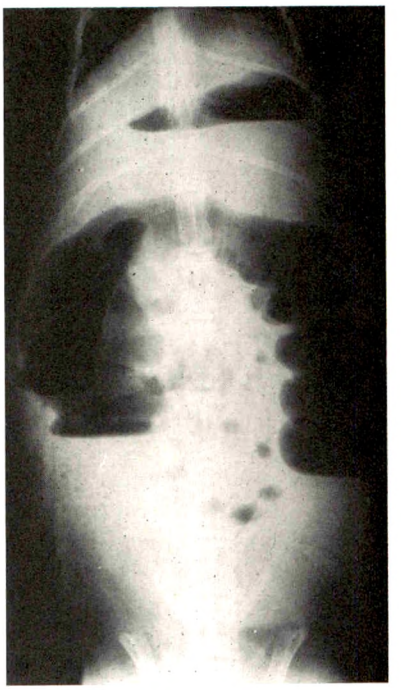


Fig. 10. Rabbit O. Erect roentgenogram. Closed loop cecal obstruction of 8 days' duration. Intraluminal pressure of 16 cm. of water. There is gaseous distention with a moderate amount of fluid. The fluid is roentgenographically homogeneous.

Table II

COLON OBSTRUCTION IN RABBITS

Rabbit	Time Interval from Colon Obstruction (da.)	Average Intraluminal Pressure (cm. water)	Degree of Colonic Distention
K	12	5.3	2+
J	14	9.4	4+
L	14	8.1	4+
M	4	5.9	3+
G	7	5.8	3+
H	7	5.0	1+
I	8	5.0	2+
	Cecal Obstruction		
O	8	16.0	
N	6	15.2	

The ragged inner colonic contour seen roentgenographically in clinical colonic obstruction was produced in some of the rabbits studied. It was shown to be due to adherent secretions and fecal matter since it was not present when the cecum was removed, washed and reinflated. Since it was seen when there was only minimal fluid in the distended cecum, it appears that it develops when the obstructed colon still retains the ability to re-absorb the fluid entering it from the small bowel so that semisolid fecal matter is formed which adheres to the inner wall of the distended colon. Thus, correlating previous clinical studies with the present experimental study, it would appear that the ragged colonic contour can be seen either in early obstruc-

tion or in chronic obstruction in which episodes of improvement have occurred.

It must be differentiated roentgenographically from a somewhat similar appearance seen in ulcerative colitis with toxic dilatation, where it is due to the inflammed edematous mucosal lining with pseudopolyp formation.[19] It has also been reported in cases of ulcerative colitis proximal to obstructing colonic lesions.[3,15] Differential diagnosis is possible because of the other manifestations of ulcerative colitis, seen in the distended colon in these cases.

Correlation of the changes in the colonic haustrations and septa seen in clinical colonic obstruction with that of experimental colonic obstruction produced in this study was not completely satisfactory because of the lack of a good experimental model. The dog colon is tubular lacking haustrations[14] so that the dog study was of no value from the standpoint of the haustral pattern. The rabbit study provided a few interesting correlations but the correlation was poor because of the differences in the anatomy of the human and rabbit colon.

The major portion of the rabbit colon is the blind cecum and appendix. The cecum is divided internally by a fixed anatomic structure known as the spiral valve which produces fixed sacculations of contour between the inner constrictions produced by this valve. The spiral valve consists of mucosa, submucosa, and an invagination of the cecal muscular coat. The muscular coat of the cecum contains a complete circumferential layer of both circular and longitudinal muscle. The spiral valve or fold roentgenographically produces septa which completely cross the lumen of the gas filled cecum.[1]

The rabbit anatomy is thus different from the human colon where the longitudinal coat is incomplete and consists of 3 teniae coli which are shorter than the colon, thus causing sacculations referred to as the colonic haustrations.[8] These are not fixed anatomic structures but vary with the activity of the teniae. Similarly, the septa seen roentgenographically are not fixed structures but are the infoldings produced internally by these haustrations. Since there are 3 rows of sacculations produced by the 3 teniae, the septa usually do not completely cross the colonic lumen as seen roentgenographically.

The rabbits studied did demonstrate roentgenographic evidence both *in vivo* and *in vitro* of thickening of the septa produced by the spiral valve. This was predominantly due to submucosal edema and minimally to hypertrophy of the muscle at their base. The other factor causing *in vivo* roentgen thickening was adherent secretions and fecal matter. Thus the septal thickening seen roentgenographically in the obstructed human colon is probably due to submucosal edema and muscular hypertrophy with superimposed thickening due to adherent secretions and fecal matter.

The evaluation of this finding roentgenographically is as difficult in the human as it is in the rabbit due to the fact that both angulation of the axis of the roentgen beam with respect to the septa and differences in magnification resulting from different object film distances can produce apparent thickening.

In the rabbit, the cecal septa are not obliterated by colonic distention as is noted in human colonic obstruction, since they are fixed anatomic structures which cannot be obliterated by chronic distention.

There is greater similarity between the haustrations of the rabbit ascending colon and those of the human. The proximal portion of the rabbit ascending colon contains 3 rows of small sacculations produced by 3 longitudinal teniae. The diameter, however, of the ascending colon is very small compared to that of the cecum. In addition, the haustrations are extremely small and closely packed as compared to those of the human colon.[1]

Their visualization was therefore inconstant in the obstructed rabbit probably because they were obscured by the predominant cecal and small bowel distention. Ap-

parently, only if this small segment of colon was oriented parallel to the roentgen beam and was located in the lateral aspect of the abdomen, where there was less obscuring bowel, was it visible roentgenographically.

The only correlative observation seen, as far as the septa and haustrations are concerned, was the fact that in the *in vitro* studies and in a few of the *in vivo* studies there was an accentuation of the septa and haustrations with increased number of septa per unit length, apparently secondary to the histologically demonstrated muscular hypertrophy resulting from colon obstruction. This is similar to the same finding described in early acute human colon obstruction.[2] The development of muscular hypertrophy in the obstructed human colon has also been histologically demonstrated.[9]

Haustral and septal obliteration was not produced, since the more chronic type of obstruction with chronic distention which is probably the factor in its production, was not seen in the experimentally produced rabbit colon obstruction.

SUMMARY

Experimental colon obstruction was produced in dogs and rabbits. The roentgenographic image as seen in the recumbent and erect abdominal roentgenograms was correlated with the clinical picture, the duration of obstruction, the intraluminal pressures and the autopsy studies. Roentgen findings seen in human colon obstruction such as fluid filled colon, mottled fluid content, ragged inner colonic contour, thickened septa and accentuated haustrations with increased number of septa were produced in the experimental animals.

The correlative studies were indicative of the following: (a) gaseous distention with particulate fecal matter is indicative of early obstruction or mild obstruction with low intraluminal pressures. Mottled fluid is seen in more chronic obstruction with episodes of improvement. The degree of roentgen colonic distention correlates with the intraluminal pressure. (b) The ragged inner colonic contour is due to adherent secretions and fecal matter. (c) Thickened colonic septa are due to submucosal edema, muscular hypertrophy and adherent secretions and fecal matter. Early obstruction with muscular hypertrophy produces accentuated haustrations with increased number of septa.

This experimental study thus provides a better understanding of the roentgen findings seen in the obstructed human colon.

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ROENTGENOGRAPHIC APPEARANCE OF CANINE HISTIOCYTIC ULCERATIVE COLITIS*

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CANINE ulcerative colitis was first described as an entity in 9 boxer dogs by Van Kruiningen and co-workers[8] in 1965, and was called "granulomatous colitis." The term, "canine histiocytic ulcerative colitis" was introduced by Sander and Langham[5] in 1968 and is a more descriptive term.

This type of colitis is seen in timid, but excitable boxer dogs. There appears to be a familial tendency. Clinically the dogs have a chronic, intermittent, bloody diarrhea, a progressive course and, in severe cases, cachexia.[1,8]

The roentgenographic changes in canine histiocytic ulcerative colitis have not been described in detail. Since the disease presents similarities to idiopathic chronic ulcerative colitis and granulomatous colitis of man, this canine entity is of interest.

MATERIAL AND METHOD

From more than 100 cases of canine colitis in the files of the Veterinary Medical Teaching Hospital, University of California at Davis (UCD), 24 were selected for review. The selection was based on 3 criteria: a clinical history of colitis; barium enema examination of the colon; and histopathologic sections of the colon.

The 24 dogs included 17 boxers ranging in age from 6 months to 8 years. The remaining 7 dogs were of different breeds and ranged in age from $2\frac{1}{2}$ to $12\frac{1}{2}$ years. These dogs had been brought to the Veterinary Medical Teaching Hospital by their owners for diagnosis and treatment of persistent or recurrent bloody diarrhea.

The clinical histories were obtained from the Veterinary Medical Teaching Hospital records and the histologic specimens from the Department of Veterinary Pathology UCD and the Department of Pathology at the University of California, San Francisco (UCSF).

The barium enema examinations were done in the radiology section of the Veterinary Medical Teaching Hospital at UCD and in the Experimental Radiology Laboratory UCSF.

The dogs were prepared for roentgenographic study by 24 hours of fasting and cleansing enemas. Most of the dogs were under barbiturate sedation at the time of examination; however, several were studied without sedation. A Bardex balloon catheter was inflated in the rectum and a 25 to 30 per cent barium suspension was instilled by gravity flow. All roentgenograms were obtained when the colons were filled with barium. Postevacuation and air-barium contrast ronetgenograms were obtained in 7 dogs. Plain roentgenograms of the abdomen were obtained in 4 dogs, 1 to several days before the barium enema examination (Fig. 1).

The roentgenograms were reviewed separately by 4 radiologists. The observations were tabulated and compared to clinical histories and proctoscopic descriptions. The histologic specimens were obtained by a biopsy through a proctoscope or at necropsy. The sections were examined and the results correlated with the roentgenograms.

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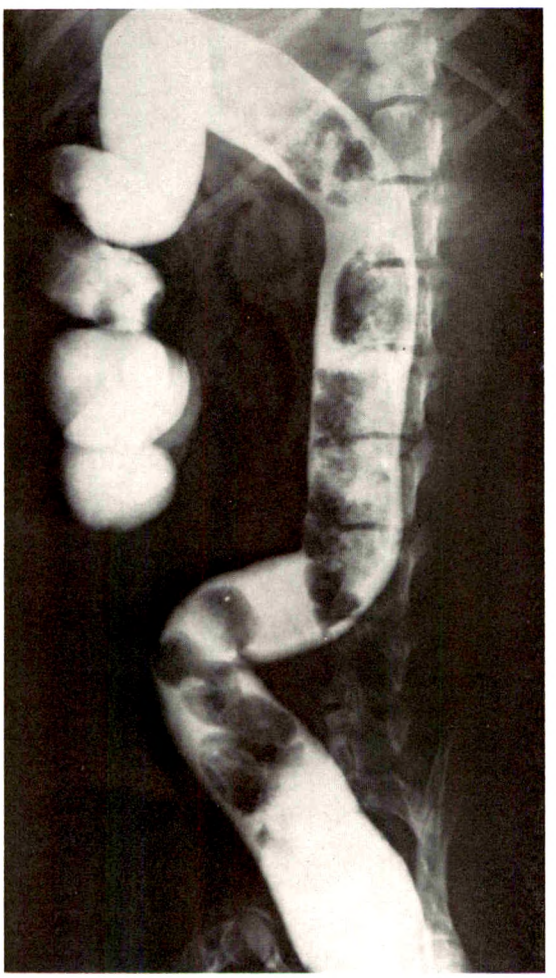


FIG. 1. Roentgenogram of the normal barium-filled canine colon. The walls of the colon are easily distensible and the mucosal surface is smooth and without haustra. In the postevacuation state, thin, transverse and longitudinal folds are present in the mucosal surface. The colon is divided into ascending, transverse, and descending segments similar to those in man. There is no sigmoid colon. The rectocolonic junction is placed at the pelvic inlet. The cecum is a diverticulum of the colon with a cecocolic valve. The ileocolic valve is in close proximity to the cecocolic valve and connects the ileum directly to the ascending colon. The cecum is corkscrew-like with a smooth mucosal surface. The ileum is frequently difficult to fill in retrograde manner but reflux will occur in the normal dog.[4]

TABLE I

ROENTGENOGRAPHIC EVALUATION OF BARIUM ENEMA EXAMINATION IN 24 DOGS WITH COLITIS

Roentgenographic Criteria	Frequency
Type of change	
Mucosal and submucosal abnormalities	
Thickened mucosal folds	16
Mucosal serrations (fine ulceration)	20
Deep ulcers	6
"Collar button" ulcers	3
Major deformity	
Spasm	11
Shortening and narrowing	7
Stricture	4
Dilatation (plain roentgenograms)	4
Degree of involvement	
Entire colon involved	7
Regional involvement	
Rectum	17
Descending colon	17
Transverse colon	9
Cecum	8
Terminal ileum	1

TABLE II

ROENTGENOGRAPHIC CLASSIFICATION OF CANINE COLITIS

Extent of Involvement	Mucosal and Submucosal Abnormalities	Major Colon Abnormalities
Mild		
Focal or regional	Edema, fine shallow ulcers	None or mild spasm
Moderate		
Long regional or multiple skipped areas	Fine shallow ulcers and deep ulcers	Spasm, slight to moderate rigidity, and shortening
Severe Total		
	Deep ulcers, "collar button" ulcers, and large areas of denuded mucosa	Marked shortening, rigidity, and narrowing; atonic gas-filled colon on plain roentgenogram

RESULTS

The roentgenographic changes and frequency of findings are summarized in Table I. The roentgenographic spectrum of change is categorized in Table II.

The barium enema examination revealed fine mucosal serrations as the most frequent abnormality (Fig. 2; and 3, *A–C*). These were seen throughout the colon,



Fig. 2. Mild colitis with mucosal serrations along the margins of the barium-filled colon. No contraction or spasm is present. Contrast medium in the bladder is from a prior arteriogram.

depending on the extent of involvement, but were most common in the distal colon and rectum. Thickened, irregular, mucosal folds were the second most frequent change (Fig. 4, *A* and *B*). These were seen in contracted portions of the colon, and on postevacuation roentgenograms. They were not seen in the distended colon.

Deep ulcers and "collar button" ulcers were infrequent but, when seen, were invariably associated with severe disease (Fig. 5, *A* and *B*). Large segments of denuded mucosa were difficult to detect, as the margins tended to blend into adjacent intact mucosa. The loss of normal plasticity of the colon was common. In mild cases, spasm and irritability were localized, usually in the rectum and distal

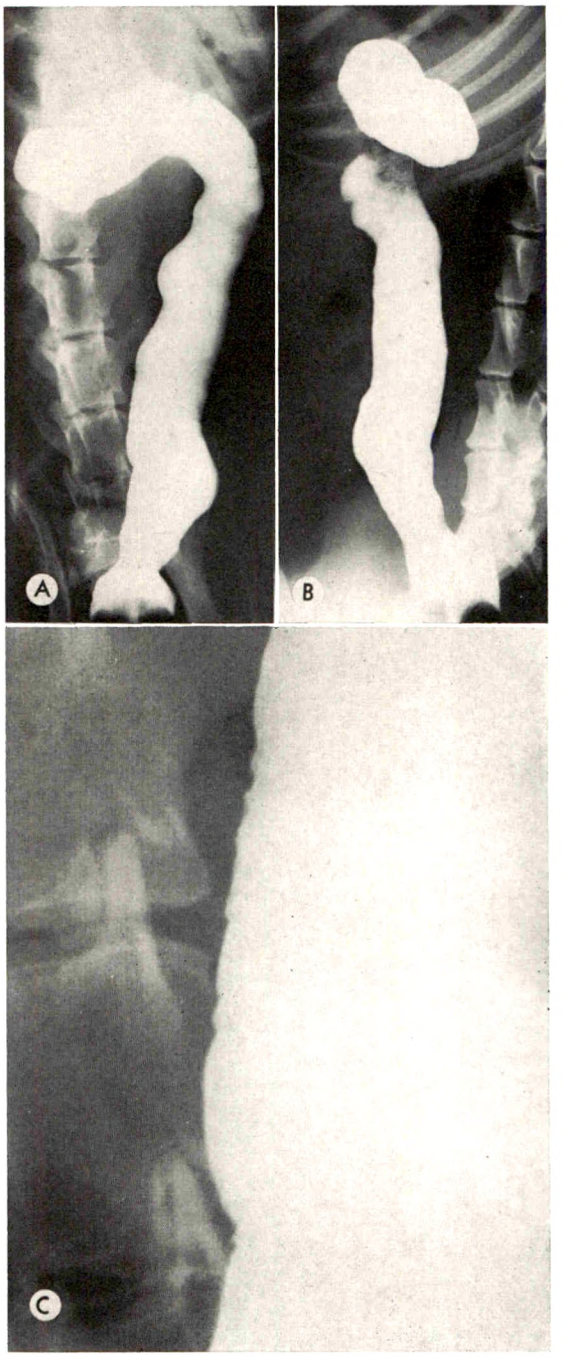


Fig. 3. Moderate colitis. (*A* and *B*) There is shortening and irregular narrowing of the colon with nonfilling of the cecum. (*C*) Magnification of the descending colon shows mucosal serrations.

colon. Progressive severity was manifested by persistent narrowing of large areas of the colon. Severe disease was associated with pronounced shortening and irregular

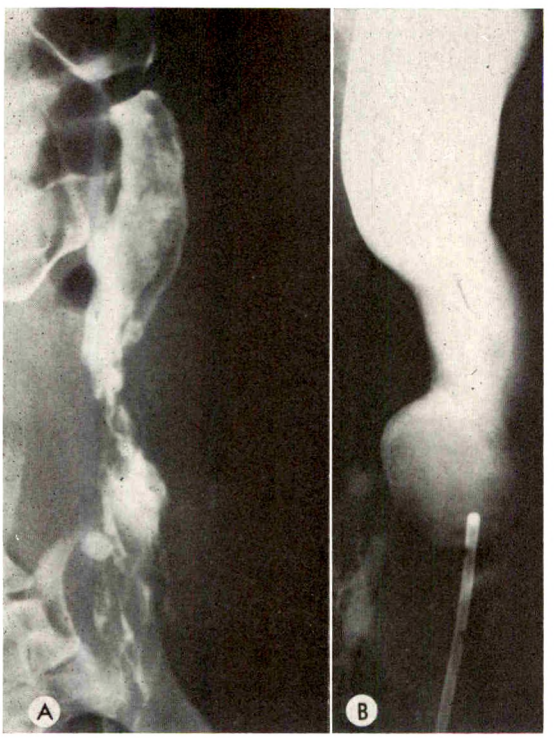


FIG. 4. (*A*) The thickened, irregular, mucosal folds are seen on the postevacuation roentgenogram but (*B*) disappear on distention of the colon.

narrowing of the entire colon (Fig. 6, *A* and *B*). The cecum in severe instances was contracted and frequently appeared ulcerated; in 2 dogs it was not filled by retrograde barium flow. Strictures, noted in 4 of the severely affected dogs (Fig. 7, *A* and *B*), were all in the distal descending colon.

The terminal ileum appeared ulcerated in only 1 dog; however, roentgenograms infrequently showed reflux of barium into the small bowel. Barium examination of the upper gastrointestinal tract and small bowel was obtained in 4 dogs and no abnormalities were seen. No fistulas, pericolonic abscesses, or carcinomas were seen. Plain roentgenograms of the abdomen showed an atonic, air-filled colon in 4 of the severely affected dogs (Fig. 8, *A* and *B*).

Roentgenographic abnormalities lagged behind clinical signs and were less severe than suggested by the clinical records. In several dogs with mild disease, only spasm and irritability were observed on roentgenograms. When proctoscopic appearance had been documented, the description of

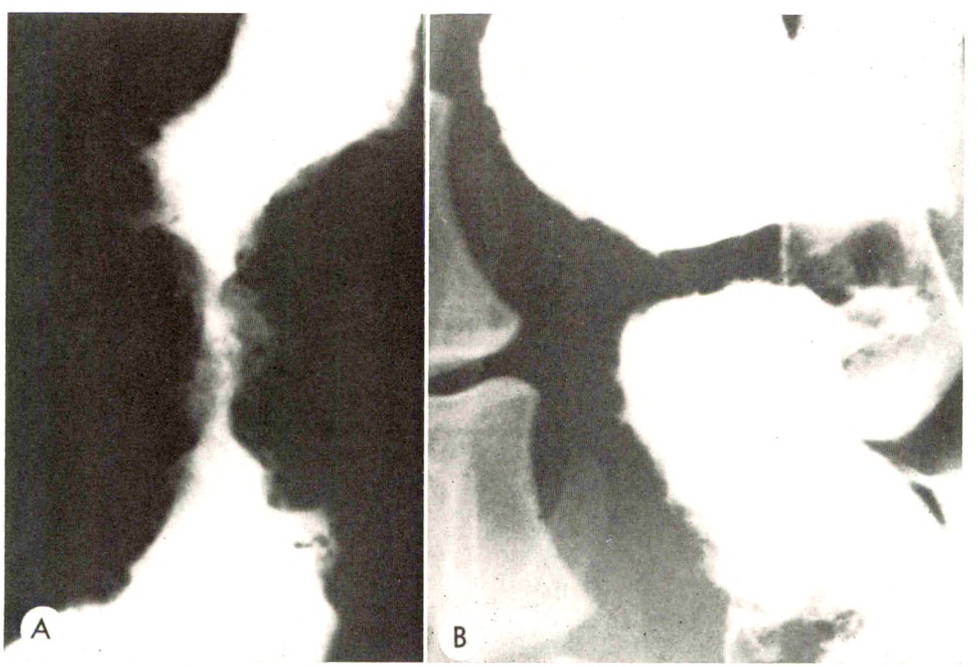


FIG. 5. Detailed magnification of 2 colons shows deep ulcers (*A*) and "collar button" ulcers (*B*). These were infrequent, but were associated with severe disease.

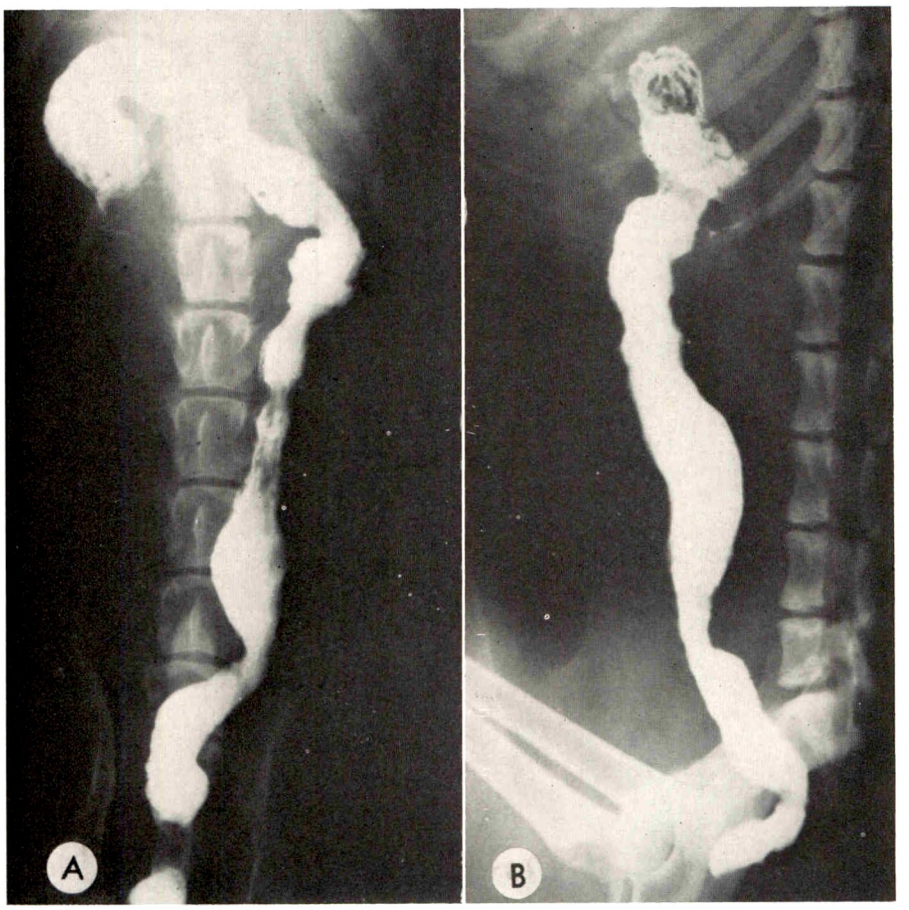


Fig. 6. (*A* and *B*) Severe colitis. The colon is markedly contracted and irregular, and would not distend. The cecum is not filled.

the distal colon and rectum usually indicated more severe disease than did the roentgenograms.

Histologic review of the colons of 24 dogs showed a spectrum of disease. The dominant microscopic feature of the boxer colon was aggregates of PAS-positive macrophages in the mucosa and submucosa. The mucosal histiocytes were frequently concentrated in a zone of even width between the muscularis mucosae and the bases of the crypts; they rarely infiltrated the muscularis mucosae but were found in less even concentrations immediately beneath it. Acute inflammatory cells were seen around the base of the mucosal ulcerations. Occasional submucosal abscesses were present below the ulcers. Crypt abscesses and hyperplastic mucosa were not seen. Ulceration varied, ranging from small, superficial defects to completely denuded colonic segments. Deep penetration was rarely seen and the disease was never transmural. Colonic lymphoid follicles were spared by the histiocytic infiltration and granulomas were not present. Mesenteric lymph nodes contained masses of large PAS-positive histiocytes; these were concentrated in cortical and medullary sinuses and usually spared the follicles.

Mild colitis was seen in both the boxer and non-boxer breeds, but severe colitis only in the boxers. In the colon specimens of the non-boxers, nonspecific inflamma-

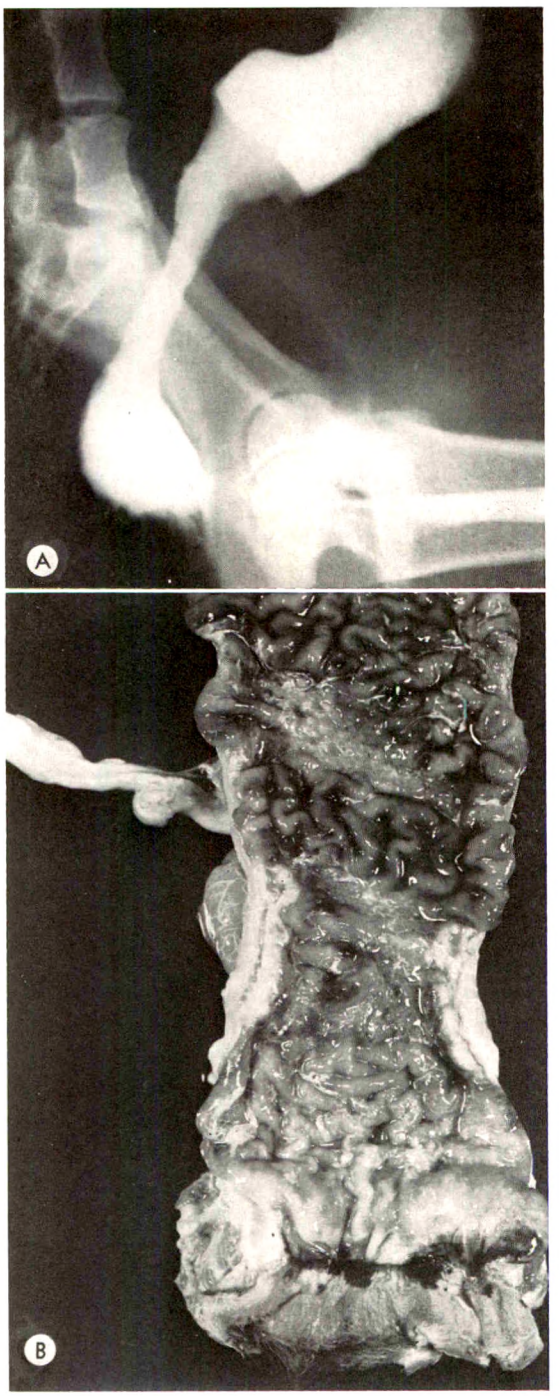


FIG. 7. (*A*) A localized stricture in the descending colon and rectum. Irregularities of contour in the narrow segment represent islands of intact mucosa. (*B*) Photograph of the gross necropsy specimen of *A* shows the stricture, mucosal islands, skipped areas with large denuded areas of mucosa, and the thickened, irregular mucosal folds.

tory changes with few ulcerations were noted. PAS-positive histiocytes were seen only occasionally.

DISCUSSION

The clinical and histologic changes in canine ulcerative colitis have been described by others.[1–3,5–8] The entity of histiocytic ulcerative colitis appears to be confined to the boxer breed. Electron microscopy, histochemical study, and routine bacteriologic methods have failed to identify a specific etiologic agent, although an infectious organism is suspected.[5–8]

Gross specimens were not available for comparison with roentgenograms. The fine serrations seen along the contour of the colon were presumed to represent small ulcers. These were present in some dogs in which the ulcers were seen on proctoscopic and histologic examinations; in others, however, such serrations were not observed on barium enema examination. The serrations were not seen in dogs without ulcers. It is not known to what degree edema produced the derangement of the mucosal folds, but, presumably, the histiocytic infiltrates create much of the thickening and irregularity.

Contraction, with shortening and narrowing, was a striking roentgenographic feature and was apparent in some colons in which mucosal abnormality was minimal. It was the best indicator of the extent of involvement. Diffuse fibrosis was not a pronounced histologic feature but was present in the areas of stricture formation.

On roentgenograms, canine histiocytic ulcerative colitis appears most like the idiopathic chronic ulcerative colitis of man. In the dog, the lesions are most common in the rectum and distal descending colon and consist of derangement of the mucosal folds and mucosal serrations. Usually the lesions are in continuity but skip areas may be present. The atonic, air-filled colon as seen on plain roentgenograms, and the shortening and narrowing of the colon are also seen frequently in human idiopathic chronic ulcerative colitis.

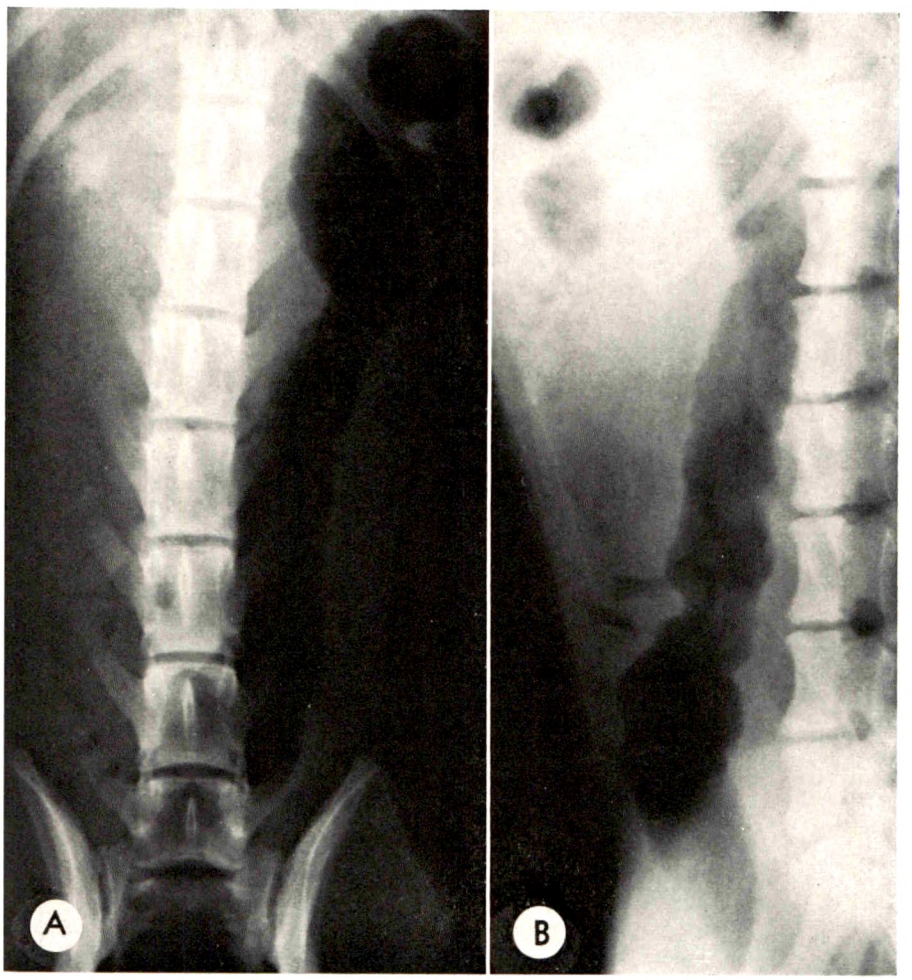


Fig. 8. (*A* and *B*) Roentgenograms of an atonic air-filled colon as was seen in 4 severely diseased dogs.

Canine histiocytic ulcerative colitis has been compared to Whipple's disease of man because of the similar PAS-positive histiocytes.[2,3,5,7,8] Histologically, the canine disease is not similar to either idiopathic ulcerative colitis or granulomatous colitis (Crohn's disease). In canine histiocytic ulcerative colitis, no crypt abscesses, fistulas, or granulomas are present. The lamina propria and submucosa are infiltrated with histiocytes and the mucosa is secondarily ulcerated.[7] The colon is thick but the muscularis and serosa are not affected. Regional lymph nodes are hyperplastic in severe disease and are infiltrated by the histiocytes.

The roentgenographic similarities of canine histiocytic ulcerative colitis and idiopathic ulcerative colitis in man warrant further study. Despite histologic differences, basic disease processes may be similar. Histologic identification of specific abnormalities noted on barium examination is also of interest to the roentgenologist. Work on these aspects is currently in progress.

SUMMARY

Barium enema examinations and histologic specimens were evaluated in a retrospective study of 24 cases of canine ulcerative colitis.

Mucosal serrations, ulcerations, and irregular mucosal folds, with shortening and

rigidity of the colon were the most frequent roentgenographic changes in these cases.

Histologically, the dominant feature was the PAS-positive histiocyte infiltration of the colonic mucosa and submucosa. Variable ulceration was present, ranging from small superficial defects, to completely denuded segments of colon.

Canine histiocytic ulcerative colitis shares many roentgenographic features with idiopathic chronic ulcerative colitis of man, despite histologic differences.

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ANGIOGRAPHIC STUDIES IN CROHN'S DISEASE AND ULCERATIVE COLITIS*

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CROHN's disease and ulcerative colitis are 2 diseases whose etiology is unknown. It is sometimes difficult to differentiate them pathologico-anatomically or even roentgenologically, since they often show a very similar appearance and localization in the colon. Differential diagnosis between these 2 diseases is, however, of great therapeutic and prognostic importance.

Both Crohn's disease[5] and ulcerative colitis[10] have been observed in an increasing frequency during the last decade. This may be due partly to greater diagnostic experience and interest in these diseases. In barium contrast examinations of the digestive tract, especially of the colon, extensive changes may completely escape discovery or may be misinterpreted, and increased surgical activity has revealed many cases of so-called ulcerative colitis as being Crohn's disease.

During recent years the intestinal blood flow in different pathologic conditions has been subject to investigation. For technical reasons it has been difficult to obtain information on local blood flow changes with normal variations in the different activities of the intestine, such as absorption, secretion and motility, although it seems probable that the blood flow can exhibit changes in these connections. Bacaner[1] found, using isotope methods (P^{32}) in man, a greatly increased blood flow in the colon in ulcerative colitis. He considered that only a minor part of this was nutritive, while most passed through the intestine as an arteriovenous shunt flow. He assumed that this might be due to instability of the vasomotor system, possibly characteristic of ulcerative colitis. In one case of regional enteritis without involvement of the colon he observed a reduced blood flow in the colon compared with presumed normal values. His material is small and allows no definite conclusions to be drawn, however.

Lunderquist and Lunderquist[8] and Lunderquist, Lunderquist and Knutson[9] reported a study comprising 10 patients with ulcerative colitis and 14 with Crohn's disease. In ulcerative colitis they found a relatively high frequency of wide arteries, including both the main branches and vasa recta, without a normal diminution in caliber; *i.e.*, with abrupt terminations as in occlusions, and also small, narrow, irregular vessels peripherally. In the capillary phase there was a dense accumulation of contrast medium in a thick intestinal wall and early, pronounced contrast medium filling of veins in both the intestinal wall and mesentery. In Crohn's disease they observed, in the region of the superior mesenteric artery, wide afferent arteries with sudden reductions in caliber in the mesentery, and right-angled bifurcations and constrictions in the intestine. In the capillary phase there was dense filling with contrast medium and a profuse retrograde venous flow. In other cases, however, they found normal-sized or constricted arteries, no dense contrast medium filling in the intestinal wall and a hardly discernible venous phase. They considered that these findings might represent different stages of the same disease. In 7 cases of ulcerative colitis, Boijsen and Reuter[2] found increased vascularization with regular, peripherally dilated vasa recta and wide, tortuous efferent veins. In 11 cases of Crohn's disease they also found increased vascularization with slight dilatation of arteries and veins and signs of a rapid

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TABLE I

THE MATERIAL INVESTIGATED
(32 cases)

Diagnosis	No.	Sex	Mean Age (yr.)	Range (yr.)
Crohn's Disease	17	male	27	16–44
	9	female	26	13–44
Ulcerative Colitis	4	male	37	24–53
	2	female	36	33–38

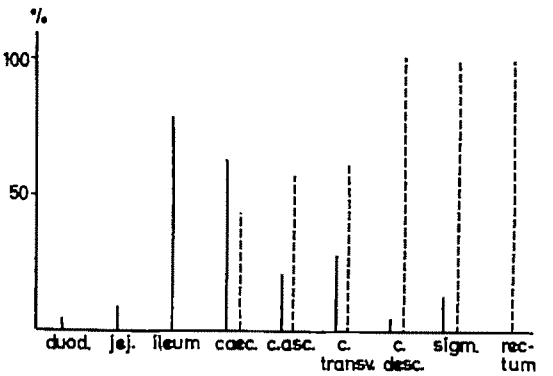


FIG. 1. The frequencies of the localization in different parts of the digestive tract for Crohn's disease (——) and ulcerative colitis (- - - -) are shown.

blood flow. The vasa recta were slightly irregular, some with an abrupt decrease in caliber, and similar changes were found in both the small intestine and colon. He considered that the filling of the veins with contrast medium was proportional to the activity of the disease.

Brahme,[8] in a small material of 5 cases of Crohn's disease, found hypovascularization with a hardly discernible venous phase in the small intestine. In the colon the vasa recta were tortuous and irregular, but otherwise the conditions were normal. Bradykinin produced essentially no change in the vascular pattern but an increase in the blood flow. In healthy areas the capillaries showed more dense filling with contrast medium, compared with the diseased areas.

The series of patients with these two diseases are generally small and the findings partly contradictory. We decided, therefore, to present our own series, which, as regards Crohn's disease, is larger than those reported previously in the literature. Dur-

TABLE II

VASCULAR CHARACTERISTICS ARE GIVEN FOR CROHN'S DISEASE AND ULCERATIVE COLITIS IN SUPERIOR AND INFERIOR MESENTERIC ARTERIES (MEAN VALUES AND RANGE)

Diagnosis	No.	Duration of Disease (yr.)	Vascular Characteristics			
			Superior Mesenteric Artery			
			shunt 0 1 2	venous filling 0 1 2	vascular richness 0 1 2 3	tortuosity 0 1 2
Crohn's Disease	22	2 (0.1–11)	0 (0–1)	1.3 (0–2)	2 (1–3)	1 (0–2)
Ulcerative Colitis	6	7.5 (2–15)	0.2 (0–1)	1 (0–2)	2 (0–3)	1.3 (0–2)
			Inferior Mesenteric Artery			
Crohn's Disease	12	3.5 (0.2–11)	1 (0–2)	1.8 (0–2)	2 (0–3)	1.5 (0–2)
Ulcerative Colitis	6	7 (2–15)	1.5 (0–2)	2 (2–2)	3 (3–3)	2 (1–2)

ing recent years we have performed abdominal angiography almost consistently in the preoperative investigation of Crohn's disease and ulcerative colitis, in addition to the conventional intestinal investigations with contrast medium. In several cases angiography has been performed postoperatively on the excised intestinal segment, and microangiographic studies are in progress.

MATERIAL AND METHOD

The series is comprised of 32 patients, and its composition can be seen in Table 1. All patients underwent operation and the diagnosis of Crohn's disease was verified pathologico-anatomically. For this pathologic diagnosis the criteria of Lockhard-Mummery and Morson[6] were used. In order to differentiate ulcerative colitis from Crohn's disease in the colon, it is also necessary in some individual cases to take into account the localization of the lesions (skip lesions) as well as the entire clinical picture.[4,7]

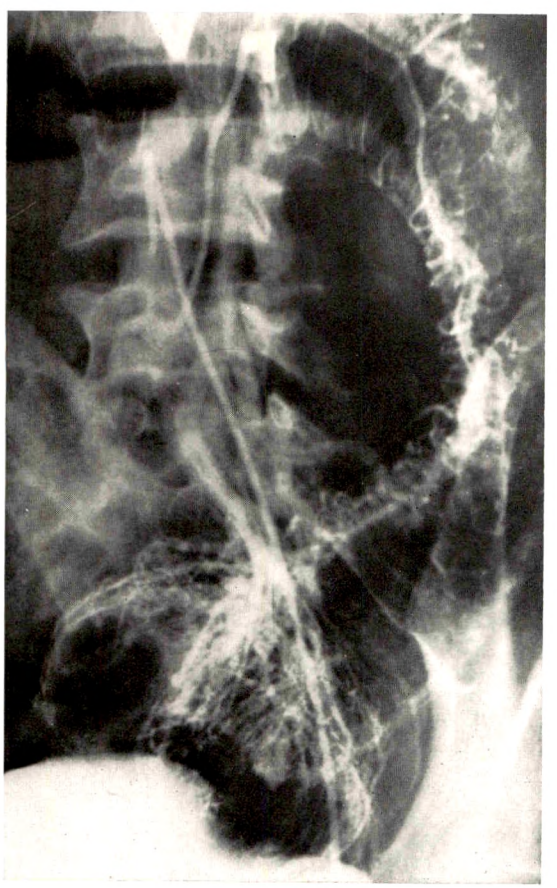


FIG. 2. A 25 year old man with ulcerative colitis for the past 2 years. Angiography of the inferior mesenteric artery shows hypervascularity, shunts and dense contrast medium filling of the veins.

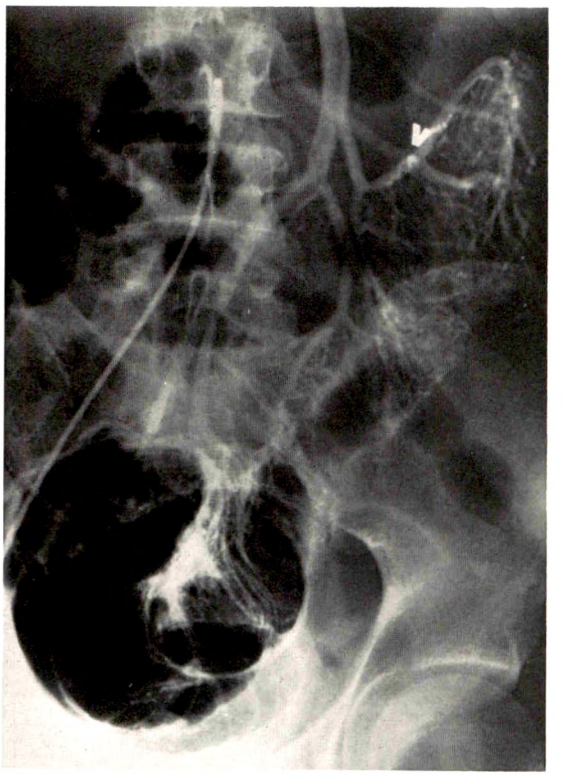


FIG. 3. A 25 year old man with Crohn's disease for the past 3 years. Angiography of the inferior mesenteric artery shows hypervascularity and dense contrast medium filling of the veins.

In 10 of our patients angiographic studies on specimens of the excised intestinal segment were performed postoperatively.

Abdominal angiography was done by the Seldinger technique, from the femoral artery, selectively in both the superior and inferior mesenteric artery in 16 cases and in 1 of these arteries in the remaining 16 cases, depending upon the localization of the lesions. Gray and red Ödman catheters were used in the superior and inferior mesenteric arteries, respectively, and 10–20 ml. of urografin 45 per cent or 60 per cent was injected during 0.7–1 seconds.

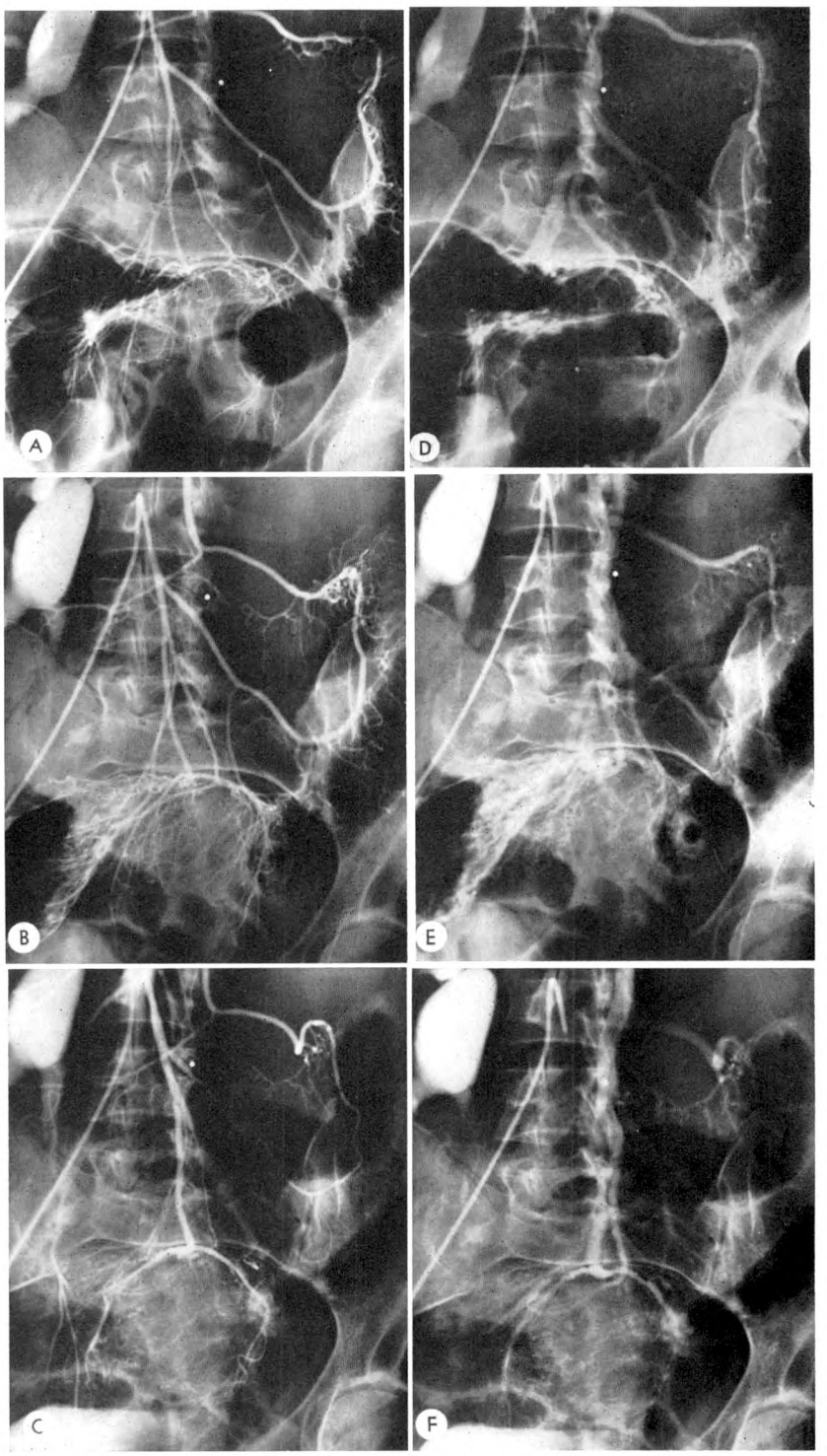


FIG. 4. A 32 year old woman with ulcerative colitis for the past 2 years. Angiography of the inferior mesenteric artery. (*A* and *D*) Arterial and venous phases without any preceding intraarterial injections of vasoactive drugs. (*B* and *E*) Effects of 45 μg. bradykinin, *i.e.*, vasodilatation and increased visualization of the veins. (*C* and *F*) Effects of 0.3 I.U. vasopressin, *i.e.*, vasoconstriction and reflux of the contrast medium into the aorta as a result of increased peripheral vascular resistance.

Exposures were made according to the following program: 3 exposures/sec. for 3 seconds; 1 exposure/sec. for 4 seconds; 1 exposure every alternate second for 10 seconds. The injections and exposures were recorded with a direct-writing recorder. In 15 patients the selective angiography was preceded by an intraarterial injection of approximately 50 μg. of bradykinin*; this was injected in about 5 minutes at a dose rate of approximately 10 μg./min. In 4 patients selective arteriography was preceded by a slow intraarterial injection of 0.2–0.4 international units of vasopressin.* The arterial diameters were measured on the roentgenograms with the aid of a magnifying glass (with an accuracy of 0.1 mm.).

In the postoperative studies of the intestinal specimens, micropaque was injected into the arteries and a roentgenologic examination was then performed with the specimens in close contact with the film.

RESULTS

The percentage distribution of the localization of the lesions in the whole series can be seen in Figure 1. Some lesions in the small intestine in the ulcerative colitis patients are not represented, while in 6 cases of Crohn's disease, lesions were only found in the colon and in 3 cases only in the small intestine.

The angiographic results are presented in Table II. Consideration has been taken of those parameters which we were able to evaluate, *viz.*, shunt, degree of retrograde venous filling, vascularity and tortuosity of the vessels; no account has been taken of caliber changes or type of ramification. A numerical code has been used and the mean values and ranges given. A shunt was considered to be present if arterial and venous filling were observed simultaneously; the absence of a shunt is denoted by 0, and a pronounced shunt by 2. Corresponding gradations have been used for the other parameters.

The angiographic pattern in the supply area of the superior mesenteric artery was similar in the 2 diseases, both as regards mean values and ranges. Cases with hypo- or hypervascularization were found in both groups, as well as both straight and tortuous vessels; with some predominance, however, of straight vessels in the small

* Bradykinin and vasopressin were kindly supplied by Sandez AG, Basel.

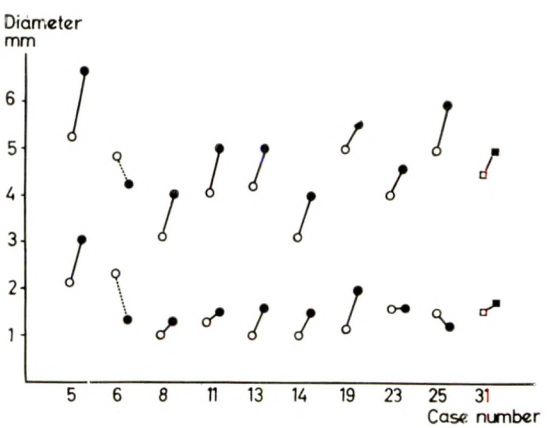


Fig. 5. The effects of bradykinin —— and vasopressin - - - - upon the diameters of a central branch (upper symbols) and of a peripheral branch (lower symbols) of superior mesenteric artery. Crohn's disease: ○ before and ● after the injection. Ulcerative colitis: □ before and ■ after the injection.

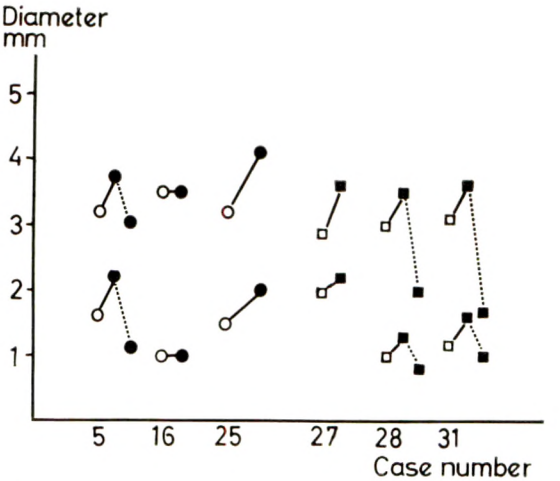


Fig. 6. The effects of bradykinin —— and vasopressin - - - - upon the diameters of a central branch (upper symbols) and of a peripheral branch (lower symbols) of inferior mesenteric artery. Crohn's disease: ○ before and ● after the injection. Ulcerative colitis: □ before and ■ after the injection.

intestine and tortuous vessels in the colon. Also, in the area of the inferior mesenteric artery the findings in Crohn's disease and ulcerative colitis were similar, but the mean values and ranges in the colitis group suggested higher grades of shunts, venous filling and hypervascularization than in Crohn's disease (Fig. 2; and 3).

The diameters of the central and peripheral arteries increased after injection of bradykinin and decreased after injection of vasopressin (Fig. 4, *A–F*, 5; and 6), with essentially the same type of reaction in healthy as in diseased parts of the intestine. The decrease or increase in the peripheral resistance which occurred resulted in a redistribution of the contrast medium in some cases.

The angiograms from intestinal specimens removed at operation were compared with preoperative angiograms (Table III; and Fig. 7, *A* and *B*). The greater vascularity and increased tortuosity observed in the intestinal specimens are certainly an expression of more complete vascular filling and more favorable conditions of reproduction, which allow a good evaluation of the blood vessels. No comparison between the 2 groups of patients can be made in this respect, however, since only 1 case of ulcerative colitis is represented. No typical changes such as decreases in caliber, luminal variations or abnormal branching, as have been described by other authors, were observed.

TABLE III

A COMPARISON OF THE PREOPERATIVE ANGIOGRAMS AND THE ANGIOGRAMS OF THE SPECIMENS OF THE SAME CASES

	Vascular Characteristics of Preoperative Arteriograms		
	No.	Vascular Richness	Tortuosity
Crohn's Disease	9	2.2 (2–3)	1.2 (0–2)
Ulcerative Colitis	1	3	1
	of Specimens		
Crohn's Disease	9	2.7 (2–3)	2 (2–2)
Ulcerative Colitis	1	3	2

DISCUSSION

Angiographic studies of the intestine comprise a complement to the conventional barium contrast investigations; they cannot replace them. The findings in the 2

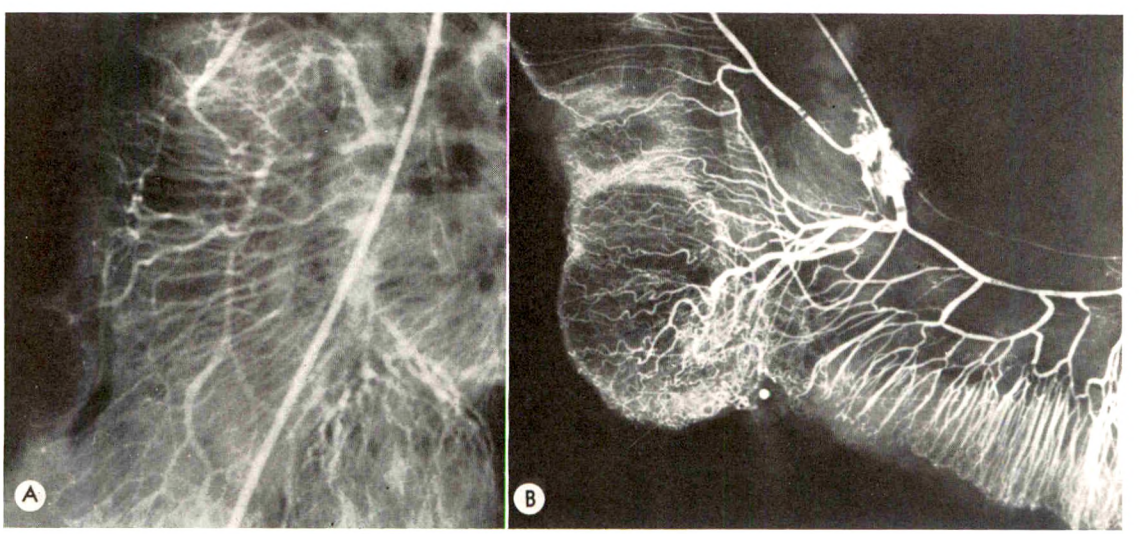


FIG. 7. An 18 year old man with Crohn's disease for the past 4 years. (*A*) The preoperative angiogram of the superior mesenteric artery shows hypervascularity. (*B*) Ileocecal specimen after injection of micropaque also shows a high degree of vascularization.

types of investigation correspond well in the small intestine but less in the colon. In the present series, the barium enema examination showed normal conditions in 2 patients in whom pronounced vascular changes were seen, while in 2 other patients the opposite was found. In the latter patients the lesions were localized to the transverse colon where experience has shown the vascular filling with contrast medium to be poorer and where, in addition, overlying vessels of the small intestine render evaluation difficult. The angiograms can provide the surgeon with important practical information, for example, concerning the left flexure of the colon which is difficult to inspect.

The results show that in both Crohn's disease and ulcerative colitis there is hypervascularization and an increased frequency of shunts in the intestine. These roentgenologic findings comprise the anatomic basis of a probably increased blood flow in the affected parts of the intestine.[1] However, there are also cases with relatively sparse vascularization, especially in Crohn's disease. A definitive angiographic differential diagnosis between Crohn's disease and ulcerative colitis is therefore not possible (Fig. 8, *A* and *B*).

The angiograms preceded by injection of bradykinin and vasopressin show that the large efferent arteries and their peripheral branches located in the diseased parts of the intestine react relatively normally to both these drugs. Their patterns of reaction cannot increase the possibilities of differential diagnosis, but are of physiologic interest in these vascular regions that are relatively difficult to investigate. In some cases, especially of ulcerative colitis, it was noted how a very abundantly vascularized part of the sigmoid colon received its arterial supply from both the superior and inferior mesenteric arteries. A type of "visceral steal syndrome" may possibly exist here. A profuse blood flow in one part of the intestine may conceivably mean a reduced blood flow in other parts. In 1 patient with lesions of Crohn's disease in the small intestine, Bacaner[1] observed a considerably lower blood flow in the colon than normal.

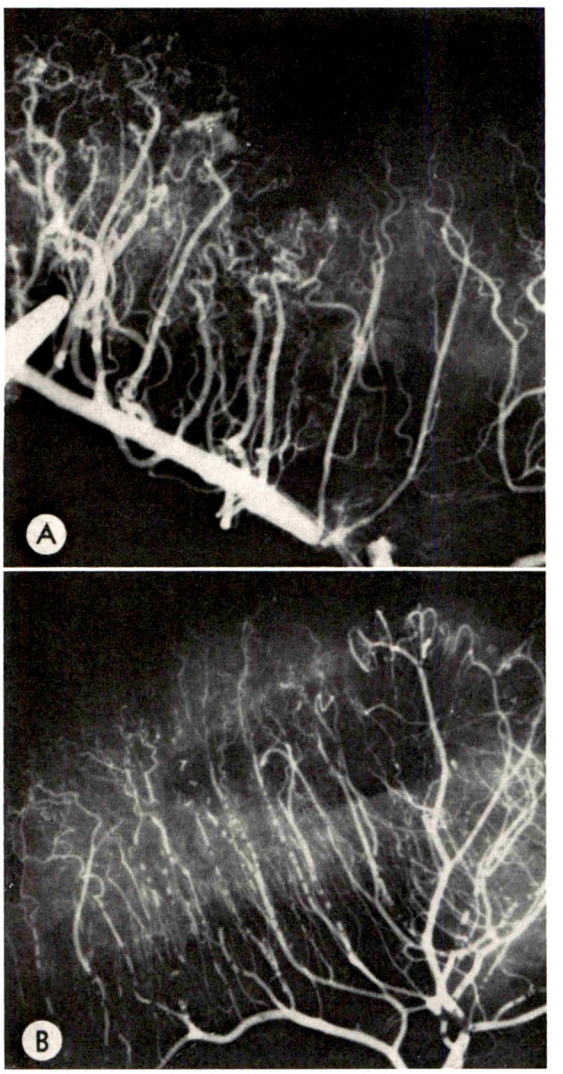


Fig. 8. (*A*) A 23 year old man with Crohn's disease for the past year. A colectomy was performed. Specimen from the transverse colon shows hypervascularity and some tortuous arteries. (*B*) A 38 year old woman with ulcerative colitis for the past 14 years. A colectomy was performed. Specimen from the descending colon also shows hypervascularity and tortuous arteries. Angiograms *A* and *B* show similar patterns.

The discrepancy between the results of different studies may be due to the fact that both diseases are characterized by pathologico-anatomically, roentgenologically and clinically very different stages; this would seem to explain the varying angiographic patterns.

The angiographic changes seen in these 2 diseases are possibly expressions of secondary events, rather than of the primary bases which are yet unknown. Abdominal angiography can, however, provide information on the normal and pathologic anatomy of the intestine, and to some extent its physiology. The microangiographic studies which we have started are of great interest.

SUMMARY

Twenty-one patients with Crohn's disease and 11 patients with ulcerative colitis were examined angiographically before operation, in some cases after an intra-arterial injection of bradykinin or vasopressin.

In addition, in 10 cases (9 Crohn's disease and 1 ulcerative colitis) angiographies were performed on intestinal specimens postoperatively.

The results show great angiographic similarities between the 2 diseases, and angiography thus allows no conclusive differential diagnosis.

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THE ANGLE PRONE PROJECTION: ITS VALUE IN DIAGNOSIS OF LOW-LYING LESIONS*

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INVESTIGATION of rectal bleeding should always include proctosigmoidoscopy. Aside from the obvious advantage of direct viewing of the mucosal surface, it affords investigation of the normally redundant rectosigmoid area—a roentgenologic blind spot which may be difficult to define by conventional barium and air contrast enema examinations.

The purpose of this communication is to advocate the use of a simple, comfortable projection of the rectosigmoid area which has proved valuable in delineating lesions of the rectosigmoid which may have been overlooked.

METHOD

Following the usual examination of the colon by barium enema, including image-intensification fluoroscopy and spot filming of the flexures and redundant portions, anteroposterior and posteroanterior overhead roentgenograms utilizing high kilovoltage techniques are obtained. The patient is left in the posteroanterior position and the central ray directed 35° to 45° caudad, emerging at the level of the anterior superior iliac spine (Fig. 1). Target-to-film distance is 45 inches. For a 25 cm. patient, the technical factors are 200 mas. at 110 kvp. for the barium-filled colon and 90 mas. and 85 kvp. for the air-filled colon. These factors are used with a 60 line, 12:1 grid; DuPont Par Speed Screens; and Cronex IV film, processed in a Kodak M4B X-Omat, converted for 90 second processing in Hunt 90–90 chemistry.

If the patient is unable to assume the prone position, similar results may be obtained with the patient supine, central ray directed 35° to 45° cephalad and entering at the level of the anterior superior iliac spine. Either of these projections will demonstrate the rectosigmoid and sigmoid segments relatively free of superimposed colon.

REPORT OF CASES

Case 1 (Fig. 2). B.A., 082963, a 60 year old white male developed sharp, lancinating pain in the epigastrium and right upper quadrant. He had noted increasing constipation for the previous 4 to 5 weeks with dark red rectal bleeding. Rectal examination disclosed a hard friable nontender mass which bled easily. Sigmoidoscopy revealed a friable hard raised lesion at 11 cm. which on biopsy proved to be adenocarcinoma of the rectum. The liver scan demonstrated a filling defect in the liver which at surgery was found to be a metastatic lesion.

Comment. This lesion was initially discovered by the attending physician. Barium and air contrast enema examinations failed to demonstrate the lesion. A postevacuation roentgenogram suggested the presence of tumor. The tumor shelf is clearly shown in the angle prone projection.

Case 11 (Fig. 3; and 4). O.H., 067813, a 78 year old white male was admitted with a diagnosis of acute epididymitis as a late complication of a transurethral resection performed 4 weeks previously. He complained of rectal bleeding. Sigmoidoscopy revealed a rectal tumor which on biopsy proved to be a poorly differentiated adenocarcinoma.

Comment. This is another case of a low-lying tumor discovered by the attending physician and demonstrated subsequently by barium enema examination. The lesion is demonstrated in its entirety in the angled prone projection.

Case 111 (Fig. 5, *A* and *B*; and 6). E.M., 056773, a 51 year old white male with a several month history of rectal bleeding, constipation, and decrease in stool diameter. The patient was referred for barium enema examination which disclosed a lesion in the rectum. Sub-

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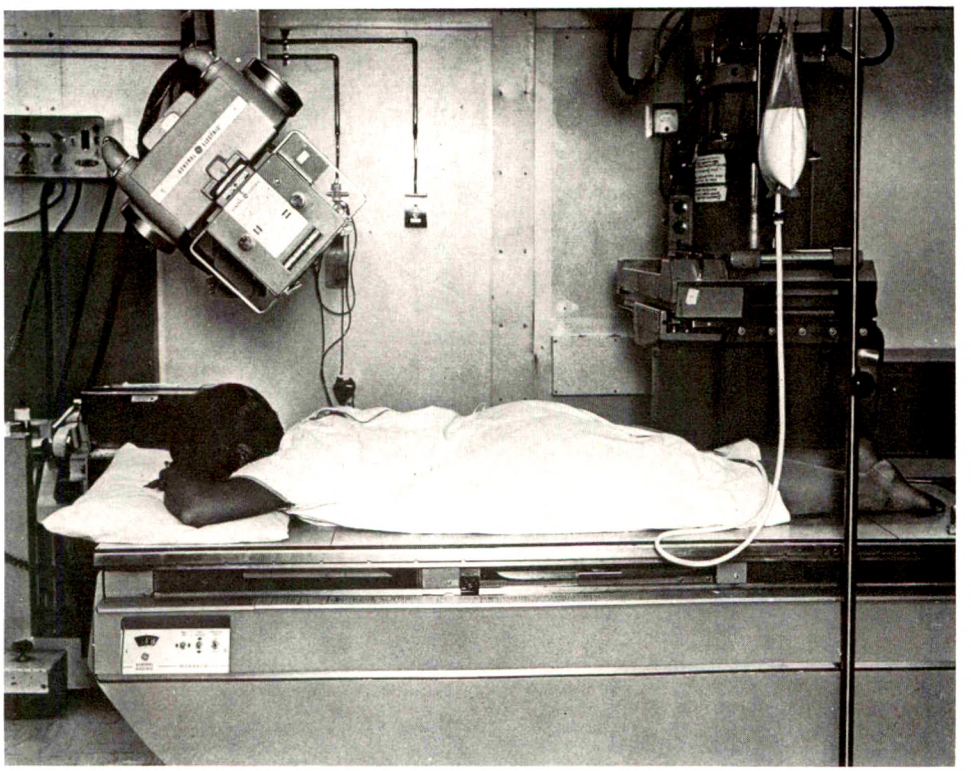


Fig. 1. Patient in prone position for angle projection of the rectosigmoid. Note 45° caudad angle of x-ray tube. The enema tip remains in place.

sequent sigmoidoscopy demonstrated a large fungating lesion at 10 cm., biopsy of which revealed a well differentiated infiltrating adenocarcinoma.

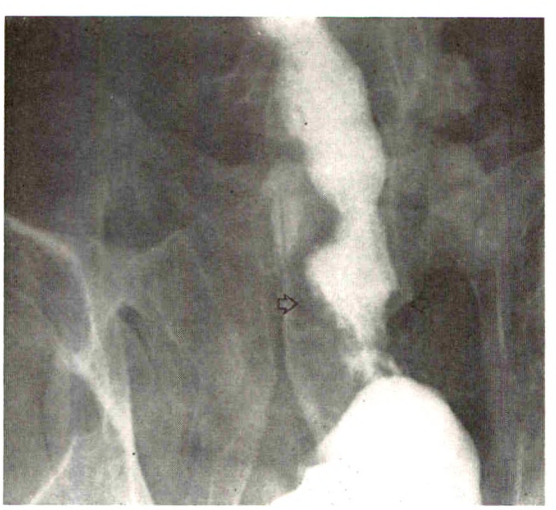


Fig. 2. Case 1. Roentgenographic detail in angle prone projection which demonstrates tumor shelf from an annular carcinoma of the rectum. This lesion did not show in the conventional projections.

Comment. This patient was first examined by the radiologist, who discovered the lesion. Subsequent endoscopy confirmed its presence and furnished a histologic diagnosis.

Case IV (Fig. 7; 8; and 9). M.L., 061577, a 52 year old white female with a history of rectal bleeding for 4 months prior to admission. She

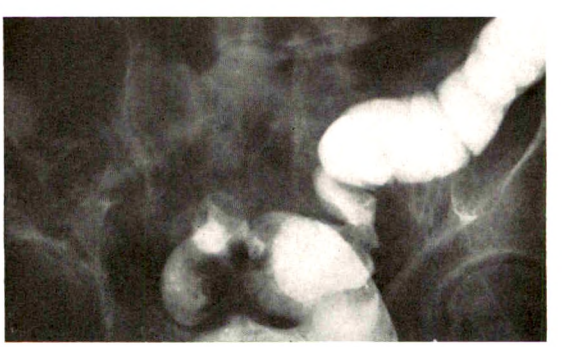


Fig. 3. Case II. Detail of anteroposterior roentgenogram of the colon. The tumor is hidden by the redundant rectosigmoid. A portion of the tumor was visible on 1 oblique spot roentgenogram of the sigmoid area.

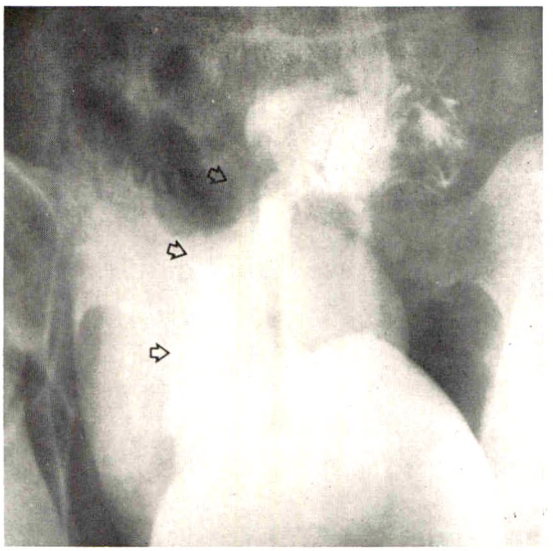


Fig. 4. Case II. Angle prone projection demonstrating a long, annular carcinoma involving the distal sigmoid colon.

noted mucus in her bowel movements, abdominal soreness, and occasional nausea and vomiting. She complained of alternating diarrhea and constipation. Barium enema examination initially was interpreted as showing probable polypoid lesions in a poorly cleansed colon and a re-study was suggested. Re-study 10 days later confirmed the polyps and demonstrated an annular lesion of the sigmoid colon.

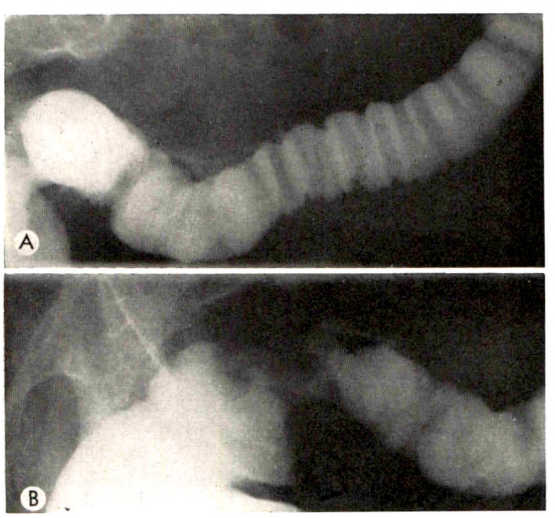


Fig. 5. Case III. (*A* and *B*) Spot roentgenograms of the rectosigmoid colon. The lower spot roentgenogram (*B*) is almost a lateral projection. The lesion is not visible in *A*.

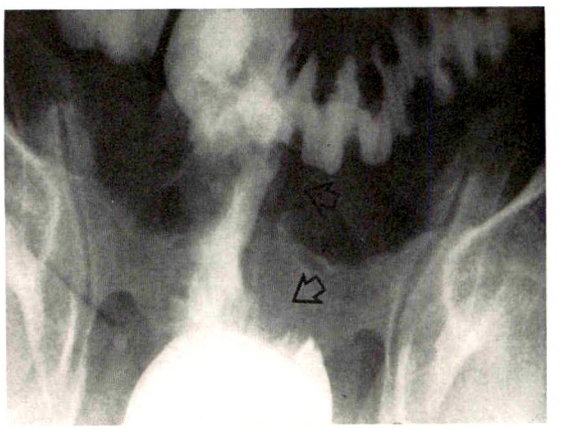


Fig. 6. Case III. The rectosigmoid carcinoma is clearly demonstrated in the angle prone projection. The lower tumor shelf is indicated by the lower arrow.

Surgery revealed adenocarcinoma, Dukes C, Grade 4.

Comment. This case, more dramatically than others, demonstrates the value of the angled prone projection. Both studies were carefully done, yet the lesion was not appreciated until clearly shown in the angled projection.

Case V (Fig. 10). D.B., 071427, a 26 year old Negro female, was admitted for sigmoidoscopy under anesthesia. Barium enema examination 1 week prior to admission showed poor prepara-

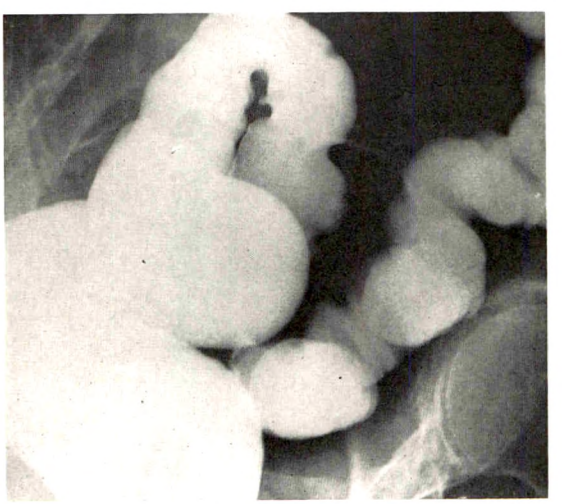


Fig. 7. Case IV. Oblique spot roentgenogram of the rectosigmoid colon. The tumor is hidden by the redundant rectosigmoid junction. Note the filling defect in the distal sigmoid.



Fig. 8. Case iv. Anteroposterior air contrast roentgenogram to confirm the presence of a small adenomatous polyp (arrow). The tumor is not visible in this projection.

tion with some irregularity of the sigmoid. The patient admitted to bloody diarrhea, containing mucus, and temperature elevations for the previous month. Laboratory studies showed anemia with a normal white blood cell count. Subsequent endoscopy initially did not reveal the lesion. Under anesthesia, however, a cauliflower lesion was seen and biopsied. This was reported as a villous adenoma. Subsequent surgery revealed the lesion actually to be adenocarcinoma arising in a villous adenoma. This was classified as Dukes C, Grade 2.

Comment. This lesion, initially confused with fecal material, is clearly seen as a neoplasm in the modified angle projection.

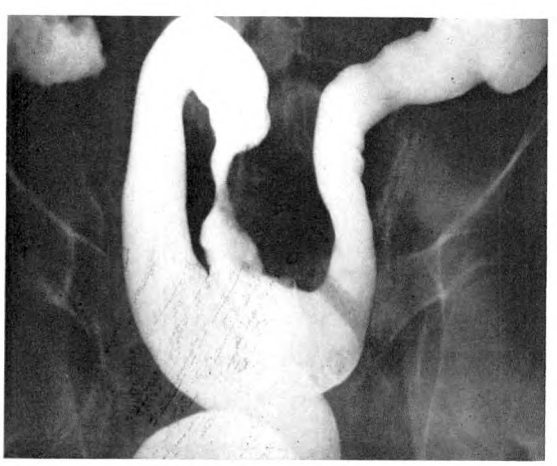


Fig. 9. Case iv. Angle prone projection demonstrating the carcinoma of the midsigmoid colon. The distal tumor shelf is clearly shown.



Fig. 10. Case v. Angle prone projection demonstrating irregularity and narrowing of the distal sigmoid colon. Barium trapped within the interstices of the lesion suggests villous adenoma. This lesion could have been confused with retained fecal material in the conventional projections. The lesion proved to be malignant.

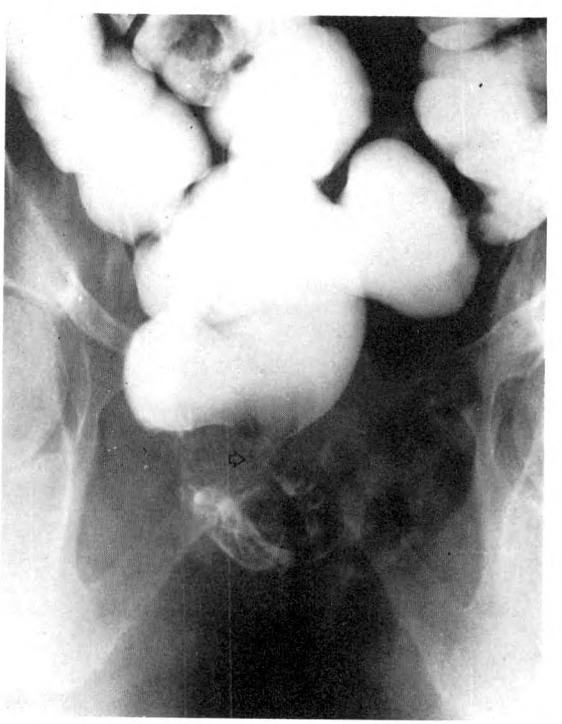


Fig. 11. Case vi. Barium-filled angle prone projection demonstrating an annular lesion of the rectum. The lesion was not identified in other projections, including spot roentgenograms.



Fig. 12. Case vi. Air contrast angle prone projection confirms the presence of an annular rectal lesion. No mucosal detail is seen.

Case vi (Fig. 11; and 12). D.J., 067891, a 42 year old white female with history of endometriosis and recent onset of bloating, "difficult" bowel movement, and ribbon-shaped stools. Barium enema examination revealed narrowing of the rectosigmoid. Sigmoidoscopy demonstrated a submucosal lesion at 14 cm. obstructing the colon. Biopsy showed benign lesion.

Surgery: Anterior resection of colon for obstructing mass.

Pathology: Diverticulitis with pericolonic abscess. Endometriosis.

Comment. This is another case first examined by barium enema. The lesion was suspected at fluoroscopy but demonstrated only in the angle projection.

DISCUSSION

Ettinger and Elkin[3] reported a comprehensive study of special roentgenographic views for evaluation of the sigmoid colon. They included the classical Chassard-Lapine projection, an angled oblique projection and an angled anteroposterior projection. These investigators concluded that the rectum was best evaluated in the lateral projection, that the rectosigmoid colon was best shown in the lateral or angled oblique projection, and that the iliac colon was best demonstrated in all views. The Chassard-Lapine projection was useful in demonstrating the proximal portion of the rectosigmoid and the pelvic sigmoid colon.

The Chassard-Lapine view, however, is difficult for both patient and technologist. The enema tip must be removed, the patient must be re-positioned in a sitting position and the technologist is forced to center the x-ray tube by working over the patient. Assuming the flexed position necessary for this projection is difficult for an elderly patient.

Dysart and Stewart[2] described a special angled projection which permitted simpler, faster and more comfortable positioning of the patient. Reviewing 5 years of barium enema studies, they stated that 81 of 314 lesions of the rectosigmoid were shown by this view and 34 of 35 proven carcinomas of the rectum were more clearly shown in the angled prone projection. The distance from the anal verge to the lesion could also be accurately assessed in this projection.

Dunbar and his co-workers[1] compared the efficacy of the horizontal axial view (or angled prone projection) and the Chassard-Lapine view and concluded that the 2 were equal in ability to separate redundant loops. In view of the relative ease of performing the horizontal axial view, this was the projection of choice.

Gardiner[4] recently reported his experience in a review of 2,500 consecutive routine lateral studies of the barium-filled rectum and concluded that only 1 lesion overlooked endoscopically was demonstrated by this projection. He also commented on the variability of the width of the presacral space and its controversial clinical significance.

It would therefore appear mandatory that all patients presenting with colon symptoms, including rectal bleeding, be subjected to endoscopic examination. Until this desirable situation is universally adopted, however, it remains incumbent upon the radiologist to investigate the rectosig-

moid area as thoroughly as possible before rendering a report of his barium enema study. The angle prone projection greatly simplifies this task and is a valuable addition to the routine projections used in studying the colon.

CONCLUSIONS

1. The routine use of an angled prone projection in evaluation of the colon is recommended. Its value is described and emphasized by the presentation of representative cases.

2. Similar results may be accomplished by the use of the Chassard-Lapine view, but this imposes difficulties on both patient and technologist.

3. This procedure, ideally, should be employed as adjunctive to endoscopy and is not recommended as a replacement to direct vision of the distal colon.

SUMMARY

Low-lying lesions of the rectum and sigmoid colon may be difficult to demonstrate, especially in the presence of redundant bowel. This becomes critical in cases referred to the radiologist for examination without previous endoscopy.

The use of a simple angle prone projection for evaluation of the rectosigmoid is recommended.

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UNRAVELLING THE TANGLED SIGMOID*

ROUTINE ANGULATION OF THE TUBE IN THE DETECTION OF OCCULT LESIONS

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THE detection of cancer of the sigmoid is a formidable challenge. Almost 50 per cent of colonic neoplasms above the rectum lie in its tortuous coils. To demonstrate the sigmoid completely, angulation of the roentgen-ray tube, cephalad when the patient is supine and caudad when the patient is prone, is necessary. These additional projections should, therefore, be a part of the routine barium enema examination.

ANATOMY

The anatomy of the sigmoid is variable. Usually, in its retrograde course, the sigmoid passes anteriorly from right to left in front of the rectum, then posteriorly as it ascends to its junction with the descending colon. Angulation of the roentgen-ray tube, cephalad when the patient is supine and caudad when the patient is prone, will direct the central ray across the long axis of that portion of the sigmoid which is directed anteroposteriorly.

REVIEW OF THE LITERATURE

The earliest reference to angulated projections of the colon in the radiographic literature was made by Billings[1] in 1944. He suggested a left posterior oblique (supine) projection of the sigmoid with a 35° superior angulation. In addition, the urinary bladder was filled with saline to elevate the sigmoid from the pelvis. Fletcher[5] reported this information in the American literature and suggested distending the bladder by having the patient drink large quantities of water before the examination, thus eliminating catheterization. Ettinger and Elkin[4] have suggested the use of a left or occasionally right posterior oblique projection angling cephalad or the Chassard-Lapine view to better demonstrate the sigmoid. They advised the use of this technique in extremely obese patients, in cases where demonstration of the sigmoid was unsatisfactory due to severe coiling, and in cases with previous negative examinations in which there was a strong suspicion of a lesion. They usually resorted to this technique on the second examination. Dysart and Stewart[3] reported on their 5 years' experience with a projection made with the patient prone, angling the tube caudad routinely in the postevacuation double contrast study and occasionally in the barium filled colon examination. All of these authors report the demonstration of lesions not demonstrable without angulation. Dunbar *et al.*[2] compared the Chassard-Lapine and the prone angulated projections and concluded that they were equal in the demonstration of the sigmoid but that the prone angulated projection was easier to obtain, particularly in an older patient.

TECHNIQUE

Cephalic Angulation. Barium is introduced slowly under fluoroscopic control with appropriate spot filming as far as the splenic flexure, at which point the instillation is interrupted. The patient is rotated into the optimum left posterior oblique position. The overhead tube is angled 30 to 40° cephalad (depending upon the degree of coiling) and the central ray is directed through a point just above the pubic

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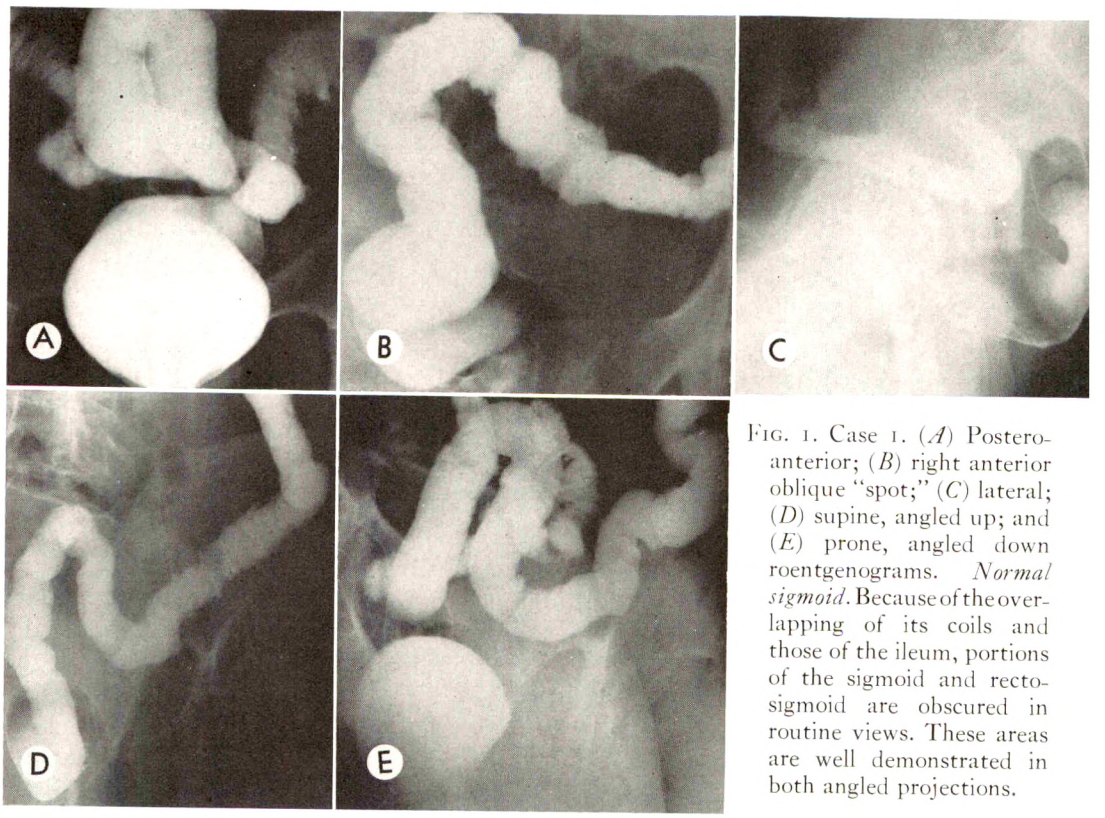


FIG. 1. Case 1. (*A*) Posteroanterior; (*B*) right anterior oblique "spot;" (*C*) lateral; (*D*) supine, angled up; and (*E*) prone, angled down roentgenograms. *Normal sigmoid.* Because of the overlapping of its coils and those of the ileum, portions of the sigmoid and rectosigmoid are obscured in routine views. These areas are well demonstrated in both angled projections.

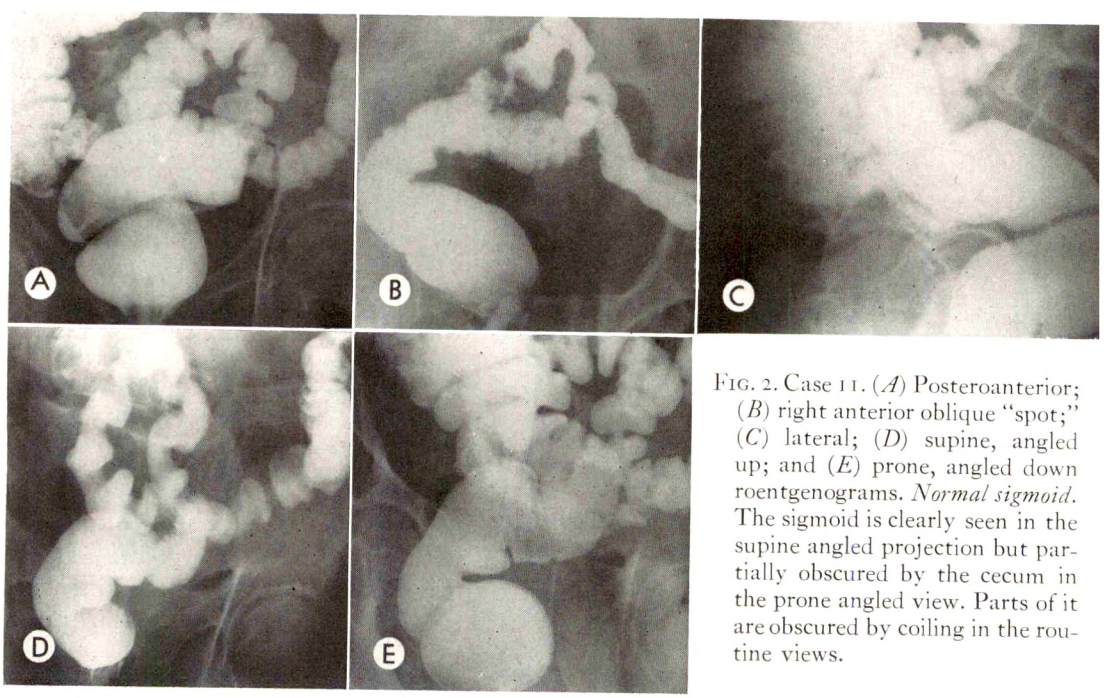


FIG. 2. Case 11. (*A*) Posteroanterior; (*B*) right anterior oblique "spot;" (*C*) lateral; (*D*) supine, angled up; and (*E*) prone, angled down roentgenograms. *Normal sigmoid.* The sigmoid is clearly seen in the supine angled projection but partially obscured by the cecum in the prone angled view. Parts of it are obscured by coiling in the routine views.

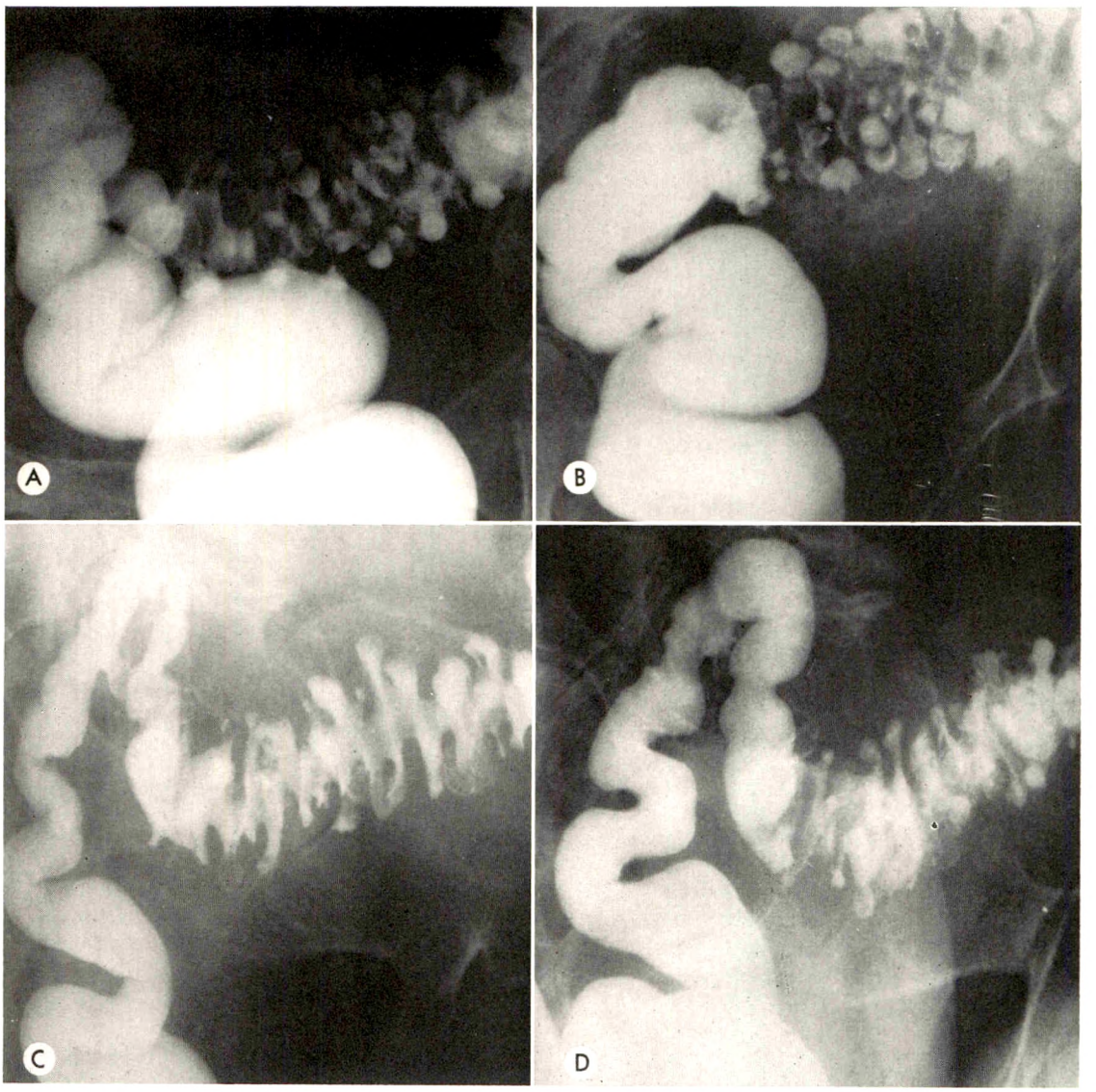


Fig. 3. Case III. (*A*) Posteroanterior; (*B*) right anterior oblique "spot;" (*C*) supine, angled up; and (*D*) prone, angled down roentgenograms. *Diverticulosis*. The angled projections give better visualization and permit the exclusion of diverticulitis.

symphysis to the center of an 11×14 inch film. A left lateral roentgenogram is also made at this time.

Caudad Angulation. Following the completion of the filling of the colon under fluoroscopy, a 14×17 inch posteroanterior roentgenogram is made. The patient is then rotated into the right anterior oblique position. With the tube angled 30 to 40° caudad (depending upon the degree of coiling) the central ray is directed through the superior margin of the left sacroiliac joint to the center of an 11×14 inch film.

ILLUSTRATIVE CASES

Cases I and II are normal studies with varying degrees of coiling of the sigmoid. In each case the sigmoid is completely demonstrated only when angled projections are added (Fig. 1, *A–E*; and 2, *A–E*).

Cases III through VII demonstrate pathology which can be better visualized in the

Fig. 5. Case v. (*A*) Posteroanterior; (*B*) lateral; (*C*) right anterior oblique "spot;" and (*D*) prone, angled down roentgenograms. *Annular carcinoma of the upper rectum.* The angled projection best demonstrates the extent of the lesion.

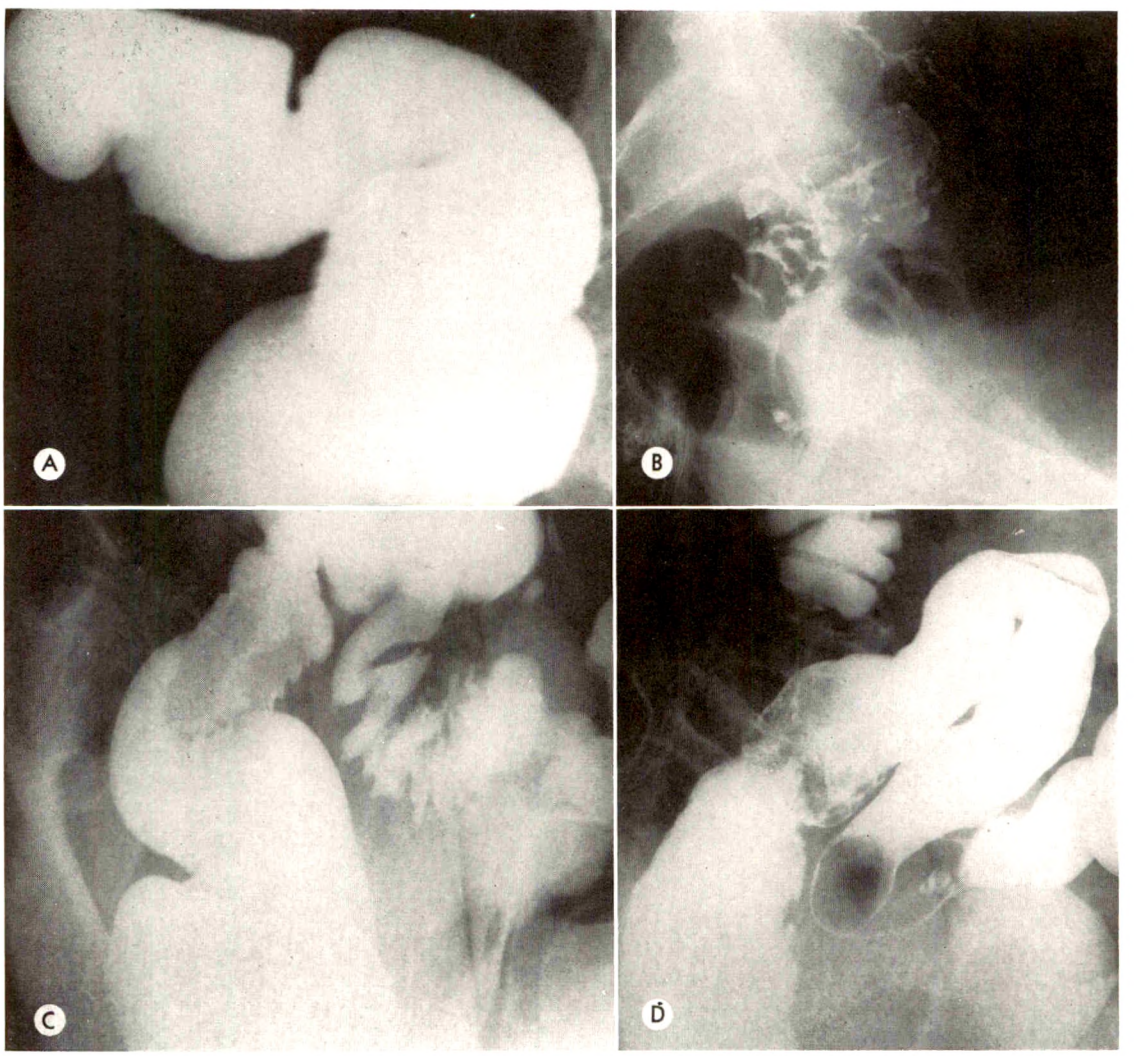
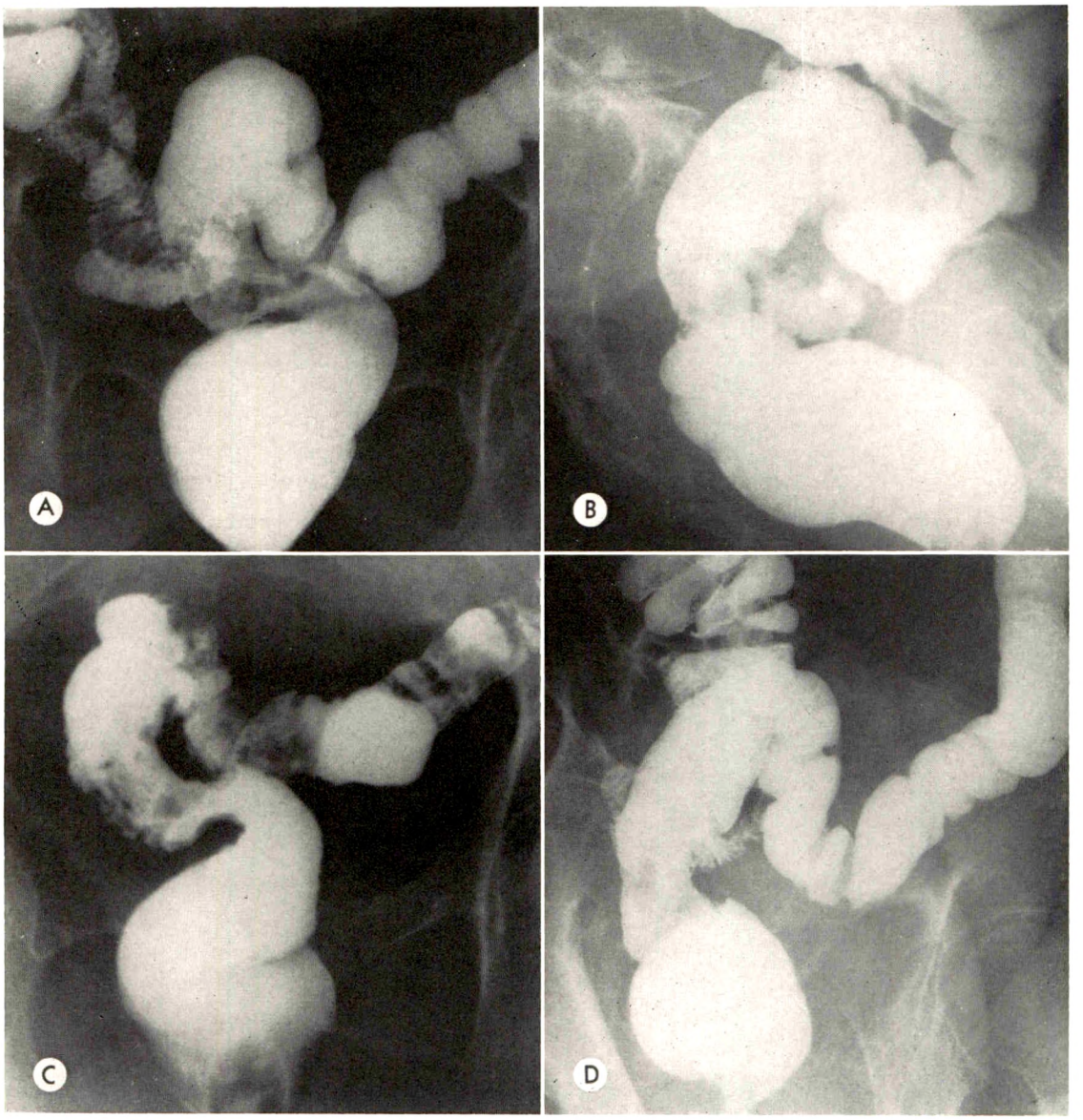
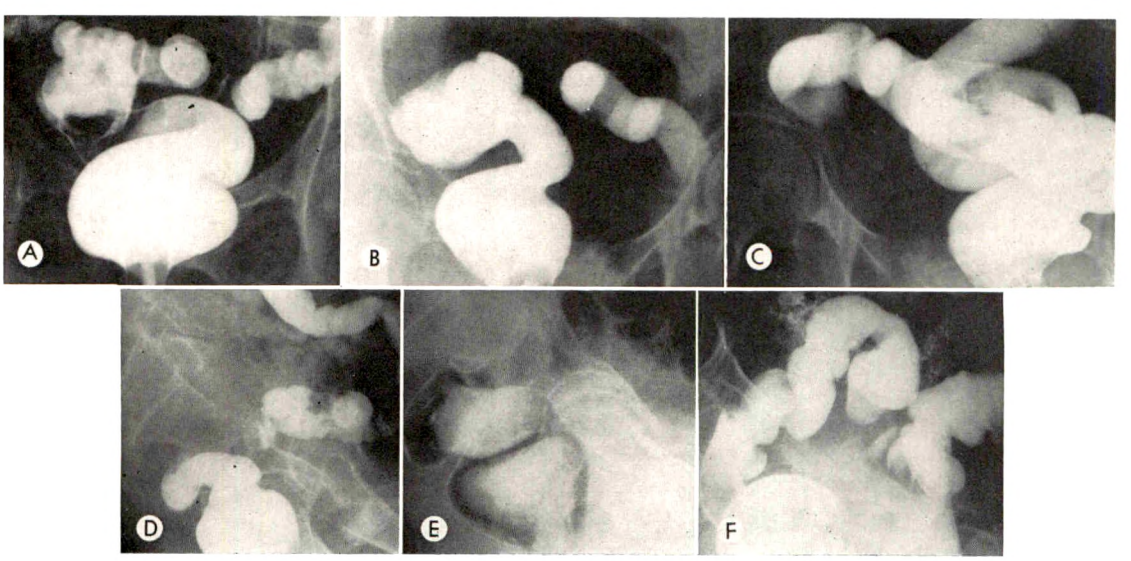


Fig. 4, Case iv. (*A*) Posteroanterior; (*B*) lateral with air; (*C*) supine, angled up; and (*D*) right anterior oblique "spot" roentgenograms. *Carcinoma.* A lesion of the rectosigmoid junction is demonstrated in the angled projection only.

Fig. 6. Case vi. (*A*) Posteroanterior; (*B*) right anterior oblique "spot;" (*C*) left anterior oblique "spot;" (*D*) lateral; (*E*) lateral, with air; and (*F*) supine, angled up roentgenograms. *Carcinoma of the rectosigmoid.* The lesion is seen in the prone angled and the left lateral double contrast views only.


A
B
C
D


A
B
C
D
E
F

angulated projections (Fig. 3, *A–D*; 4, *A–D*; 5, *A–D*; 6, *A–F*; and 7, *A–D*). In Case IV, the pathology is seen in the angled projection only (Fig. 4*D*).

DISCUSSION

It is our contention that angulated projections of the sigmoid and rectum should be a routine part of the barium enema examination. There are a few cases in which the degree of coiling is so slight that this is unnecessary, but this cannot always be anticipated, even at fluoroscopy. In almost all cases, angulation has improved the demonstration of this area and, in a considerable number, has permitted the authors to visualize lesions not otherwise demonstrable.

Until 1965, we considered the sigmoid to be adequately visualized by fluoroscopy with appropriate spot roentgenograms and routine posteroanterior and lateral projections. Since 1965, we have routinely made a right anterior oblique (prone) roentgenogram of the sigmoid, angulating caudad when the colon was filled with barium. This demonstrated a considerable number of lesions which were partially or completely hidden in other projections. In a number of cases, the barium in the termi-

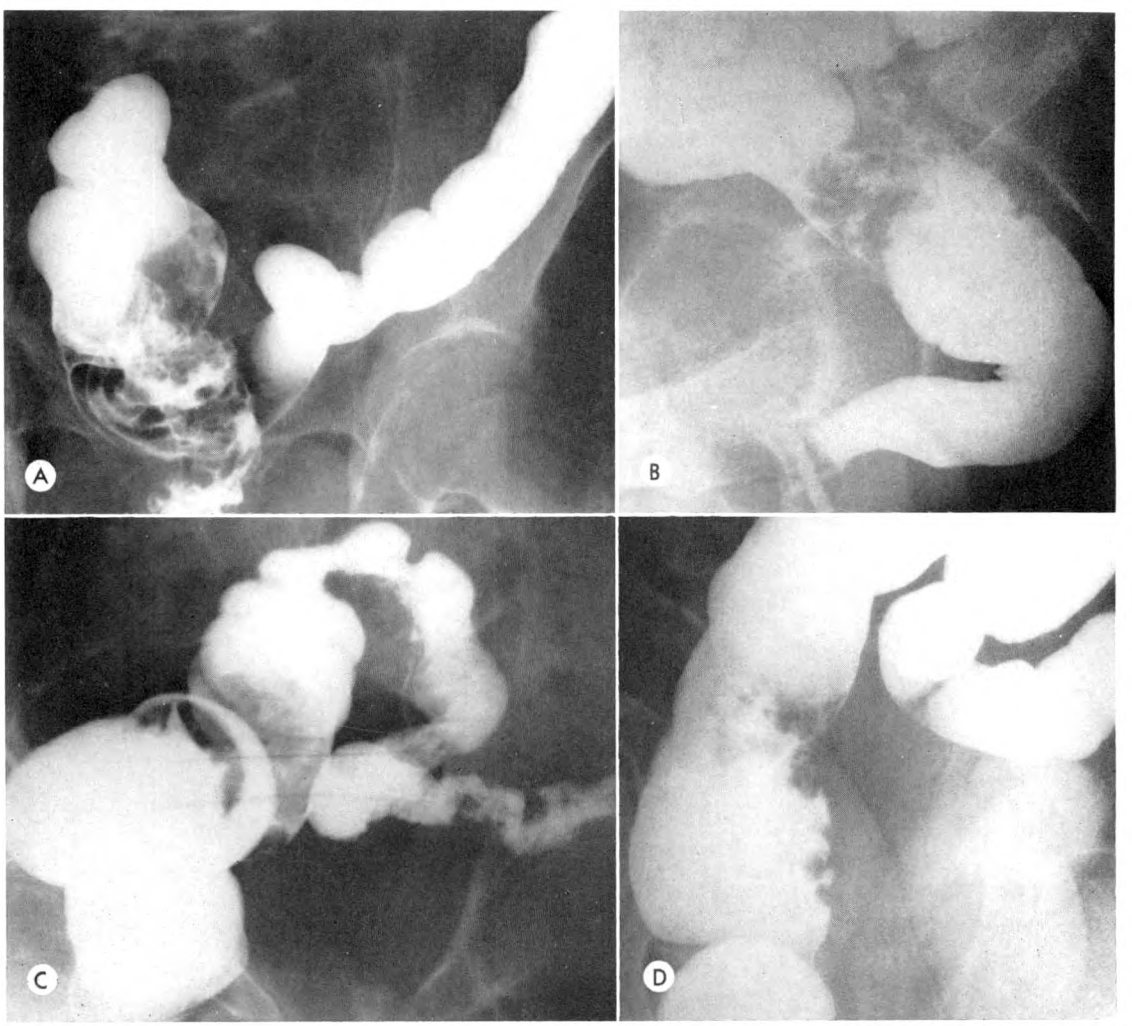


FIG. 7. Case VII. (*A*) Posteroanterior; (*B*) lateral; (*C*) right anterior oblique "spot;" and (*D*) supine, angled up roentgenograms. *Carcinoma of the rectosigmoid.* A multicentric lesion is demonstrated in all projections The extent of the lesion and its multicentric nature are best evaluated in the angled projection.

nal ileum and cecum obscured the sigmoid in this view and rendered it less valuable. For this reason, since 1968, we have added a left posterior oblique (supine) roentgenogram of the rectum and sigmoid with the tube angulated cephalad. It is made when only the left side of the colon is filled with barium. This method requires that the barium instillation be interrupted, but it is only a short delay. With proper planning only slight complexity is added to the examination. The left side of the colon is often less completely distended than when the entire colon is filled, but this does not seriously diminish the quality of the demonstration. The 2 views often complement each other.

CONCLUSION

Almost 50 per cent of colonic carcinomas are located in the sigmoid colon. Because of coiling of the sigmoid, this portion of the colon is often incompletely visualized in the routine barium enema examination. To solve this problem, we have added supine and prone angulated projections to the routine study of the colon. In this way, the nature and extent of lesions can be more accurately determined and lesions otherwise obscured can be demonstrated.

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ROENTGENOLOGIC APPEARANCE OF SYSTEMIC AMYLOIDOSIS INVOLVING GASTRO-INTESTINAL TRACT*

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IN EARLY gastrointestinal involvement by amyloidosis, the amyloid is commonly laid down in 2 sites:[13] (1) in and around the walls of small blood vessels; and (2) between the muscle fibers of the muscularis mucosae and main muscle coat. As deposition becomes heavier, the whole thickness of the intestinal wall may be involved, the affected segment being converted to an almost rigid tube. This can result in a wide spectrum of symptoms.

Occlusion of the small blood vessels may cause infarction of the intestine, with resultant hemorrhage and, in rare instances, even perforation. With deposition in muscle, motility disturbances may occur, manifested by dysphagia, gastric retention, constipation or diarrhea, and, eventually, intestinal obstruction which can prove fatal. In familial amyloidosis there is, in addition, selective deposition about the visceral nerves, and this will contribute to these motor symptoms. With heavier amyloid deposition, mucosal atrophy occurs, and this, combined with ischemia, leads to erosion and ulceration. Malabsorption is also known to occur in amyloidosis and is most likely the result of mucosal destruction, intestinal stasis, and vascular insufficiency.[7]

Correct diagnosis is important in these cases because surgical treatment may be useless and will result in high mortality and complications. Because of the symptoms, roentgenologic studies of the gastrointestinal tract will usually be performed, and the radiologist should therefore be able to contribute to the clinical diagnosis.

This paper reviews the roentgenologic appearances we found and develops some criteria for this diagnosis.

A simplified clinical classification for amyloidosis was proposed by Symmers.[13] The categories are: (1) generalized secondary amyloidosis (this is associated with a recognizable predisposing disease[3] such as chronic infections, paraplegia [40 per cent], rheumatoid arthritis [5 per cent], and multiple myeloma [15 per cent]); (2) generalized primary amyloidosis (there is no recognizable primary disease; we can include the familial amyloid syndrome,[3] recognized since Symmers' article); and (3) localized amyloidosis. Systemic amyloidosis secondary to multiple myeloma has characteristics more of the primary type, and indeed it is becoming apparent that many patients presenting with primary amyloidosis subsequently are found to have multiple myeloma, so this classification is not ideal. Neither is it completely tenable on the basis of the distribution of deposits or staining characteristics. However, it is clinically useful because it is based on the presence or absence of underlying disease, and for this reason will be utilized in this paper.

Gastrointestinal involvement is generally thought to occur more frequently in the generalized primary (70 per cent)[13] than in the secondary (55 per cent)[4] type of amyloidosis. Gilat and associates[6] found gastrointestinal involvement in 68 of a series of 70 cases which included both types of systemic amyloidosis, studied post mortem; this high incidence was attributed to a sys-

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tematic search and improved means of detection.

Amyloidosis has been the subject of much interest over the past 2 decades, due to techniques for direct organ biopsy and the availability of new and more efficient staining methods. Roentgenologic studies have been described by Wang and Robbins[14] and by Brown,[1] and sporadic individual case reports of amyloid disease involving the alimentary tract are available.[2,8–10,12] However, these have been too inconsistent to make possible the formulation of a diagnostic pattern, and descriptions of roentgenologic findings in amyloidosis of the esophagus, colon, and, to a lesser degree, small bowel have been notably scarce.

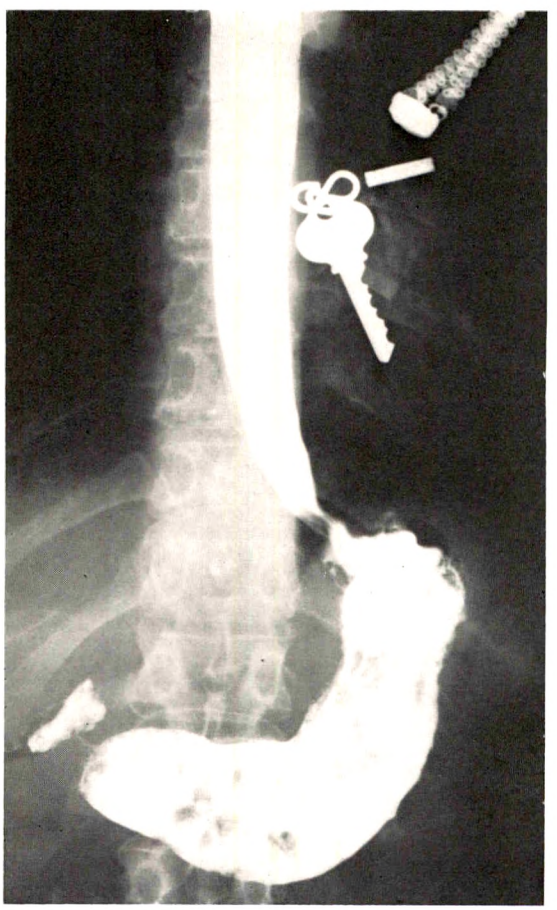


Fig. 1. A 52 year old man with longstanding rheumatoid arthritis, who presented with clinical steatorrhea. Rectal and jejunal biopsies were positive for amyloid. At fluoroscopy, weak motor activity of esophagus was observed. There was considerable amount of retained matterin stomach.



Fig. 2. A 62 year old man with clinical malabsorption due to primary amyloidosis. Rectal, gastric, and jejunal biopsies were positive for amyloid. Roentgenologic examination of small bowel reveals diffuse coarsening of mucosal pattern throughout.

MATERIAL AND METHOD

The records of all cases of systemic amyloidosis seen over a 10 year period at the Mayo Clinic were reviewed. One hundred and twenty-one biopsy proved cases were available. Of these, in only 24 was there both known gastrointestinal involvement and corresponding roentgenograms; in the other cases, either roentgenograms were not made or they had been discarded. In 18 cases there had been direct biopsy of the organ studied roentgenologically; in 6 cases the biopsy specimen was taken from another site (such as the rectum)[5] but the clinical symptoms pointed strongly to the organ roentgenographed as being involved with amyloid. These 24 cases form the basis of our study.

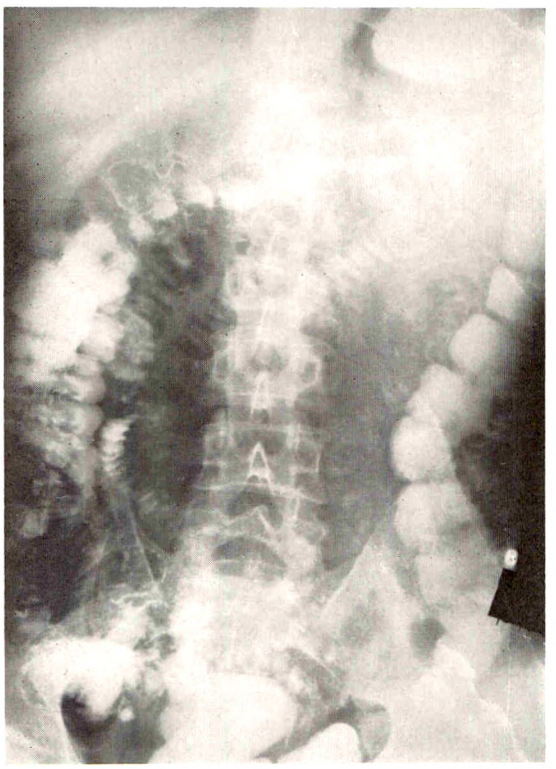


Fig. 3. A 50 year old woman with multiple myeloma; amyloid was found in muscle and mucosa of entire small bowel at autopsy. Plain roentgenogram of abdomen reveals barium, which was ingested 4 days previously, residual in stomach and throughout intestine.

RESULTS

Three cases of esophageal involvement were available. The abnormality was weak motor activity (Fig. 1) in 2 and gastroesophageal reflux in 1.

Gastric involvement was present in 12 cases. Two of these were normal roentgenologically even though direct biopsies were positive. Four of these cases showed marked retention of secretions in the stomach, in spite of adequate fasting and preparation, as the only abnormality. One case displayed both absence of normal rugal pattern and retention of secretions. The 5 other cases revealed absence of normal rugal pattern; in addition, in 1 instance the stomach actually appeared diminished in size. Abnormal gastric emptying was noted on 3 small bowel roentgenograms, barium remaining in the stomach for more than 5 hours after its ingestion.

The small bowel was involved in 16 cases. In 3 of these the roentgenologic appearance was normal, in spite of positive biopsies of the jejunal mucosa and the demonstration of malabsorption in 2 instances. Three cases manifested diffuse coarsening of the mucosal folds (Fig. 2). The remaining cases displayed a wide spectrum of motor disorders. Three had decreased propulsive activity of the entire small bowel; the barium failed to reach the colon within 5 hours in 1 case and 7 hours in another, and it was still present throughout the stomach and small bowel 4 days after ingestion in the third (Fig. 3). In the first 2 cases, this decreased motor activity resembled that seen in scleroderma of the small intestine because a continuous column of barium was apparent extending from the stomach throughout the proximal portion of the small bowel (Fig. 4). Three cases displayed dilatation of the proximal

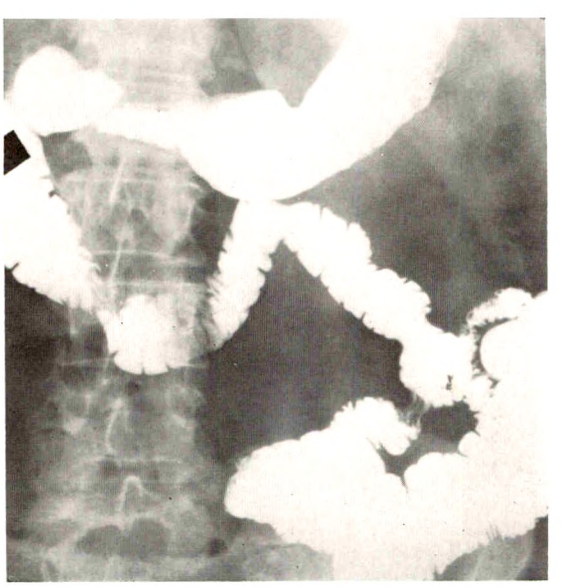


Fig. 4. A 66 year old man with amyloidosis secondary to longstanding lymphosarcoma of orbit. Jejunal biopsy was positive for amyloid. Roentgenologic examination reveals decreased propulsive activity of stomach and small bowel. Note continuous column of barium extending from stomach into proximal small bowel. Barium is still present in stomach 5 hours after ingestion.

small bowel and in 1 of these there was transient intussusception (Fig. 5). Two cases displayed segmental dilatation and apparent narrowing of the small bowel (Fig. 6). Five patients had symptoms of bowel obstruction and, on abdominal plain roentgenograms, showed distended loops of both small and large intestines (Fig. 7).

In 3 cases the colon was involved. In 1 case, amyloid deposits were found in the cecum and sigmoid at autopsy but the barium study gave normal results. In 1 case, distention of the colon was seen on the plain roentgenogram (Fig. 8*A*). Barium study the next day revealed abnormal motility with segmental narrowing and dilatation of the colon, but no definitive mucosal changes (Fig. 8, *B* and *C*). In the third case, the plain roentgenogram revealed a grossly distended colon, as is seen in mechanical large bowel obstruction. At autopsy this was found to be the result of almost total replacement of colonic muscle by amyloid material which was most heavily deposited at the splenic flexure.



Fig. 5. A 47 year old man with clinical malabsorption due to primary amyloidosis. Jejunal biopsy was positive for amyloid. Roentgenologic examination reveals dilatation of the proximal jejunum and transient intussusception.



Fig. 6. A 62 year old man with longstanding rheumatoid arthritis. Jejunal biopsy was positive for amyloid. Small bowel roentgenogram reveals segmental dilatation and apparent narrowing throughout. Barium remains in stomach 7 hours after its ingestion.

DISCUSSION

It is apparent from this study that amyloid deposits in the gastrointestinal tract will cause changes that can be detected by roentgenologic means. Mucosal changes consist of loss of rugal folds in the stomach and, in contrast, coarsening of the mucosal folds in the small bowel. No definite abnormalities of the mucosa could be detected in the esophagus or colon. These mucosal changes are nonspecific and are certainly less common than motor abnormalities.



FIG. 7. A 46 year old quadriplegic with amyloidosis proved by gingival biopsy; he had symptoms of intestinal obstruction for 6 weeks prior to death. Roentgenogram of abdomen reveals gaseous distention of both small and large intestines.

These motor abnormalities were manifested in the esophagus by reflux and weak motor activity, in the stomach by retained gastric secretions and delayed emptying, and in the small bowel by prolonged transit of barium, localized dilatation, and, in further advanced cases, generalized dilatation. In the colon we found abnormal motor activity with segmental narrowing and dilatation and eventual obstruction, presumably due to lack of propulsion.

Similar motor abnormalities in the esophagus, stomach, and small bowel have been noted in some previous case reports.[1,8–10,14] In reports of colonic involvement, emphasis has been placed on prominence of mucosal folds and features of chronic ulcerative colitis, such as absence of haustrations and narrowing.[2,12] In our cases, however, the mucosal pattern and haustrations appeared normal and other motor disorders, particularly gaseous distention, were more apparent.

These motor changes involving the entire gastrointestinal tract are in fact what can be expected from the known pathologic features—deposition of amyloid in and around the musculature and blood vessels in early cases and later extending to the full thickness of the bowel wall, with selective visceral neuropathy, in addition, in the familial type. This deposition is not always uniform, a fact which probably accounts for the normal results of barium studies we found in some of our cases even in the known presence of disease. The location and extent of amyloid deposition required to produce roentgenologic changes is not known, but in 2 of our cases it produced malabsorption without causing recognizable changes. Clearly then, a normal barium study will not exclude the presence of amyloid.

Differential diagnosis of amyloidosis from other, more common, diseases may not be possible from plain roentgenograms of the abdomen. In roentgenograms showing gaseous distention, mechanical obstruction from tumor or adhesions will be the primary consideration unless the radiologist is informed of the patient's history. With available barium studies, scleroderma and ischemic disease of the bowel must receive particular consideration in the differential diagnosis. Because the predominant pathologic feature in scleroderma of the gastrointestinal tract is muscle atrophy, the roentgenologic abnormalities (gastroesophageal reflux, delayed gastric emptying, and hypomotility and dilatation of the small bowel and colon) are quite similar to what has been noted in amyloidosis. However, characteristic cutaneous involvement is present in the majority of cases of scleroderma involving the gastrointestinal tract and the esophagus is invariably involved, so that the diseases often can be differentiated clinically. Roentgenologic findings in ischemia of the gastrointestinal tract include distention of the bowel and diminished or absent motility and so are similar to some cases of amyloidosis. Indeed, ischemia may be part of the

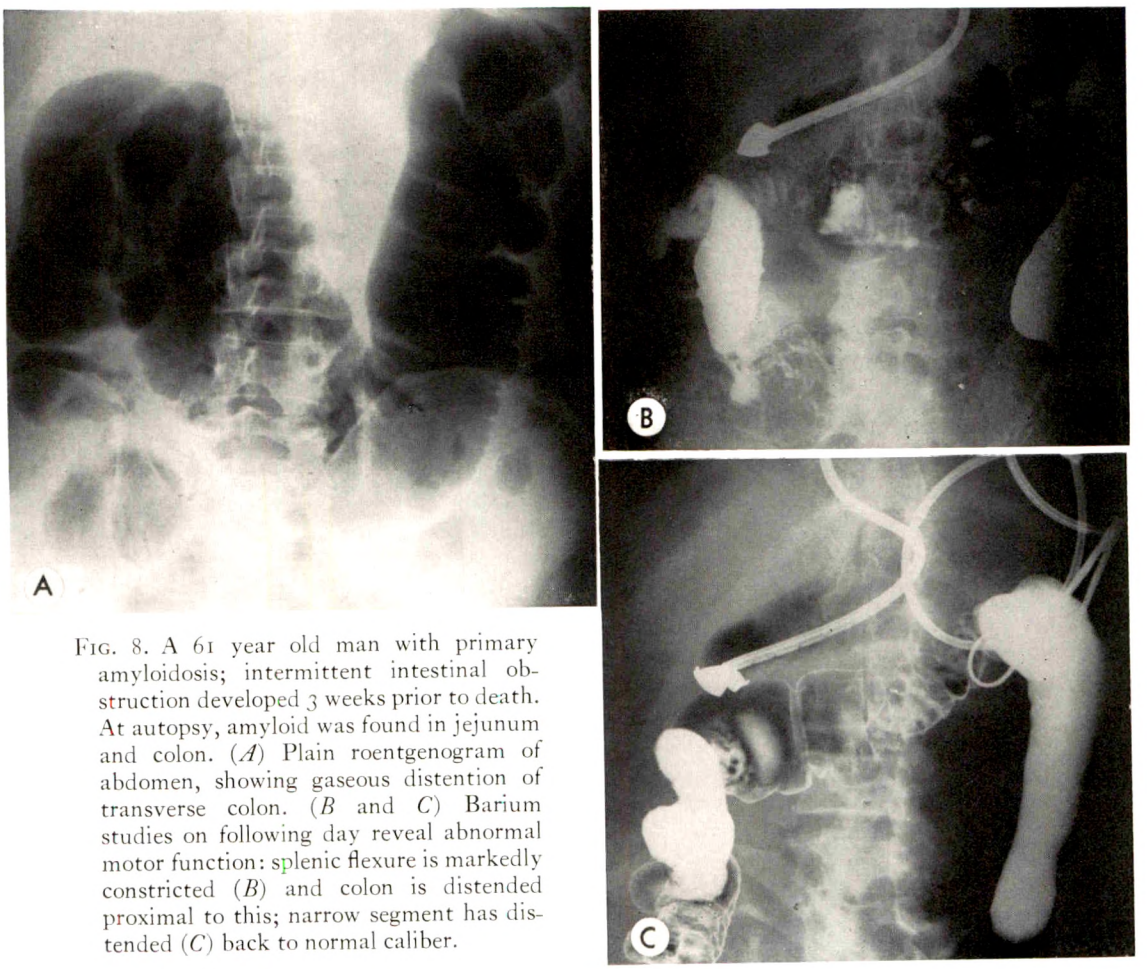


FIG. 8. A 61 year old man with primary amyloidosis; intermittent intestinal obstruction developed 3 weeks prior to death. At autopsy, amyloid was found in jejunum and colon. (*A*) Plain roentgenogram of abdomen, showing gaseous distention of transverse colon. (*B* and *C*) Barium studies on following day reveal abnormal motor function: splenic flexure is markedly constricted (*B*) and colon is distended proximal to this; narrow segment has distended (*C*) back to normal caliber.

mechanism for the roentgenologic changes seen in amyloidosis. In ischemia due to atherosclerosis, thrombosis, or embolism, the patients will usually manifest evidence of vascular disease elsewhere in the body. In amyloidosis, the changes in the bowel are chronic and slowly progressive.

It is sometimes stated that gastrointestinal deposition is of little significance because of the associated involvement of the heart or kidneys, which dominates the clinical picture in most cases of systemic amyloidosis. This was not the case in our series. Ten patients presented with steatorrhea (fecal fat more than 7 gm./24 hr.). Three patients died as a direct result of intestinal obstruction, and 5 additional patients had obstruction during their terminal illness.

Amyloidosis can be a difficult clinical diagnosis to make; in Mathews' series,[11] only 12 of 50 cases were diagnosed ante mortem. The radiologist should be able to suggest this diagnosis when he finds the roentgenologic features described, especially in a patient with a known predisposing disease (secondary type) or with cardiac or renal problems (primary type).

SUMMARY

The roentgenologic findings in 24 patients with known gastrointestinal deposition of amyloid are reviewed. The most significant finding was motor dysfunction, manifested by esophageal reflux and weak motor activity, gastric retention, intestinal dilatation or segmental dilatation and contraction, diminished motility, prolonged

transit, and, eventually, obstruction. Scleroderma and ischemia of the bowel can also produce these changes, but these diseases often can be differentiated clinically from amyloidosis, and a diagnosis of amyloid can be easily confirmed by biopsy of the involved organ or by rectal biopsy.

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GASTROINTESTINAL ROENTGENOGRAPHIC MANIFESTATIONS OF HEMOPHILIA*

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ONLY a few hemophiliac patients with roentgenographically documented gastrointestinal complications have been described in the literature.[3,4,7,11,13]

This report presents 3 additional cases. Recognition of the roentgenographic features of intramural intestinal bleeding coupled with the clinical history permits successful nonsurgical management of most hemophiliacs with gastrointestinal complications.

REPORT OF CASES

CASE I. A 15 year old hemophiliac male was admitted with complaints of nausea and abdominal pain of 24 hours' duration. Previous complications due to hemophilia included numerous episodes of hemarthrosis and one episode of gastrointestinal bleeding. On admission, the abnormal physical findings were tachycardia, generalized abdominal tenderness and limitation of joint movement.

Pertinent laboratory values were: hematocrit 31 per cent, white blood cell count 9,600, and negative stool guiac test. A small bowel series performed 24 hours after admission demonstrated obstruction of the distal ileum (Fig. 1, *A* and *B*). Surgery was contemplated but was deferred because hematologic control could not be achieved due to the presence of anti-antihemophiliac globulin. Nasogastric suction was instituted and 24 hours later a repeat roentgenogram showed partial resolution of the obstruction (Fig. 1*C*). The patient improved steadily, became asymptomatic and was discharged 8 days after admission. A small bowel examination 10 weeks later was normal (Fig. 1*D*).

CASE II. This 21 year old hemophiliac male was admitted with complaints of hematemesis and abdominal pain of 24 hours' duration. Previous symptoms relative to hemophilia consisted of swollen joints, nosebleeds and hematuria. On physical examination abnormal physical findings included tachycardia, hyperactive bowel sounds and limited knee and elbow motion.

On laboratory examination the hematocrit value was 30 per cent and a stool guiac test was positive. A small bowel series showed evidence of intramural jejunal bleeding (Fig. 2*A*). Hemostasis was accomplished by medical management and the clinical symptoms subsided. Ten weeks later on repeat examination, the small bowel appeared normal (Fig. 2*B*).

CASE III. A 10 year old hemophiliac male was admitted with a complaint of rectal bleeding of several hours' duration. Previous complications due to hemophilia were hemarthrosis, intramuscular bleeding and hematuria. On physical examination the only abnormal finding was limitation of motion of the involved joints. The hematocrit was 29 per cent. Although no lesions were evident on proctoscopy, blood was seen proximal to the tip of the proctoscope.

A barium enema examination demonstrated an intramural lesion of the distal descending colon which was consistent with localized intramural bleeding (Fig. 3, *A* and *B*). After institution of medical therapy, rectal bleeding ceased. No follow-up roentgenograms of the colon were obtained.

DISCUSSION

Hemophilia is a constitutional anomaly of blood coagulation characterized by hereditary transmission of a sex-linked recessive mendelian trait, decreased or absent serum concentrations of antihemophiliac globulin (AHG) and a life long tendency of spontaneous hemorrhage. The malady occurs clinically in males.

Clinical symptoms result from recurrent bleeding which is either spontaneous or

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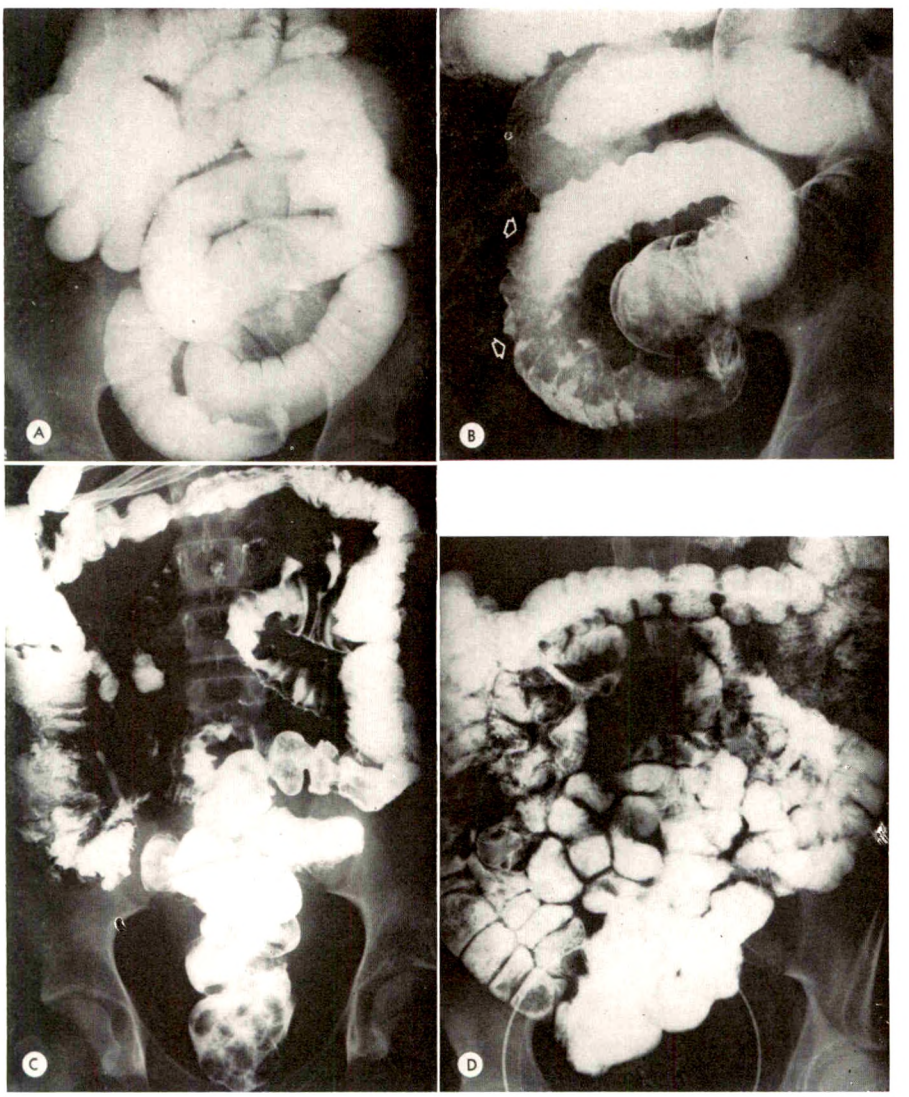


Fig. 1. Case 1. (*A*) Small bowel series demonstrates ileal obstruction with dilatation of proximal loops of small bowel. (*B*) Twelve hours later evidence of obstruction persists. The thickened and straightened ileal folds (arrows) and thickened ileal wall constitute roentgenographic features of intramural bleeding. (*C*) Twenty-four hours later most of the barium has passed through the distal ileum into the colon. Blood clots are present in the rectum. (*D*) Ten weeks later the small bowel appears normal.

follows minimal trauma. Soft tissue and large appendicular joints are the most common sites of spontaneous bleeding, accounting for 66 per cent of clinically demonstrated hemorrhages. Urinary tract bleeding is next in frequency, 38 per cent; followed by alimentary tract bleeding, 19 per cent; and central nervous system bleeding, about 5 per cent.[10,12]

Mortality in hemophilia is most frequently caused by gastrointestinal or central nervous system hemorrhage. Gastrointestinal symptoms caused by intramural bleeding into the bowel wall range from mild abdominal pain to those of obstruction. Significant gastrointestinal obstruction is unusual but does occur (Case 1). Periumbilical or lower abdominal pain is common[1] and must be differentiated from pain caused by bleeding into the iliopsoas muscle. Pain may also occur in the upper abdomen.

Either gross or microscopic evidence of blood is often present in the stool. A tender intra-abdominal hematoma is occasionally

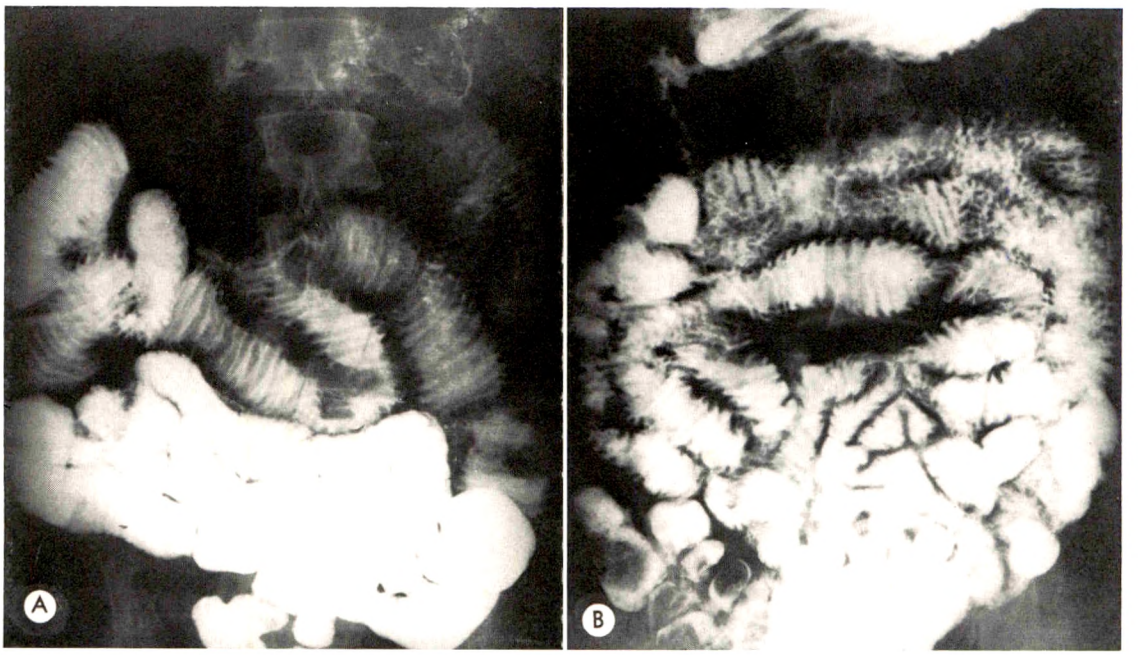


Fig. 2. Case 11. (*A*) Intramural bleeding has resulted in thickening and straightening of the jejunal folds and thickening of the wall. (*B*) Ten weeks later the jejunum appears normal.

palpable which, after several days' duration, may discharge into the bowel with the sudden appearance of melena.[9] Occasionally, hemorrhage into the bowel wall may rupture into the peritoneal cavity.[4] Protein-losing enteropathy associated with prolonged gastrointestinal bleeding has been reported.[4]

Roentgenographic examination of hemophiliac patients with abdominal symptomatology should include plain roentgenograms of the abdomen and a small bowel series or a barium enema examination, or both. Intramural bleeding is more often found in the small bowel than in the colon.[3] Spontaneous esophageal or gastric bleeding has not been documented.[6] The extent of intramural bleeding is variable and either a short or long segment of intestine may be involved. The roentgenographic features of intramural bleeding are often characteristic (Fig. 2*A*). Mucosal folds which are uniformly thickened, straightened and sharply outlined with narrow troughs in between result in a "picket fence" or "stacked coin" appearance.[3,11] The affected bowel is moderately rigid, its wall thickened and peristalsis is frequently absent or diminished. Mesenteric bleeding often contributes to variable degrees of luminal narrowing but complete obstruction is infrequent. Localized masses or contour defects are seldom present in intramural bleeding caused by clotting disorders[6,11] but may occur (Fig. 3, *A* and *B*).

Abdominal symptoms in hemophiliac patients often present difficult problems in differential diagnosis. Severe epigastric pain due to intramural hemorrhage may resemble a perforated peptic ulcer. Periumbilical or lower quadrant pain must be distinguished from that caused by appendicitis and other disorders. Full knowledge of the clinical history and roentgen findings, however, permits accurate diagnosis in most instances.

After intramural bleeding is established as a cause of gastrointestinal symptoms, hemostasis can usually be achieved by medical management. Surgery in hemophiliacs results in a high operative mortality and should be avoided whenever possible.[1] Even those patients with high grade intestinal obstruction, as in Case 1, may respond to medical therapy.[8] Once bleeding is controlled and reabsorption of intramural he-

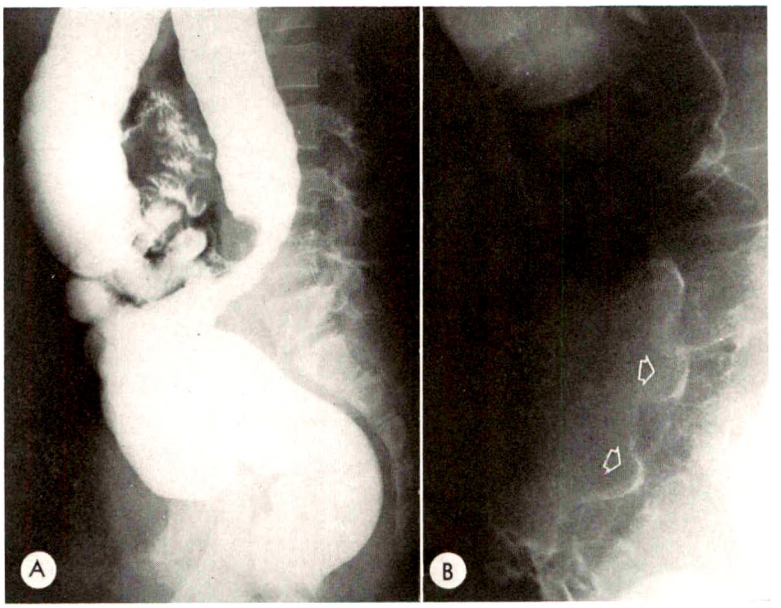


Fig. 3. Case III. (*A*) Barium enema examination demonstrates asymmetric narrowing, about 6 cm. in length, in the descending colon. (*B*) On a spot roentgenogram, the proximal sigmoid is seen to have a scalloped medial margin (arrows).

matoma begins, obstruction subsides. The patient's clinical symptoms and roentgenographic findings quickly resolve and full recovery is the rule.

SUMMARY

The clinical and roentgenographic features of intramural intestinal bleeding in hemophiliacs are illustrated by 3 case reports. Medical therapy as a rule results in recovery with complete reversal of abnormal roentgenographic findings.

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Janeway Lecture Committee: A. N. Arneson, Chairman, '71 St. Louis, Mo.; Milford D. Schulz, '73; Franz J. Buschke, '75.

Representatives on The American Board of Radiology: Justin J. Stein, Los Angeles, Calif.; Ralph M. Scott, Louisville, Ky.; E. Richard King, Richmond, Va.

Liaison Representatives on the National Council on Radiation Protection and Measurements: Herbert M. Parker, Richland, Wash.; Marvin M. D. Williams, Rochester, Minn.

Representative on The Board of Chancellors of the American College of Radiology: Juan A. del Regato.

Fifty-third Annual Meeting: Maria Isabel Hotel, México, D.F., México, March 15–18, 1971.

EDITORIALS

WHY COLOR ROENTGENOGRAPHY?

COLOR Roentgenography! I wonder what these words connotate? There would be widely varied expressions from both extremes—at least such has been the reaction so far.

What is wrong with conventional radiography? Why must color enter into roentgenography? Will it show different colored organs? These are but a smattering of the innumerable questions encountered when the subject of *Color Roentgenography* is discussed.

The Radiologist is the one most aware that the roentgenogram must be read in order to be interpreted, but in order to interpret one must see the variables of density to understand their significance. The work of Garland,[1] Greening and Pendergrass,[2] and Newell and Garneau[3] has indicated the great limitation of interpretation in the black and white roentgenogram. More recently attention has been focused on the physiology of the eye. It has been shown that visual acuity is accentuated by color; actually the eye is physiologically adjusted to color stimuli and has a greater sensitivity to these variables than to restricted grays and blacks.

The physicists have outstripped the radiologist in providing equipment with the ability to "disclose" changes which it is now capable to record on film. Advances have been made in every field of roentgenology—equipment, film, lighting, and many more developments too numerous to mention—all for the dynamic progress of Radiology. Yet we are limited by our ability to "see." Some amplification must occur to advance our observability threshold of visual acuity so that we may see smaller abnormalities earlier and recognize minor changes easily.

Many experimental efforts have been directed toward color in roentgenograms using color paper, color film, color fluorescent tubes, color fluorescing intensifying screens and even color light development. In addition, there has been our own method of simulated color roentgenography and the experimental incorporation of a sensitive chemical within the film emulsion. Any experiment that will lead finally to direct color roentgenography should theoretically be an improvement in diagnostic roentgen visualization. It is our belief that the first result will probably come from the fact that the chemical effect of x rays will actuate color change in various chemicals in direct proportion to the amount of transmitted photon energy. Thus the resultant roentgenogram will be a direct measure of density in accordance with the transmission of the roentgen beam through the subject and onto the film emulsion containing the sensitive chemical that will undergo color change in proportion to the amount of transmitted x ray. The problem is the chemical or chemical combination that will be so selectively sensitive.

Diagnosis in roentgenography requires accurate definitive observation of the visible changes that permit of interpretation. To do so, the radiologists must *see* the abnormality. Obvious gross abnormalities offer no difficulty. However, faint, small, obscure, borderline shadows present a great problem and challenge every skill of the

[1] Garland, H. L. Scientific evaluation of diagnostic procedures. *Radiology*, 1949, *52*, 309–327.

[2] Greening, R. R., and Pendergrass, E. P. Postmortem roentgenography of lung. *Radiology*, 1954, *62*, 720–725.

[3] Newell, R. R., and Garneau, R. Threshold visibility of pulmonary shadows. *Radiology*, 1951, *56*, 409–415.

interpreter. Consequently, reliability in obscure diagnosis is variable and sometimes almost impossible.

The relative diagnostic efficiency of various roentgen techniques and the variable interpretations of various outstanding interpreters, have been thoroughly investigated.[1] It was discovered that not only did the interpreter differ from his colleague in simple interpretations, but he differed with himself in repeat examinations. As Garland stated: "The human equation is present to a greater or lesser degree—in nearly every activity which can be tested—, and the medical profession can not be expected to be immune to this weakness." Add to this inherent variable the restrictive limitations of visual acuity and there is a combination that is difficult to overcome. However, we must hasten to add and stress that we are concerned with hardly visible, minimal, or borderline changes which are presented to the roentgenologist. He has through many years more than proved his reliability, accuracy, and invaluable diagnostic assistance in the evaluation of most clinical and pathologic problems with conventional roentgenography.

Since the eye is a fixed organ it must be helped from without and by adding color we allow a physiologic sensitivity to color hue to augment variables of brightness and gray differentiation. Lesions can be made more obvious by increasing the retinal response. The brightness pattern and illumination level will not alter the visual response beyond its present limitations of retinal reception. The differential visual stimulus is directly enhanced by color hue and color variation. The relative effect of successive contrast and temporal induction with color has been indicated by various physiologic studies.[4]

According to the Duplicity Theory of retinal function which was orginally proposed by Schultz in 1886,[4] it is contended that the rods of the retina function in dim light within the light intensity of less than 0.01 foot candle, and do not register sensations of color. The cones of the retina are sensitive only to higher levels of illumination and register the perception of color. The first is Scotopic Vision—the vision of the dark adapted eye; the latter is Photopic Vision—the vision of the light adapted eye.[4]

The retinal fovea is the location of most acute vision and contains cones primarily with but few if any rods. Therefore, at higher levels of illumination visual acuity is enhanced because of the added stimulus of color which stimulates only the cones and is registered only by the cones which permit of the most acute vision. The extrafoveal retina contains mostly rods which become more numerous toward the periphery where there is a complete absence of cones. The sensitivity of the rods varies with the proportion of rhodopsin, and as the intensity of illumination increases the rods are less utilized. Thereby, visual activity and acuity decrease proportionately.

Color vision is possible only with the light adapted eye and is directly associated with the function of the cones. The eye adapts readily to light as compared to the dark and readily perceives color. The dark adapted eye, on the other hand, is insensitive to color and consequently the rods have no color receptivity. The brightest colors for receptivity are grouped near the middle of the color spectrum and are predominantly green and yellow.

Tuddenham[5] in an excellent article entitled "Visual Physiology of Roentgen Diagnosis" explains explicitly the importance of the cones in perception and the delineation of a roentgen image. He justifies the contention that under conditions where cones are exposed to a subthreshold difference in illumination to perceive an image, the stimulus must be increased. He suggests that the retinal illumination gradient must be increased or it is neces-

[4] Best, C. H., and Taylor, N. B. Physiological Basis of Medical Practice. Fifth edition. Williams & Wilkins Co., Baltimore, 1950, pp. 1116–1125.

[5] Tuddenham, W. J. Visual physiology of roentgen diagnosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1957, *78*, 116–123.

sary to lower the threshold level. When an ideal threshold difference in illumination occurs across the cones, a roentgen image is perceived. However, it is possible with additive color to alter the illumination and threshold stimulus at the retinal level. This added stimulus along with the control of the factors governing spatial luminance gradient as described by Tuddenham results in selectively enhancing visual perception of the eye. The color variations aid the eye to perceive the impression of contrast enhancement. Tuddenham indicates that one of the important factors in differential threshold illumination is the number of cones involved in the judgement as well as the background, the total illumination, the fixation time, and the magnitude of the differential illumination of cones scanning the image.

It is further indicated that maintaining the adapting illumination of the environment at as high a level as possible without producing glare is ideal for the observer. Finally, the understanding of the visual physiology supports the thesis that the activity of the cones of the retina may be stimulated by the color contrasts in the visual field, so that color differences are more easily appreciated by the human eye than are the black-gray-white scale used in conventional roentgenography.

Thus *color roentgenography* may usher in a chromatic decade to Radiology. Minor changes which may presently escape detection may thus be disclosed. Contrast enhancement by color in roentgenograms is substantiated by a short review of the visual physiology and a brief survey of the intended experimental possibilities.

Louis J. Bonann, M.D.
1245 Glendon Avenue
Los Angeles, California 90024



ROBERT DRANE, M.D.
1887-1970

THE death of Dr. Robert Drane brought to a close the brilliant career of Georgia's oldest radiologist. Dr. Drane died April 11, 1970, at the Oglethorpe Sanatorium.

Dr. Drane was a native of Edenton, North Carolina, where he was born December 15, 1887. His parents were the late Rev. Robert Brent Drane and Mrs. Maria Louisa Drane. Dr. Drane was a graduate of the University of North Carolina, 1910; he received his Medical Degree from the University of Pennsylvania in 1913, where he was a member of Delta Kappa Epsilon, Phi Chi, and Alpha Omega Alpha. He served his internship at Protestant Epis-

copal Hospital, Philadelphia, Pa., 1913–1915; he was instructor of clinical medicine at the University of Wisconsin, 1915–1917. In 1917 Dr. Drane enlisted in the Medical Corps of the U. S. Army and shortly thereafter was assigned to the British Expeditionary Forces, 17th Division, 52nd Field Ambulance, operating on the Western Front in and around Arras, France. He was wounded in January 1918, and received the Purple Heart. He was detached and served with the Scottish Highland Division during the big March Drive. Upon separation from the army in 1918, he decided to specialize in Radiology.

Dr. Drane moved to Savannah during the fall of 1919. Early in 1920 when the DeRenne Apartments were built on Liberty Street, Dr. Drane opened a private office there. It was one of the first private x-ray offices in the state. That office remained in the DeRenne Apartments for almost half a century. In 1952 Dr. Drane was joined by Dr. John B. Rabun and in 1958 a new office was built at 311 East Hall. About 1966 the DeRenne office was closed.

Dr. Drane was a Charter Member of the Georgia Radiological Society and served as its second President. He was a Past President of the Georgia Medical Society. He was a Past Chairman of the Finance Committee of the American Roentgen Ray Society and a Fellow of the American College of Radiology. He was also a member of the American Medical Association, Southern Medical Association, Medical Association of Georgia, and the Radiological Society of North America.

His local connections were: Benevolent Society, Order of Colonial Wars, Oglethorpe Club, and a former member of the Cotillion Club, Forrest City Gun Club, and Savannah Golf Club. He was a member of Christ Episcopal Church.

Dr. Drane had served on the medical staff of the following Savannah hospitals: Oglethorpe Sanatorium; St. Joseph's Hospital; Georgia Infirmary; Telfair; Central of Georgia; Memorial; and the U. S. Marine Hospital. He retired, due to ill health, in July 1967, from active practice. He was honored in May 1969, at the state meeting of the Medical Association of Georgia at its meeting in Savannah at the Savannah Inn and Country Club, with a Certificate in recognition of his 50 years of service.

Dr. Drane is survived by his wife, Mrs. Naomi Everett Drane; two sons, Robert Brent Drane of Savannah, and Jack Albert Gore of Falls Church, Virginia; one sister, Mrs. J. C. Webb of Hillsborough, North Carolina; and one brother, Rev. Fred B. Drane of Edenton, North Carolina; and several nieces and nephews.

His colleagues describe him as a brilliant diagnostician whose ability was acquired by long experience and excellent judgment plus a fabulous memory. He was noted as an extremely modest man with a high sense of medical ethics. He was known far and wide as one of Radiology's pioneers—an honest, modest, brilliant gentleman.

John B. Rabun, M.D.
311 Hall Street, East
Savannah, Georgia 31401

NEWS ITEMS

RADIATION AND THE ORAL CANCER PATIENT

A panel discussion on Radiation and the Oral Cancer Patient will be presented at the 18th Annual Meeting of the American Academy of Maxillofacial Prosthetics at the Desert Inn, Las Vegas, Nevada, November 3–5, 1970.

The panel will consist of a radiotherapist, plastic surgeon, and dentists. Dr. Theodore L. Phillips from the University of California San Francisco Medical Center will present a paper titled, "The Indications for Various Types of Radiation Therapy and the Rationale for Their Use." Also covered will be surgical aspects following radiation therapy and the complications that may occur, and methods to diminish these complications.

For further information contact Victor Matalon, D.D.S., Chairman, Public Relations Committee, 1700 Holcomb Boulevard, Room P-723, Houston, Texas 77025.

SEVENTH FORENSIC DENTISTRY COURSE OF ARMED FORCES INSTITUTE OF PATHOLOGY

Captain Bruce H. Smith, MC, U. S. Navy, The Director of the Armed Forces Institute of Pathology, is pleased to announce the Seventh Course in Forensic Dentistry to be given at the Institute October 5–9, 1970.

This program is presented to develop a critically needed nucleus of dentists trained in the principles of identification and dental jurisprudence. The Institute is responding to the urgent collective requirements of the dental, medical, legal and law enforcement professions.

To accomplish this a distinguished faculty representing the four professions and their important subdivisions will be brought together.

The faculty of the course will include dentists with experience in forensic odontology, general and oral pathologists, law enforcement officials, lawyers, anthropologists and identification experts.

Among the lecture subjects will be: Recent Advances in Identification; Dental Identification in Mass Disasters; the Relationship between Forensic Dentistry and the Federal Bureau of Investigation; Study of Bite Marks; and Professional Liability.

Unique features of the course include a laboratory session of identification of human remains by comparison of dental records and a mock trial depicting the role of the dentists as an expert witness and defendant.

Members of the dental, medical, legal, and law enforcement professions are invited to make application to the Director, ATTN: MEDEM-PG, Armed Forces Institute of Pathology, Washington, D. C. 20305.

Early reservations are advised.

SYMPOSIUM ON CLINICAL DYNAMIC STUDIES WITH RADIONUCLIDES

The Department of Radiation Therapy & Nuclear Medicine of the Hahnemann Medical College & Hospital will sponsor a Symposium on "Clinical Dynamic Function Studies with Radionuclides," November 20–21, 1970, at the Marriott Motor Hotel, Philadelphia.

The program will include: Fundamental Aspects, Cerebral, Cardiovascular, Renal, and Pulmonary Dynamic Function Studies.

For further information contact: Millard N. Croll, M.D., 230 N. Broad Street, Philadelphia, Pennsylvania 19102.

THIRD NATIONAL CONGRESS OF NUCLEAR MEDICINE IN ISRAEL

The Third National Congress of Nuclear Medicine in Israel will be held on December 20–22, 1970, in Tel Aviv University, Ramat Aviv.

Topics of the scientific sessions are: Nuclear Neurology, Oncology, Gastro-

enterology, Nephrology, Orthopedics, Pediatrics, Thyroidology, Radioimmunology, Radiopharmaceuticals, Safety Problems, and Preliminary Communications.

There will be Scientific Exhibits on medical subjects; instrumentation; radiopharmaceuticals; and bibliography.

Correspondence should be addressed to: Secretary 3rd Congress Nuclear Medicine, Tel Aviv University, Faculty of Continuing Medical Education, Section of Nuclear Medicine, Ramat Aviv, Israel.

THE TWENTY-THIRD ANNUAL MIDWINTER CONFERENCE

The Los Angeles Radiological Society is pleased to announce its Twenty-third Midwinter Radiological Conference to be held January 30th and 31st, 1971, at the International Hotel (International Airport), Los Angeles, California.

The Guest Speakers are: *Diagnosis*, Bjorn Nordenstrom, M.D., Karolinska Chest Hospital, Stockholm, Sweden; Elias G. Theros, M.D., Registry of Radiologic Pathology, Washington, D.C.; Keith Jefferson, M.D., National Heart Hospital, London, England; and Robert Wise, M.D., Lahey Clinic, Boston, Massachusetts; *Therapy*, William E. Powers, M.D., National Cancer Institute, Bethesda, Maryland; and *Nuclear Medicine*, Alexander Gottschalk, M.D., Argonne Cancer Research Hospital, Chicago, Illinois.

For advance registration please write to Richard Witten, M.D., 2131 West 3rd Street, Los Angeles, California 90057, and for Hotel Accommodations to Convention Manager, International Hotel, Los Angeles, California 90045.

ASPEN CONFERENCE ON RADIOLOGY AND THE NEW TECHNOLOGY

This conference is presented by the Division of Radiology, Denver General Hospital and will be held at the Aspen Institute for Humanistic Studies, Aspen, Colorado, March 23–27, 1971.

The meeting is designed for radiologists and scientists interested in the effect of the new technology on the clinical practice of diagnositic radiology and nuclear medicine.

The Guest Faculty in *diagnostic radiology* includes: Herbert Abrams, M.D., Harvard University; Kurt Amplatz, M.D., University of Minnesota; John Campbell, M.D., University of Indiana; Marvin Daves, M.D., University of Colorado; Eric Milne, M.D., University of Toronto; and Robert Moseley, M.D., University of Chicago. In *nuclear medicine*: Don Brown, M.D., University of Colorado; Ralph Gorten, M.D., Duke University; Alexander Gottschalk, M.D., University of Chicago; David Kuhl, M.D., University of Pennsylvania; John McAfee, M.D., State University of New York; E. James Potchen, M.D., Washington University; and Philip Rubin, M.D., University of Rochester.

Advance registration is required and should be submitted prior to December 1, 1970.

For further information please write to Emanuel Salzman, M.D., Director, Division of Radiology, Denver General Hospital, Denver, Colorado 80204.

SIXTEENTH ANNUAL MEETING OF THE HEALTH PHYSICS SOCIETY

The Sixteenth Annual Meeting of the Health Physics Society will be held at the Waldorf Astoria Hotel in New York City, July 11–15, 1971.

Abstracts (20 copies) for presentation at the meeting must be sent to: Constantine J. Maletskos, Cancer Research Institute, 185 Pilgrim Road, Boston, Massachusetts 02215, before February 1, 1971. Forms and instructions for submitting an abstract may be obtained from R. F. Cowing, Health Physics Society, P.O. Box 156, Easy Weymouth, Massachusetts 02189.

For information regarding the meeting and exhibit space, write to: Lee Gemmell, Health Physics Division, Brookhaven National Laboratory, Upton, New York 11973.

BOOKS RECEIVED

Glossary of Words and Phrases Used in Radiology, Nuclear Medicine and Ultrasound. Second edition. By Lewis E. Etter, B.S., M.D. F.A.C.R., Professor of Radiology and Chief, Radiological Service, Western Psychiatric Institute and Falk Clinic, Presbyterian-University Hospital School of Medicine, University of Pittsburgh; Consultant, C. Howard Marcy State Hospital, Leech Farm, Pittsburgh, Pa. Cloth. Pp. 355. Price, $22.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1970.

The Pharmacological Basis of Therapeutics. Fourth edition. Edited by Louis S. Goodman, M.A., M.D., D.Sc.(Hon.), Professor and Chairman, Department of Pharmacology, University of Utah College of Medicine, Salt Lake City, Utah; and Alfred Gilman, Ph.D., William S. Lasdon Professor and Chairman, Department of Pharmacology, Albert Einstein College of Medicine of Yeshiva University, Bronx, N. Y. Cloth. Pp. 1,794, with some figures. Price, $25.00. The Macmillan Company, 866 Third Avenue, New York, N. Y. 10022, 1970.

A Text-Book of X-Ray Diagnosis. Volume V: The Pelvis and the Abdomen. By British authors in six volumes. Fourth edition. Edited by S. Cochrane Shanks, C.B.E., M.D., F.R.C.P., F.F.R., Consulting Radiologist, University College Hospital, London; and Peter Kerley, C.V.O., C.B.E., M.D., F.R.C.P., F.F.R., Consulting Radiologist, Westminster Hospital and the National Heart Hospital, London, England. Cloth. Pp. 524, with 423 figures. Price, $22.00. In the Western Hemisphere, W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1970.

Breast Cancer: Early and Late. A collection of papers presented at the Thirteenth Annual Clinical Conference on Cancer, 1968 at The University of Texas M. D. Anderson Hospital and Tumor Institute at Houston, Texas. Cloth. Pp. 477, with some figures. Price, $14.50. Year Book Medical Publishers, 35 East Wacker Drive, Chicago, Ill. 60601, 1970.

Environmental Surveillance in the Vicinity of Nuclear Facilities. Proceedings of a Symposium by the Health Physics Society. Edited by William C. Reinig, B.M.E., C.H.P., Radiological Sciences Division, Savannah River Laboratory, E. I. du Pont de Nemours and Co., Aiken, S.C. Cloth. Pp. 465, with some figures. Price, $29.00. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1970.

Die Wirbelsäulenleiden und ihre Differentialdiagnose. By J. E. W. Brocher. Cloth. Pp. 881, with 709 figures. Price, DM 276.–. Georg Thieme Verlag, 7 Stuttgart 1, P. O. Box 732, Herdweg 63, Germany, 1970.

The Radiologic Clinics of North America. Symposium on Diagnostic Radiology of Bone Disease. Robert S. Sherman, M.D., Guest Editor. August, 1970, Volume VIII, No. 2. Cloth. Pp. 118. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1970.

Electrical Impedance Plethysmography. The Electrical Resistive Measure of the Blood Pulse Volume, Peripheral and Central Blood Flow. By Jan Nyboer, D.Sc., M.D., Director of Research, Rehabilitation Institute, Department of Physical Medicine, Wayne State University, Detroit, Mich.; Professor, Department of Physiology and Pharmacology, Wayne State University Medical School, Detroit, Mich.; Former Chief, Division Cardiovascular Physiology Research, Electrocardiographer and Associate Physician, Department of Medicine, Harper Hospital, Detroit, Mich. Cloth. Pp. 390, with some figures. Price, $22.00. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1970.

Trattato di Radiodiagnostica: Indice Analitico Generale. By Luigi Turano, Direttore dell' Istituto di Radiologia dell Università di Roma. Cloth. Pp. 130. Price, L 2,500. Union Tipografico-Editrice Torinese, Italy, 1970.

On Death and Dying. By Elisabeth Kübler-Ross, M.D. Paper. Pp. 260. Price, $1.95. The Macmillan Company, 866 Third Avenue, New York, N.Y. 10022, 1970.

Controlled Therapeutic Trials: Guidelines and List of Trials in Progress. Edited by R. Flamant. UICC Technical Report Series, Volume 7. International Union Against Cancer, Geneva, 1970. Paper. Pp. 55. Price, $3.00. International Union Against Cancer, P. O. Box 400, 1211 Geneva 2, Switzerland, 1970.

Arteriographic Investigation of the Hip in Adult Subjects. A Clinical Study of the Arteries in the Healthy Hip, in Neck and Pertrochanteric Fractures, and in Necrosis of the Femoral Head. By Herbert Müssbichler. Paper. Pp. 39, with some figures. Acta Orthopaedica Scandinavica, Supplementum No. 132. Munksgaard Forlag, Prags Boulevard 47, Copenhagen, Denmark, 1970.

Late Results After Meniscectomy in the Knee Joint. A Clinical and Roentgenologic Follow-up Investigation. By Helge Appel. Paper. Pp. 111, with some figures. Acta Orthopaedica Scandin-

avica, Supplementum No. 133. Munksgaard Forlag, Prags Boulevard 47, Copenhagen, Denmark, 1970.

An Experimental Study of the Effects of Growth on the Relationship of Tendons and Ligaments to Bone at the Site of Diaphyseal Insertion. By T. Videman. Paper. Pp. 22, with some figures. Acta Orthopaedica Scandinavica, Supplementum No. 131. Munksgaard Forlag, Prags Boulevard 47, Copenhagen, Denmark, 1970.

Report of the Working Party on the Organisation of Radioactive Isotope Services. Department of Health and Social Security. Reports on Public Health and Medical Subjects No. 126. Paper. Pp. 14. Price, $0.50. Sales Section, British Information Services, 845 Third Avenue, New York, N. Y. 10022, 1970.

Federal Budget Priorities for Research and Development. By Michael S. March. Paper. Pp. 45. Price, $1.50. University of Chicago Center for Policy Study, 5801 Ellis Avenue, Chicago, Ill. 60637, 1970.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual meeting: Deauville Hotel, Miami Beach, Fla. September 29–October 2, 1970.

American Radium Society
Secretary, Dr. Jerome M. Vaeth, Saroni Tumor Institute, 1600 Divisadero St., San Francisco, Calif. 94115. Annual meeting: Mexico City, Mexico, March 15-18, 1971.

Radiological Society of North America
Secretary-Treasurer, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., November 29–December 4, 1970.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: St. Louis, Mo., Chase-Park Hotel, March 30–April 3, 1971.

Section on Radiology, American Medical Association
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga., 30322. Annual meeting: Atlantic City, N. J., June 20–24, 1971.

American Board of Radiology
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Denver, Colo., Dec. 7–11, 1970, Denver-Hilton Hotel; Bal Harbour, Fla., June 7–11, 1971, Americana Hotel; and Dallas, Tex., Dec. 6–10, 1971, Statler-Hilton Hotel.

Written examinations are scheduled in June of each year in 12 large centers, and applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Beginning in 1970 the deadline for receiving applications for any examination is Sept. 30 of the year preceding.

American Association of Physicists in Medicine
Secretary, Leonard Stanton, Hahnemann Medical College, 230 N. Broad St., Philadelphia, Pa. 19102. Annual meeting to be announced.

American Society of Therapeutic Radiologists
Secretary, Dr. Carl R. Bogardus, Jr., University of Oklahoma Medical Center, Oklahoma City, Oklahoma 73104 Fall meeting: Mountain Shadows, Scottsdale (Phoenix), Ariz., November 11-13, 1970.

American Institute of Ultrasound in Medicine
Secretary, F. J. Fry, M.Sc., Bioacoustics Lab., University of Illinois, Urbana, Ill.

American Society of Neuroradiology
Secretary-Treasurer, Dr. Eugene V. Leslie, Edward J. Meyer Memorial Hospital, 462 Grider St., Buffalo, N. Y. 14215.

Thirteenth International Congress of Radiology
Meeting: Madrid, Spain, Oct. 13–19, 1973.

Tenth Inter-American Congress of Radiology
Counselor for the United States, Dr. Manuel Viamonte, Jr., University of Miami School of Medicine, Jackson Memorial Hospital, Miami Fla. 33136.
President, Dr. Victor A. Marcial, Puerto Rico Nuclear Center, Caparra Heights Station, San Juan, Puerto Rico 00935.
Meeting: San Jeronimo-Hilton Hotel, San Juan, Puerto Rico, May 16-22, 1971.

Inter-American College of Radiology
President, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.

Alabama Chapter of ACR
Secretary, Dr. William V. Weldon, Medical Arts Building, Birmingham, Ala. 35205. Meets time and place of Alabama State Medical Association.

Alaska Radiological Society
Secretary, Dr. Bruce C. Wright, Providence Hospital, Anchorage, Alaska. Meets third Wednesday each month.

Arizona Radiological Society, Chapter of ACR
Secretary, Dr. Wesley S. Fee, 2421 E. 6th St., Tucson, Ariz. 85719. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Chapter of ACR
Secretary-Treasurer, Dr. Wilma C. Diner, Univ. of Arkansas Medical Center, Little Rock, Ark. 72201. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Elliott C. Lasser, University Hospital of San Diego County, San Diego, Calif. 92103. Annual Meeting: Durham, N. C., May 13–15, 1971, with the Duke University and University of North Carolina Radiology Departments serving, as co-hosts.

Atlanta Radiological Society
Secretary, Dr. Richard S. Colvin, Emory University Clinic, Atlanta, Ga. 30322. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Advisor, Colonel Paul E. Sieber. *Secretary*, LTC Peter B. Riesz, USAH Bad Cannstatt, APO 09154, New York, N. Y. Meets quarterly.

Blockley Radiological Society
Secretary-Treasurer, Dr. R. John Gould, 41 Lombardy Rd., Drexel Hill, Pa. 19026.

Bluegrass Radiological Society
Secretary-Treasurer, Joseph A. Sayeg, Ph.D., Radiation Physicist, University of Kentucky, Lexington, Ky. 40506. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. David Bruce Hayt, 600 E. 233rd St., Bronx, N. Y. 10466. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Rubem Pochaczevsky, 300 Skillman Ave., Brooklyn, N.Y. 11211. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Herbert Pirson, 145 High Park Blvd., Buffalo, N.Y. 14226. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, Childrens Hospital of Los Angeles, P.O. Box 54700, Los Angeles, Calif. 90054.

Catawba Valley Radiological Society
Secretary, Dr. Emmett R. White, P. O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 P.M.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Central New York Radiological Society
Secretary-Treasurer, Dr. David N. Cheris, Community General Hospital of Greater Syracuse, Broad Road, Syracuse, N. Y. 13215. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary, Dr. James V. Blazek, 2586 Lane Rd., Columbus, Ohio 43220. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Chicago Roentgen Society
Secretary-Treasurer, Dr. William T. Moss, 250 E. Superior St., Chicago, Ill. 60611. Meets third Thursday of each month, October to April, except December, at the Bismarck Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. James Farmer, 18869 Canyon Rd. Fairview Park, Ohio 44126. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Marvin L. Daves, Univ. of Colorado Medical Center, 4200 E. Ninth Ave., Denver, Colo. 80220. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. V. V. Kirby, 1722 Spring Lake Dr., Arlington, Tex. 76010. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. James H. Taylor, Wilmington Medical Center, Wilmington, Del. 19899.

East Bay Radiological Society
Secretary-Treasurer, Dr. S. Walter Kran, Doctors' Hospital of San Leandro, 13855 East 14th St., San Leandro, Calif. 94578. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. Eberhard F. Besemann, Baroness Erlanger Hospital, Chattanooga, Tenn. 37403. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary, Dr. Wm. F. Lindsey, 1215 Hodges Dr., Tallahassee, Fla. 32303. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Allen L. Sheer, University Community Hospital, 13505 N. 31st St., Tampa, Fla. 33612. Meets in January, March, May, July, September and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. Walker Harris, The Medical Center, Columbus, Ga. 31902. Meets in spring and fall at Annual State Society Meeting.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. John Kathe, North Shore Hospital, Miami, Fla. 33150. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. Roland P. Ernst, 3720 Washington Ave., St. Louis, Mo. 63108.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Virgil R. Jobe, Jr., 888 South King St., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, John H. Pingel, Argonne National Laboratory, 9700 S. Cass Ave., Argonne, Ill. 60439. Annual Meeting: Waldorf Astoria Hotel, New York City, July 11–15, 1971.

Houston Radiological Society
Secretary, Dr. Kenneth M. Jensen, 1615 St. Joseph Prof. Bldg., Houston, Texas 77002. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025

Idaho State Radiological Society, Chapter of ACR
Secretary, Dr. Hugh P. Smith, Jr., 130 E. Bannock, Boise, Id. 83702. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. Jack L. Melamed, 1230 Sunset Rd. Winnetka, Ill. 60093. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. Dale B. Parshall, Elkhart General Hospital, P.O. Box 1329, Elkhart, Ind. 46514.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Wm. R. Allen, 155 S. 18th St. Kansas City, Kan. 66102. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Joan R. Hale, 402 Heyburn Bldg. Louisville, Ky. 40202. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N.Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Harold L. Atkins, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Harry T. Vanley, St. Mary's Long Beach Hospital, Long Beach, Calif. 90083. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Annual Midwinter Conference: Jan 30–31, 1971.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiolcgical Society, Chapter of ACR
Secretary, Dr. Nathan Stofberg, 4519 Hawksbury Rd., Pikesville, Md. 21208.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Norman L. Siadowsky, The Faulkner Hosp., 1153 Centre St., Jamaica Plain, Mass. 02130

Memphis Roentgen Society
Secretary-Treasurer, Dr. Webster Riggs, Jr., The University of Tennessee College of Medicine, Department of Radiology, Walter F. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Ind. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary, Dr. David P. Corbett, Harper Hospital, De-

troit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. James E. Bell, 8700 W. Wisconsin Ave., Milwaukee, Wis. 53213. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Warren L. Kump, 4243 Glenwood Ave., Minneapolis, Minn. 55422. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ottis G. Ball, 5356 Balmoral Drive, Jackson, Miss. 39211. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Arthur A. Porporis, 100 N. Euclid Ave., St. Louis, Mo. 63108.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. Jon A. Anderson, Doctor's Building, 1231 N. 29th Street, Billings, Mont. 59101.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. Gordon F. Johnson, 4239 Farnam, Omaha, Neb. 68131. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. Harris W. Knudson, 2020 W. Charleston Blvd., Las Vegas, Nev. 89102.

New England Roentgen Ray Society
Secretary, Dr. Stefan C. Schatzki, 1180 Beacon St., Brookline Mass. 02146. Meets third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary, George Farmlett, 33 Round Bay Rd., Keene, N. H. 03246. Meets four to six times yearly.

New Mexico Society of Radiologists Chapter of ACR
Secretary, Dr. Donald A. Wolfel, Albuquerque, New Mexico. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New York Roentgen Society
Secretary-Treasurer, Dr. Samuel H. Madell, 1. E. 82nd St., New York, N. Y. 10028. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference: Waldorf Astoria Hotel, New York, April 29–May 1, 1971. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. John J. Magovern, 520 Franklin Ave., Garden City, N. Y. 11530.

North Carolina Chapter of ACR.
Secretary-Treasurer, Dr. James F. Martin, 300 S. Hawthorne Road, Winston-Salem, N. C. 27103. Fall Meeting, Mid Pines Hotel, Southern Pines, N. C., Oct. 31, 1970, 2 P.M.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 13th St., So., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. John W. Morris, III., Department of Radiology, Halifax District Hospital, Daytona Beach, Fla. 32015. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Herbert F. Reilly, Jr., 12 Sandalwood Rd., Scotia, New York 12302. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiological Society
Secretary-Treasurer, Dr. Kevin Ryan, Woodland Medical group, Woodland, Calif. 95695. Meets fourth Monday of Sept., Nov., Jan., March and May at Aldo's Restaurant in Sacramento.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Joseph Hanson, 1544 South Byrne Road, Toledo, Ohio 43614.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. Richard B. Price, 204 Medical Tower Bldg., Oklahoma City, Okla. 73112. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Roderick A. Silveira, 100 E. Valencia Mesa Dr., Fullerton, Calif. 92632. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at the Orange County Medical Association Bldg., Orange, Calif.

Oregon Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Clinton B. Sayler, 214 Medical Dental Bldg., Portland, Oregon 98205. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd.; Beaverton, Oregon 97005. Meets annually in Portland, Oregon, Seattle, Washington or Victoria or Vancouver, British Columbia, in early May.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Theodore A. Tristan, Harrisburg Polyclinic Hosp., Harrisburg, Pa. 17105.

Philadelphia Roentgen Ray Society
Secretary, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Norman Williams, Department of Radiotherapy, Montefiore Hospital, 3459 Fifth Ave., Pittsburgh, Pa. 15213. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meetings are held quarterly

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Donald E. Gunderson, 3553 Bayard Dr., Cincinnati, Ohio 45208. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
Secretary, Dr. Ken C. Davidson, St. Luke's Hospital of Kansas City, Kansas City, Mo. 84111. Meets 5 times a year on given dates.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana, Chapter of ACR
Secretary, Dr. Ralph B. Bergerson, 154 Brockenbraugh Ct. Metairie, La. 70005. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic

City at time of State Medical Society meeting and in October or November in Newark, N. J.

RADIOLOGICAL SOCIETY OF RHODE ISLAND, CHAPTER OF ACR
Secretary-Treasurer, Dr. John J. O'Brien, 292 Merrymount Dr., Warwick, R.I. 02888.

RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA
Secretary-Treasurer, Dr. Gladden V. Elliott, 5565 Grossmont Center Dr., Suite 1, La Mesa, Calif. 92041. Meets three times a year, usually October, February and May.

RADIOLOGICAL SOCIETY OF THE STATE OF NEW YORK
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

REDWOOD EMPIRE RADIOLOGICAL SOCIETY
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

RICHMOND COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. M. Pinson Neal, Jr., Medical College of Virginia, 1200 E. Broad St., Richmond, Va. 23219. Meets first Thursday of each month at various hospitals.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. Kenneth E. Robinson, Rochester General Hospital, 1425 Portland Ave., Rochester, N. Y. 14621. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo. Aug. 20–22, 1970.

SAN ANTONIO-CIVILIAN-MILITARY RADIOLOGICAL SOCIETY
Secretary, Dr. Lee F. Rogers, Department of Radiotherapy, Bexar County Teaching Hospital, 4502 Medical Drive, San Antonio, Texas. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

SAN DIEGO RADIOLOGICAL SOCIETY
President-Secretary, Dr. Q. H. Lehmann, 5565 Grossmont Center Dr. Suite 1, La Mesa, Calif. 92041. Meets first Wednesday of each month at the Town & Country Hotel.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Warren M. Russel, Franklin Hospital, Castro & Duboce, San Francisco, Calif. 94114. Meets quarterly at various hospitals (contact Secretary).

SANTA CLARA COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Emory G. West, 285 S. Drive, Mt. View, Calif. 94040. Meets monthly at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

SECTION ON RADIOLOGY, MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
Secretary-Treasurer, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

SECTION ON RADIOLOGY, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Phillip W. Voltz, Jr., 120 Medical Professional Bldg., San Antonio, Tex. 78212. Annual Meeting: Dallas, Tex., Nov. 16–19, 1970.

SECTION ON RADIOLOGY, TEXAS MEDICAL ASSOCIATION
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

SOCIETY FOR PEDIATRIC RADIOLOGY
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles 27, Calif. Annual meeting: Deauville Hotel, Miami Beach, Fla., September 28, 1970.

SOCIETY OF NUCLEAR MEDICINE
Secretary, Dr. James J. Smith, 140 E. 54th St., New York, N. Y. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting: Los Angeles, Calif., June 26–July 2, 1971.

SOUTH BAY RADIOLOGICAL SOCIETY
Secretary, Dr. Emerson C. Curtis, University Dr., Menlo Park, Calif. 94025. Meets second Wednesday of each month.

SOUTH CAROLINA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

SOUTH DAKOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Haakon O. Haugan, 716 Quincy St., Rapid City, S. D. 57701. Meets in spring with State Medical Society and in fall.

SOUTHERN CALIFORNIA RADIATION THERAPY SOCIETY
Secretary-Treasurer, Dr. Aaron G. Fingerhut, 1000 W. Carson St., Torrance, Calif. 90502. Meets quarterly.

SOUTHERN RADIOLOGICAL CONFERENCE
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Annual meeting: Grand Hotel, Pointe Clear, Ala. Jan. 29-31, 1971.

SOUTHWESTERN RADIOLOGICAL SOCIETY
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

TENNESSEE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Lawrence R. Nickell, Maury County Hospital, Columbia, Tenn. 38401. Meets annually at the time and place of the Tennessee State Medical Association meeting.

TEXAS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Herman C. Sehested, 815 Medical Tower, Room 100, 1550 W. Rosedale St., Fort Worth, Tex. 76104. Annual meeting at the Flagship Hotel on Pier, Galveston, Tex.

TRI-STATE RADIOLOGICAL SOCIETY
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.

UPPER PENINSULA RADIOLOGICAL SOCIETY
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

UTAH STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Leon M. Neal, St. Benedict's Hospital, 3000 Polk Ave., Ogden, Utah 84403. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

VERMONT RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Edward A. Kupic, Mary Fletcher Hosp., Burlington, Vt. 05401.

VIRGINIA CHAPTER OF ACR
Secretary-Treasurer, Dr. James S. Redmond, Suite 7, Medical Center, Lynchburg, Va. 24501.

WASHINGTON, D. C., CHAPTER OF ACR
Secretary-Treasurer, Dr. Joan Wohlgemuth, 5021 Seminary Rd., Alexandria, Va. 22311.

WASHINGTON STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Paul S. Paulson, 1001 Broadway Seattle, Washington 98122. Meets quarterly.

WEST VIRGINIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. J. Dennis Kugel, 510-517 Med. Arts Bldg. Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

WESTCHESTER COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Edgar Palmer, 650 Main St., New Rochelle, N. Y. 10801. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert E. Douglas, 1209 S. Commercial St., Neenah, Wis. 54956. Meets twice a year, May and September.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. J. D. Grant, Memorial Hosp., Sheridan, Wyo. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Jorge Vargas Segura, Apartado 5367, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Roberto Calderón, Calle Central Oeste No. 218, Managua, Nicaragua, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de El Salvador
Secretary, Dr. Julio Astacio, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F.
Secretary-General, Dr. Bernardo Serviansky. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Apt. 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. Pierre Archambault, Hôpital Charle Le Moyne, 121 Boul. Taschereau, Greenfield Park, P.Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics.
Honorary Secretary Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada. Annual Congress, to be announced.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, 47 Lincoln's Inn Fields, London, W.C.2, England.

Faculty of Radiologists, Royal College of Surgeons in Ireland
Registrar, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists
Honorary Secretary, Dr. F. Robert MacDonald, *Associate Honorary Secretary*, Dr. Champlain Charest, 1555 Summerhill Ave., Montreal 25, Que., Canada. Annual meeting: Calgary, Alberta, March 15–19, 1971.

Montreal Radiological Study Club
Secretary, Dr. Leonard Rosenthall, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française de Radiologie
Secretary General, Dr. Guy Duckett, 1385 est, rue Jean Talon, Montréal, P.Q., Canada. Meets every third Tuesday from October to April. Annual meeting: Montreal, P.Q., Canada, November 5–7, 1970.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September, through May.

College of Radiologists of Australasia
Honorary Secretary, Dr. T. P. Loneragan, 147 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Fral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Víctor A. Añaños, Instituto de Radiologia, Urqiza 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional de Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Miguel Mario, Céntola, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiología
Secretary-General, Dr. Juan R. Heilbuth Pacheco, Santa Fe 1171, Buenos Aires. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Cxa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigaderio Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Carlos Vildósola, Casilla, 13426 Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Raul Fernández Angula, Calle 43 No. 44-70 Barranquilla, Colombia. Meets last Thursday of each month.

Sociedad Equatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 4569, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dr. Julio Ormeño del Pino, Instituto de Radiología "Cayetano Heredia" Hospital Arzobispo Loayza, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41–110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología de la Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Alejandro Glausiuss, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
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South African International radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa. Second Congress: President Hotel, Johannesburg, September 3-9, 1970.

ABSTRACTS OF RADIOLOGICAL LITERATURE

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ROENTGEN DIAGNOSIS

Genitourinary System

Williams, D. Innes, and Hulme-Moir, I. Primary obstructive mega-ureter. *Brit. J. Urol.*, April, 1970, *42*, 140–149. (From: The Hospital for Sick Children, Great Ormond Street, London, England.)

Obstructive mega-ureter is a disease of early life, although a mild degree of the disease is occasionally seen in adult life. In this series, 70 cases are reported with only 2 presenting after their twentieth birthday. Males predominated by more than 3 to 1. The left side was more commonly affected, but those presenting in the first year of life were likely to be bilateral.

In this series, 18 patients were treated conservatively. Roentgenologically the most common observation was a lack of change in degree of ureteric dilatation. Only 3 showed deterioration. Of reconstructive operations, the nonreflux preventing procedures uniformly did badly. The majority are well treated by a reflux preventing re-implantation of the ureter. In the grossly dilated unilateral case, nephroureterectomy is still the best treatment. In bilateral cases, the outlook is unsatisfactory with any means of treatment.

Roentgenologically one can distinguish between the refluxing and the obstructed ureter by noting retention of contrast medium when the bladder has evacuated. In this case the ureter is frequently indented by the adjacent bladder.

Typical complications of hydronephrosis and pyelonephritis with cortical atrophy usually occur as the condition progresses.—*Arch H. Hall, M.D.*

Weinberg, Raymond W. Extensive schistosomiasis of the ureter: the use of the ileal loop in its management. *Brit. J. Urol.*, April, 1970, *42*, 136–139. (Address: Hon. Clinical Lecturer in Urology, University College of Rhodesia.)

Schistosoma hematobium is well known to cause disease of the lower third of the ureter, but occasionally longer segments may be involved.

The author reports 4 cases in which extensive ureteral schistosomiasis was treated with an ileal loop after ureterectomy. He cautions that conservative management with ambilhar is always preferable initially.

Retrograde pyelography is frequently impossible to perform in advanced cases.

Surgical anastomoses must not involve diseased tissues, and the ureters must therefore be opened at the time of surgery.

Radiologists should be aware of this disease in areas where it is endemic, and in patients who have recently returned from such areas.—*Arch H. Hall, M.D.*

Nervous System

Weinder, Dennis S., and Macnab, Ian. The use of chymopapain in degenerative disc disease: a preliminary report. *Canad. M.A.J.*, June 6, 1970, *102*, 1252–1256. (Address: Dr. Macnab, Division of Orthopaedic Surgery, Wellesley Hospital, 160 Wellesley Street East, Toronto 284, Ontario, Canada.)

Chymopapain is a proteolytic enzyme which shows hydrolysis of disk tissue in *in vitro* studies in disk material removed surgically or from cadavers. It has been considered to catalyze the liberation of protein-polysaccharide from the water-soluble portions of the disk tissue. After initial animal experimental work by several investigators, it has been used clinically since 1960.

The 15 patients selected for study were those with a history of spondylogenic back and leg pain and clinical nerve root impairment or degenerative disk disease noted on routine roentgenograms and those with a myelographic defect.

There were 12 males and 3 females ranging in age from 25 to 70 years. Leg and back symptoms had persisted for 3 months to 20 years and all had abnormal diskograms.

After injection of 0.5 cc. of renografin 60 into the disk for localization, 4 mg. of chymopapain in 1 cc. of water was injected into the nucleus (additional technical details are included in the original paper). Twenty disk levels were injected and there were no adverse reactions. There was complete relief of back and leg symptoms in 6 patients, marked improvement with no leg symptoms but occasional low back pain in 5, and no improvement in 5.

Case histories are included showing the rather wide range of response; 1 patient with a 3 month history of back and leg pain and an extensive myelographic defect had immediate relief of leg pain. Another with a constriction of the oil column and degenerative disk changes showed good results within 3 days. Four patients had a laminectomy because of lack of response to injection and each had a mechanical problem that was corrected surgically.

There is no long-term follow-up of these patients.—*M. E. Mottram, M.D.*

Sweterlitsch, Paul R., Torg, Joseph S., and Watts, Harvey. Malignant hemangioendothelioma of the cervical spine: a case report. *J. Bone & Joint Surg.*, June, 1970, *52-A*, 805–808. (Address: Dr. Sweterlitsch, 1137 Old York Road, Abington, Pa. 19001.)

This is a case report of a rare malignant tumor

producing an appearance of bone destruction in the involved area. There was involvement of the 6th and 7th cervical vertebrae. This is the second reported case in this region.

The patient's symptoms began with pain which increased over several months with progressive neurologic findings including muscular weakness.

Following biopsy, the patient was treated with irradiation but died suddenly following the therapy. —*George L. Sackett, M.D.*

Skeletal System

van der Linden, W. The skiers' boot top fracture: rising incidence, characteristics, treatment. *Acta orthop. scandinav.*, 1969, *40*, 797–806. (From: Department of Surgery, Centrallasarettet, Ostersund, Sweden.)

van der Linden reports from Sweden a marked increase in the percentage of "boot top" ski injuries.

The responsible factors are considered to be due to the increased popularity of downhill skiing; firmer, tighter boot-tops; crowded slopes; and bindings that fail to release with a straightforward thrust or are improperly adjusted. Wet snow which freezes may also prevent binding release.

The 32 fractures reported were mainly in patients with considerable training and under 18 years of age; 13 were 12–14 years old.

"Boot top" fractures occur when the fast forward motion of the skier is abruptly stopped or slowed and the skier falls forward. If the bindings do not release, a transverse fracture occurs at the level of the boot top. The fractures are usually within 6 cm. of the ankle joint. Slight posterior angulation was present in 27 of 32 patients and in more than half there was some forward displacement of the distal fragment usually less than 1 cm. No patient had posterior shift of the distal fragment and the forward shift is assumed by van der Linden to follow attempted weight-bearing. In all cases but 2, both tibia and fibula were fractured.

In most cases reduction was maintained with the foot in moderate plantar flexion.—*M. E. Mottram, M.D.*

Fardon, David F. Fracture of neck and shaft of same femur: report of a case in a child. *J. Bone & Joint Surg.*, June, 1970, *52-A*, 797–799. (From: Department of Orthopedics, University of Missouri Medical Center, Columbia, Mo.)

Fracture of the shaft and neck of the femur is a somewhat unusual occurrence and for this reason, the author reports the case of a 6 year old child who was struck by a truck while walking. The fracture of the shaft was apparent clinically, but the fracture of the neck of the femur was discovered on the initial roentgenographic examination.

The author suggests that the neck fracture be treated with open operation and the shaft fracture nonoperatively.—*George L. Sackett, M.D.*

Støren, H. Operative elevation of the medial tibial joint surface in Blount's disease: one case observed for 18 years after operation. *Acta orthop. scandinav.*, 1969, *40*, 788–796. (From: Coast Hospital, Stavern, Norway.)

The varus deformity of the knee in Blount's disease is caused by the sloping of the medial tibial joint surface and it is aggravated by weight-bearing. In some cases an osteotomy will not adequately correct the deformity and it may be necessary to repeat the osteotomy as well as epiphysiodesis when the patient is about 12 years of age.

Støren reports one case with a marked varus deformity corrected by elevation of the medial tibial joint surface at 11 years of age following 2 unsuccessful osteotomies. The postoperative roentgenograms showed widening of the medial half of the joint space but a near horizontal medial tibial joint surface. The concavity of this surface was believed to be due to the hypertrophied joint cartilage and medial meniscus which has been demonstrated in Blount's disease. Follow-up at 18 years showed no change in the appearance or position of the elevated plateau and there was good mobility and stability.

Successful operative elevation depends on avoiding: (1) aseptic necrosis of the elevated fragment; (2) disturbance of the epiphyseal line; and (3) arthritis secondary to poor articulation of the joint surfaces.—*M. E. Mottram, M.D.*

Singh, Manmohan, Nagrath, A. R., and Maini, P. S. Changes in trabecular pattern of the upper end of the femur as an index of osteoporosis. *J. Bone & Joint Surg.*, April, 1970, *52-A*, 457–467. (From: Department of Orthopaedic Surgery, Medical College Hospital, Rohtak, Haryana, India.)

The authors investigated 35 patients to find out if the changes in the trabecular pattern of the upper end of the femur would be useful as an index to systemic osteoporosis. They correlated the material with roentgenographic and clinical findings and with iliac crest biopsy specimens that were examined and graded for osteoporosis according to the method described by Beck and Nordin.

The roentgenographic changes of the principal tensile and principal compressive trabeculae in the upper femur were shown to correlate well with the presence of systemic demineralization on the basis of osteoporosis.

The authors devised a 6 grade scale, the lowest numbers of which indicated the most demineralization.

The diagrams and roentgenographic reproductions in the article document a gradual loss of the compressive and then of the tensile trabeculae with advancing pathologic change.

The authors were consistently able to grade the osteoporosis present from roentgenograms made of the hip in slight internal rotation, and this reproducibility led to their conclusion . . . that this method offers advantages over either bone densitometry or analysis of spinal roentgenograms. One of the authors has shown that this is applicable to both Americans and Indians.

It is pointed out that the method should be universally applicable because the grading of the osteoporosis is based upon bone structure, rather than bone quality.—*Robert P. Andrews, M.D.*

O'Connor, Brian T., Steel, W. M., and Sanders, R. Disseminated bone tuberculosis. *J. Bone & Joint Surg.*, April, 1970, *52-A*, 537–542. (From: The Nuffield Department of Orthopaedic Surgery, University of Oxford, Oxford, England.)

The rare disorder, disseminated bone tuberculosis, has been the subject of some confusing terminology in the past, and the current term is used to describe this rare condition when it is considered in the contemporary literature.

The typical lesions are peripheral in distribution in children, but more likely to be in the axial skeleton in adults, and are characteristically small ovoid lesions lying in the long axis of the bone with some well defined margins and sclerosis. The title in children is multiple pseudocystic tuberculosis of bone.

The authors report a case of disseminated bone tuberculosis in a 30 year old Punjabi postal sorter who had been treated for overt pulmonary and meningeal tuberculosis 3 years earlier and had apparently recovered. When the diagnosis of disseminated bone tuberculosis was made, antituberculous chemotherapy was begun and the patient did well subsequently.

The diagnosis of disseminated bone tuberculosis can be difficult when there is no evidence of overt pulmonary or systemic tuberculosis, but local biopsy and appropriate laboratory tests should solve the problem.

It is also mentioned that hypercalcemia is sometimes seen in the adult form of the disease.—*Robert P. Andrews, M.D.*

Goldman, Marvin, Williams, R. Jean Romer, and Bulgin, Marie S. Application of ^{18}F to image ^{226}Ra-induced bone lesions. *J. Nuclear Med.*, May, 1970, *11*, 208–213. (From: University of California, Davis, Calif.)

Varying amounts of Ra^{226} were injected at intervals in several hundred Beagles to study the toxicity of bone-seeking radionuclides. Ra^{226} and its daughter products are deposited to a large extent in regions of the skeleton undergoing remodeling at the time of high plasma Ra^{226} levels. A diffuse contamination of the skeleton occurs with redistribution of the original deposits. The short-ranged alpha particles produced the greatest damage.

The authors used F^{18}, a bone seeker, to study the correlation between bone blood flow and some roentgenographically demonstrable lesions. F^{18} is a positron emitter with a half life of 1:87 hours and produces photons of 510 kev. The F^{18} uptake by the skeleton is a function of exchangeable mineral sites and of the efficiency of bone blood flow at these sites.

A dose of 50 to 100 μc of F^{18} was administered to adult Beagles previously treated with Ra^{226}. Positron scintillation camera pictures were made of the skeleton 1 to 2 hours after injection; 1 to 5 minute exposures were made. Hot spots were found over a variety of lesions. Decreased F^{18} intensity in the appendicular joints and vertebral column is an indication of impaired blood flow.

Five cases are reported showing various types of bone lesions. Ra^{226} induced lesions or pathology include trabecular coarsening, endosteal and periosteal bone production and resorption, spontaneous fractures, osteolytic lesions and skeletal neoplasias. Some of the lesions regressed. Hot spots preceded radiographic demonstration of some of the lesions. The presence of hot spots not only at tumor sites, but in fracture and local periosteal reaction, is compatible with an increased vascularity in bone undergoing remodeling and resorption. The hot spots probably appear prior to roentgenographic visualization of the lesions and are not necessarily demonstrable in long-standing lesions.

F^{18} scanning in selected cases is an adjunct to diagnostic radiology. It is possible to distinguish active from inactive lesions with some old fractures being the exception. Scanning may visualize questionable primary tumors prior to roentgenographic demonstration and possibly before metastatic spread.—*Charles W. Cooley, M.D.*

Blood and Lymph System

Carroll, S. E., and Wilkins, W. W. Two cases of brachial plexus injury following percutaneous arteriograms. *Canad. M. A. J.*, April 25, 1970, *102*, 861–862. (Address: S. E. Carroll, M.D., Department of Surgery, St. Joseph's Hospital, London, Ontario, Canada.)

Two cases of brachial plexus nerve injury are reported that occurred subsequent to percutaneous angiography.

The first patient was a young man that suffered nerve function loss and sensation loss in the right hand subsequent to his surgically proven hematoma within the neural vascular bundle of the brachial plexus. A similar case occurred in a 58 year old patient and was also due to a surgically proven hematoma within the neural vascular bundle. Much of the function was recovered in this patient and late changes were minimal.

The authors point out that anatomically the axillary artery is an infrequent site of atheroma and has excellent collateral circulation so that it is an ideal site for percutaneous puncture. It is contained within the axillary sheath, however; and hematoma is very likely to cause nerve root compression so that assiduous postoperative pressure should be properly maintained and close follow-up by the nursing service and the patient should be carried out to detect early hematoma.

The treatment is surgical and often times fairly simple if carried out early.—*Robert P. Andrews, M.D.*

Cohen, Berry H., Chinitz, Joel L., Soll, Kenneth H., Ramirez, Osvaldo, Onesti, Gaddo, Kim, Kwan E., and Swartz, Charles. Radiological contrast study of arteriovenous dialysis cannulae. *Radiology*, March, 1970, *94*, 603–606. (From: Departments of Medicine and Radiology, Hahnemann Medical College and Hospital, Philadelphia, Pa.)

Cannula failure remains the most vexing problem in maintenance hemodialysis programs.

Following heparinized saline flushing, 27 arteriographic and 23 venographic studies were done in 20 patients with shunt problems.

Rapid injection of 20 ml. of a 15 per cent solution of methylglucamine diatrizoate resulted in no morbidity and no complications.

Radiologic abnormalities were demonstrated in each patient. These included a benign beaded appearance ("standing waves"), circumferential arterial stenosis, venous segmental narrowing and thrombosis of artery, vein or shunt.

A number of abnormalities were also found in 20 patients without clinical shunt problems.

The radiologic studies were helpful in declotting procedures.—*William H. Roush, M.D.*

RADIOISOTOPES

Lukensmeyer, W. W., Hege, J. H., Theil, G. B., and Wilson, W. R. Calcium and phosphorus metabolic studies in triiodothyronine-induced hypermetabolism. *Am. J. M. Sc.*, April, 1970, *259*, 282–291. (From: Sections of Clinical Pharmacology and Nephrology, Cardiovascular Research Laboratories, Departments of Internal Medicine and Pharmacology, University of Iowa, College of Medicine, Iowa City, Iowa.)

The effects of triiodothyronine (T_3) on calcium and phosphorus balance in 8 normal males receiving diets high in calcium and phosphorus were studied. Renal clearances and tubular resorption of phosphorus were also studied to determine the influence of T_3 on these aspects of renal function.

Special balanced diets were given. Additional calories were added during the induced hypermetabolic periods to minimize weight loss. The amounts of calcium and phosphorus were kept constant. Triiodothyronine was the only drug given. The study lasted 54 days for each subject. The initial control period was for 20 days. Triiodothyronine (400–500 μg. per day) was given for 17 days and the last 17 days was a control period. Each control period was divided into 3 subperiods of 4 to 8 days for convenient pooling of stool specimens. Body weight, urine volume and urinary calcium, phosphorus, creatinine and sodium were measured daily. Fecal calcium and phosphorus were determined in the pooled specimens. Plasma levels of calcium, phosphorus, creatinine and potassium were measured. Renal clearances of phosphorus and creatinine were determined. The tubular resorption of phosphorus was determined by subtracting the urinary excretion rate from the filtered load of phosphorus. The creatinine clearance was used in computing the filtered load. The value for the tubular resorption was divided by the filtered load and multiplied by 100 to give the per cent tubular resorption of phosphorus.

Triiodothyronine produced hypermetabolism as evidenced by increases in sleeping pulse rate and oxygen consumption, and decreases in serum cholesterol and body weight. Increased irritability, decreased heat tolerance, tremor and nervousness were noted in each subject. The clinical manifestations of hyperparathyroidism disappeared within 10 days after stopping triiodothyronine. The oxygen consumption, sleeping pulse rate and serum cholesterol levels also returned to normal in the same time.

Triiodothyronine produced an increase in urinary calcium and phosphorus excretion rate. There was no change in the plasma values of sodium, potassium and calcium. There was a rise in the plasma phosphorus and a decrease in the plasma creatinine. There was an increase in creatinine and phosphorus clearances and a transient decrease in the tubular resorption of phosphorus. There was no change in the net calcium or phosphorus balance.

The study showed that triiodothyronine significantly increases urinary calcium and phosphorus excretion rates and that an adequate dietary intake

of calcium and phosphorus can protect the body against negative balances of these essential elements.—*Charles W. Cooley, M.D.*

Kirkham, Bruce C., Tyson, Ian B., and Wirtanen, George W. Comparison of ^{131}I-macroaggregated liver scanning and selective hepatic arteriography. *J. Nuclear Med.*, May, 1970, *11*, 196–202. (From: University of Wisconsin, Madison, Wisc.)

Clinical data and an animal experiment were used to compare selective hepatic arteriograms and I^{131}-MAA and Au^{198} scans. It is known that I^{131}-MAA injected into percutaneously placed intraarterial hepatic catheters in patients with liver metastases shows positive images of abnormal areas of increased uptake which correlate with the negative images produced by the Au^{198} scans.

Twenty-one patients with percutaneous transbrachially placed catheters receiving continuous hepatic artery chemotherapeutic infusions were studied with Au^{198} liver scans, selective hepatic arteriograms and I^{131}-MAA liver scans. A dose of 6 μc/kg. body weight of I^{131}-MAA was diluted to a volume of 20 cc. with normal saline and injected through the infusion catheter, and followed by 20 cc. of normal saline. The liver was scanned immediately. Selective hepatic arteriography was done using 40 cc. of 60 per cent meglumine diatrizoate.

Metastatic replacement of liver tissue was indicated by decreased uptake on the Au^{198} scans, while I^{131}-MAA scans showed an increased uptake in these same areas. These areas of increased uptake on the I^{131}-MAA scans corresponded to the arteriographically neovascular areas which were more vascular than the normal areas of the liver. There were 4 cases with normal Au^{198} scans and with positive arteriograms and I^{131}-MAA scans. The abnormal areas were in the region of the liver hilus. There was an increased uptake of I^{131}-MAA in the left lobe of the liver in 3 cases in which there was no neovascularity as shown in the arteriograms and the difference was thought to be due to less contrast medium reaching the left lobe through a smaller hepatic artery and that a relatively small amount of radioactivity can be detected easier in a large vascular organ which requires larger amounts of contrast material to be visualized arteriographically.

I^{131}-MAA particles produce a capillary blockade. A positive liver scan could be produced by the greater concentration of isotope in areas of neovascularity due to tumor and the absence of reticuloendothelial cells resulting in a reduced disposal rate in the tumor compared to disposal rates in normal liver tissue. The ability to detect tumor is increased as compared to the Au^{198} scans, especially in the region of the liver hilus.

The single dog experiment in which an "irritant granuloma" was produced (sephadex G-25 suspension injected into the liver) showed a decreased uptake in the Au^{198} liver scan, an increased uptake in the I^{131}-MAA liver scan and hypervascularity in the selective hepatic arteriograms. Neovascularity with foreign body reaction was found in the pathologic specimen.

I^{131}-MAA injected into the hepatic artery via a percutaneous transbrachial catheter produced superior scans for tumor detection in the liver. The main disadvantage is the arterial catheterization.—*Charles W. Cooley, M.D.*

Loken, Merle K., Ponto, Richard A., and Kush, George S. Quantification of image studies obtained with a scintillation (Anger) camera. *Radiology*, June, 1970, *95*, 505–516. (From: Division of Nuclear Medicine, Department of Radiology, University of Minnesota Hospitals, Minneapolis, Minn.)

The authors present their experience in developing a system of quantifying data from images obtained with a scintillation camera, to display it as graphic analyses or contour plots. A number of dynamic and static studies so handled are discussed to illustrate the usefulness of this approach.

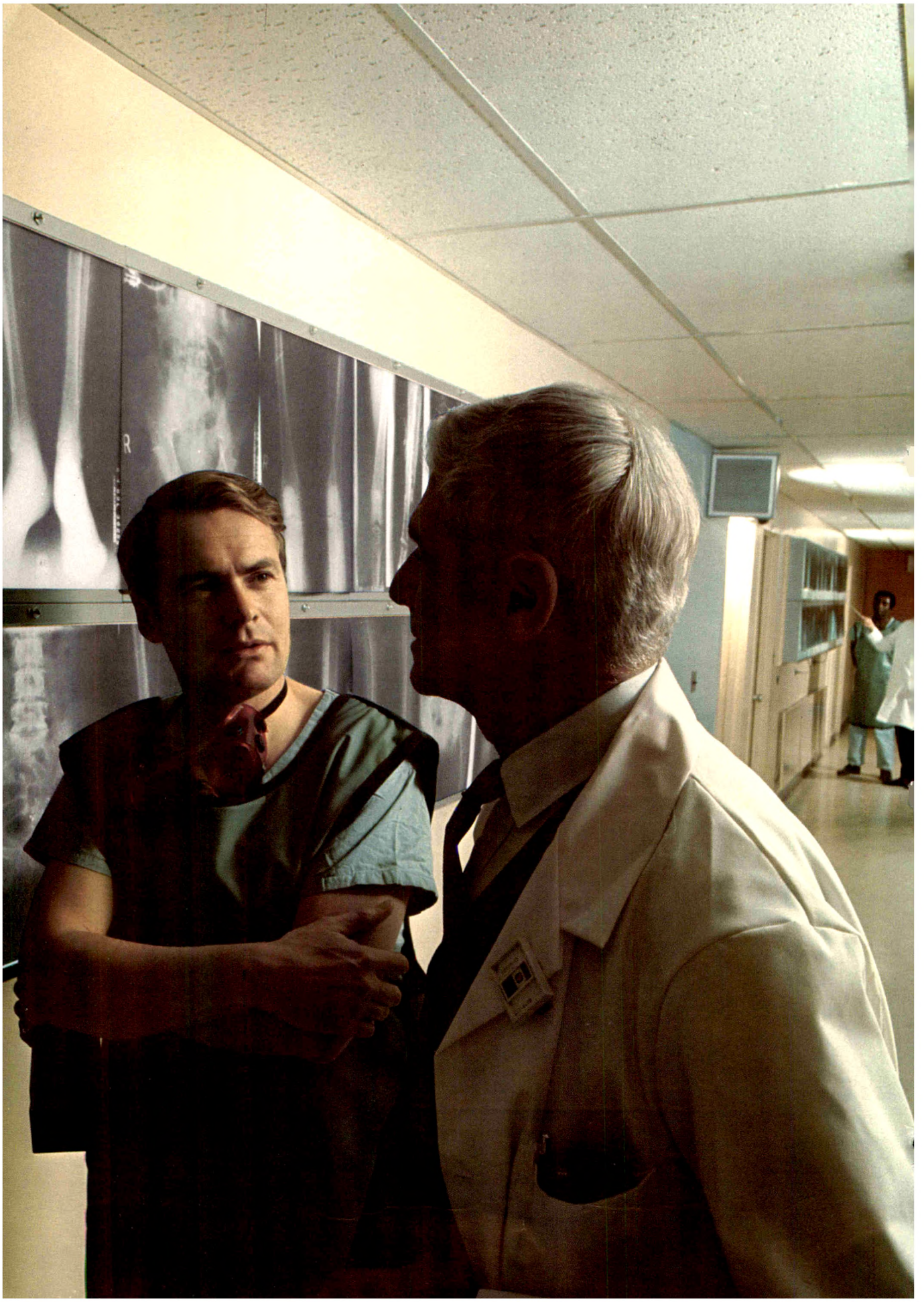
The X- and Y-analogue signals of the scintillation camera system which indicate the position of gamma-ray photon interactions in the camera crystals are passed through a dual analogue-to-digital converter. The resultant digital information goes to a 1,600 word memory multichannel analyzer where it is properly channelled into a 40×40 grid. Depending on the type of study being performed, the memory contents are transferred to high-speed synchronous magnetic tape in computer-compatible format at varying time intervals from a few seconds to several minutes. A Fortran IV program on a CDC-3300 series computer accepts the data from the magnetic tape, calculates the contour positions and plots the coordinates. A Calcomp incremental plotter produces the actual map of the contour as a 2 dimensional display of activity as a function of position in the human anatomy.

The iso-intensity contour map of the quantitative distribution of radioactive tracers in human organs during dynamic and static studies appears to lend itself well to interpretative processes. "The mathematical procedures utilized in developing the contours appear to emphasize important diagnostic information that may not readily be appreciated on images."—*F. C. Petty, M.D.*

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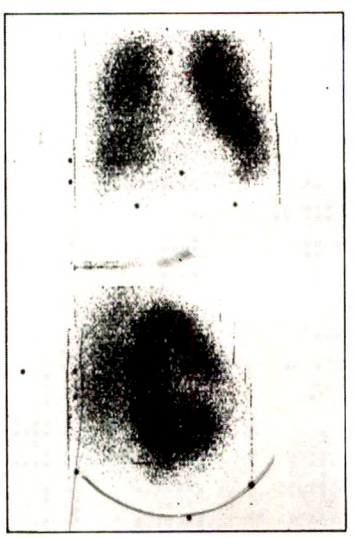
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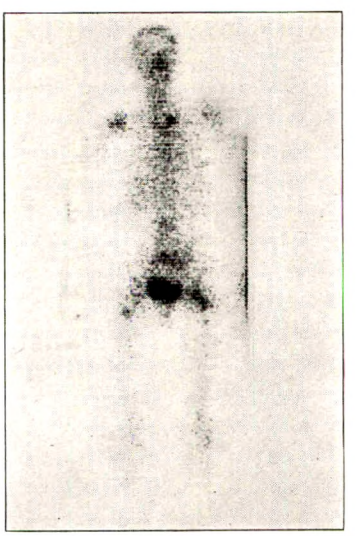
ohio-nuclear, inc.

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think scan speed



AP & Right Lateral 84FD
(opposing views done simultaneously not shown)
Minified 2:1 Lung Scan
Dose: 300μCi Radionuclide: ^{131}I (MAA)
Scan Speed: AP—PA 380 cm./min.
RL—LL 285 cm./min.
TOTAL SCAN PROCEDURE TIME:
25 min. (4 views)
Courtesy of Ernest G. Smith, Jr., M.D.
Crawford W. Long Hospital, Atlanta, Ga.

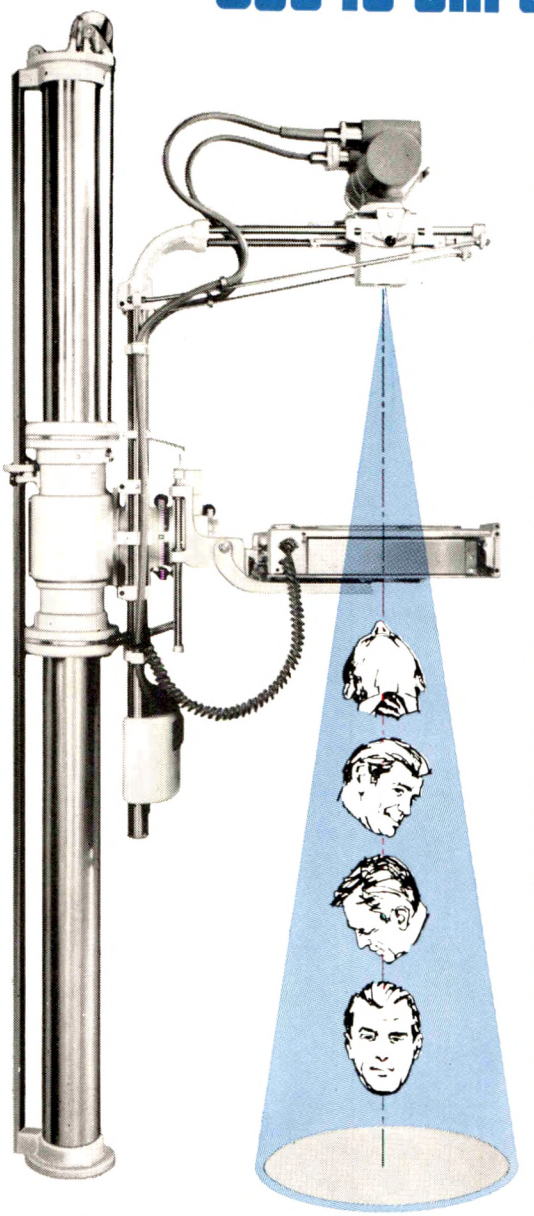


AP 84HD
(opposing view done simultaneously not shown)
Minified 5:1 Whole Body Bone Scan
Dose: 3 mCi Radionuclide: ^{87m}Sr
Post Injection Time: 4 hrs.
Scan Speed: 750 cm./min.
TOTAL SCAN PROCEDURE TIME:
25 min. (2 views)
Courtesy of Univ. of Iowa, Dept. of Radiology
Section of Nuclear Medicine, Iowa City, Iowa



AP & Right Lateral 84FD
Minified 2:1 Liver Scan
Dose: 1.5 mCi Radionuclide: ^{99m}Tc (SC)
Scan Speed: 300 cm./min.
TOTAL SCAN PROCEDURE TIME:
10 min. (2 views)
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Radiographic Head Stand

Excellent Film Detail
Easy Positioning
Accurate Alignment

This versatile stand is designed for a broad range of skull and sinus examinations, and special studies including pneumoencephalography and ventriculography. In addition, it serves as a support unit to regular x-ray facilities, handling chests, extremities and erect gall bladder views. With the body section attachment, it provides for both erect and recumbent tomographic studies.

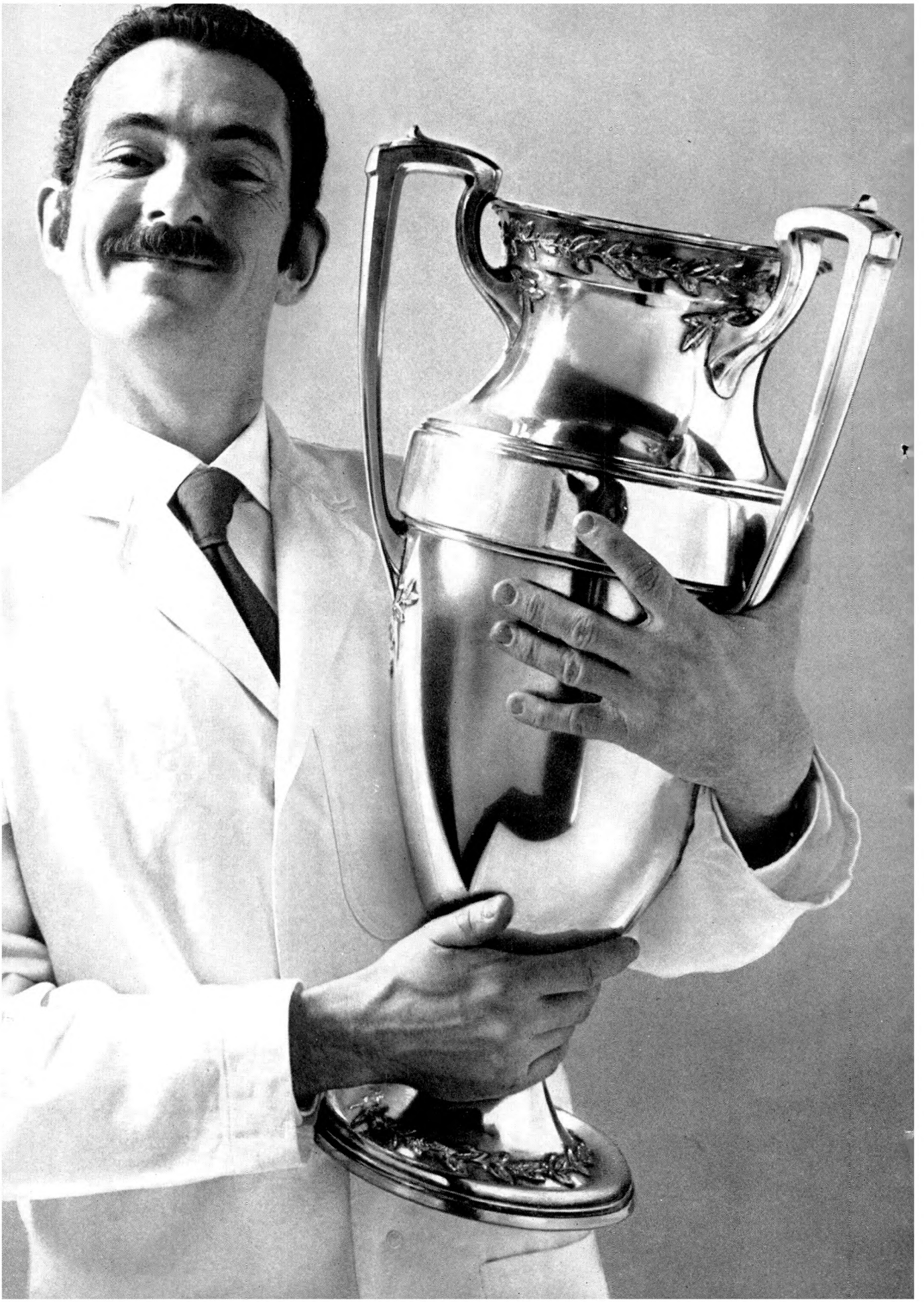
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Res-O-Mat™ Free Thyroxine (FT4) Index— easiest way to a more reliable determination of thyroid function

The Res-O-Mat FT4 Index comes closest to being the most reliable assessment of thyroid function with the easiest procedure. The combined use of Mallinckrodt's Res-O-Mat T3 and Res-O-Mat T4 Tests gives an FT4 Index that compensates for conditions of pregnancy, estrogen medication, and other factors affecting this measurement.

It is so much easier and time-saving because the Res-O-Mat T3 and T4 strips simplify procedures. In the T3 measurement the strip eliminates all pipetting except initial transfer of serum to the vial. There is no washing, no critical temperature control, and the T4 procedure requires no evaporation or ice bath. There are fewer counting steps. Merely rotate the vials, remove the strips, and count the serum directly.

The Res-O-Mat FT4 Index is the ratio of Res-O-Mat T4 and T3 values. The FT4 index has been shown to have a high degree of correlation with the blood level of free thyroxine.* And this simple Res-O-Mat FT4 method makes this determination a routine laboratory procedure.

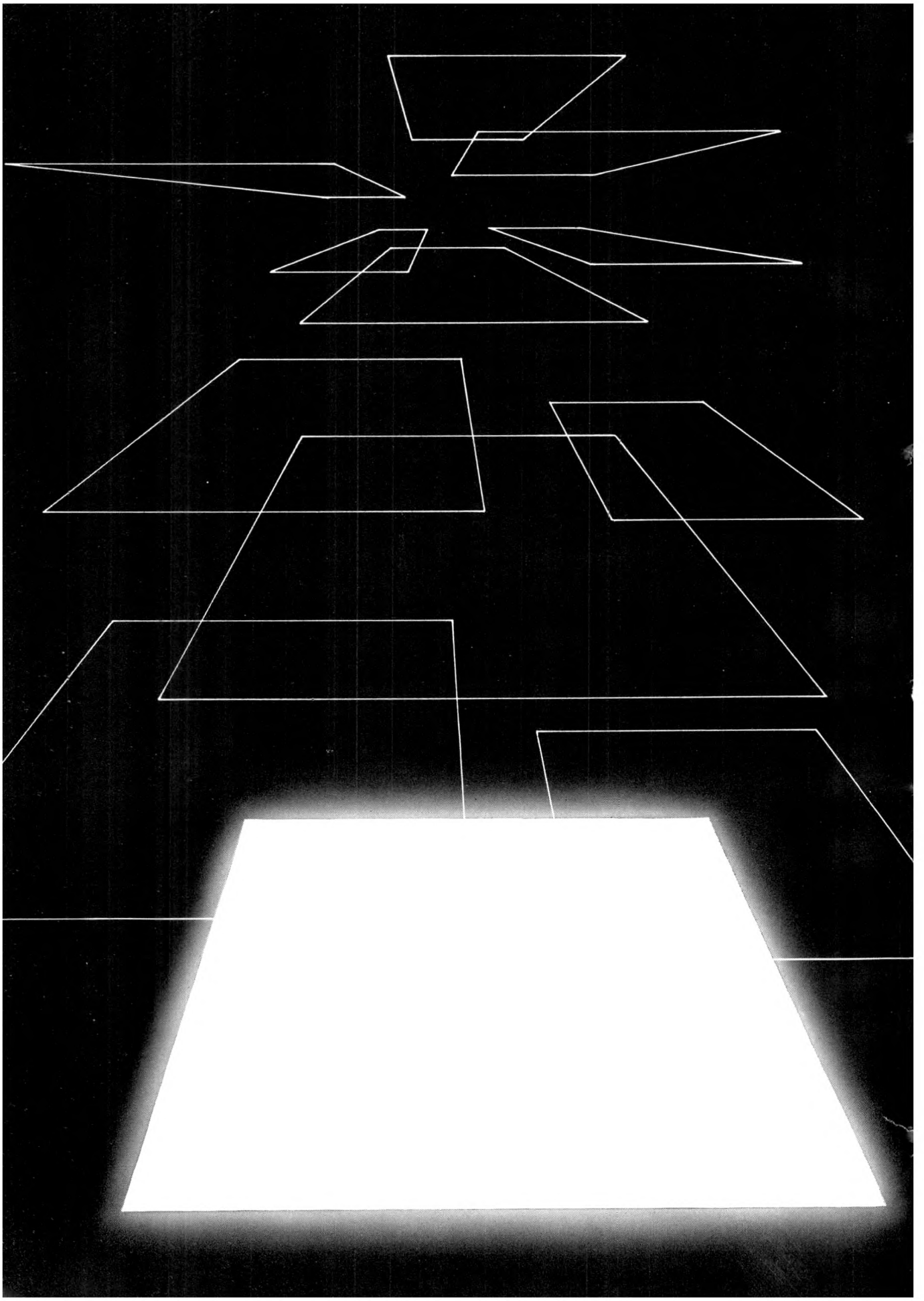
Send for complete information on the Res-O-Mat FT4 Index, or contact your Mallinckrodt sales representative.

*F. Clark and D. B. Horn, Journal of Clinical Endocrinology, 25:39-45, Jan. 1965.

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Ask for a demonstration. Your Du Pont Technical Representative can bring in a cassette with Hi-Plus screens installed. He can help you compare the results in your own operation.

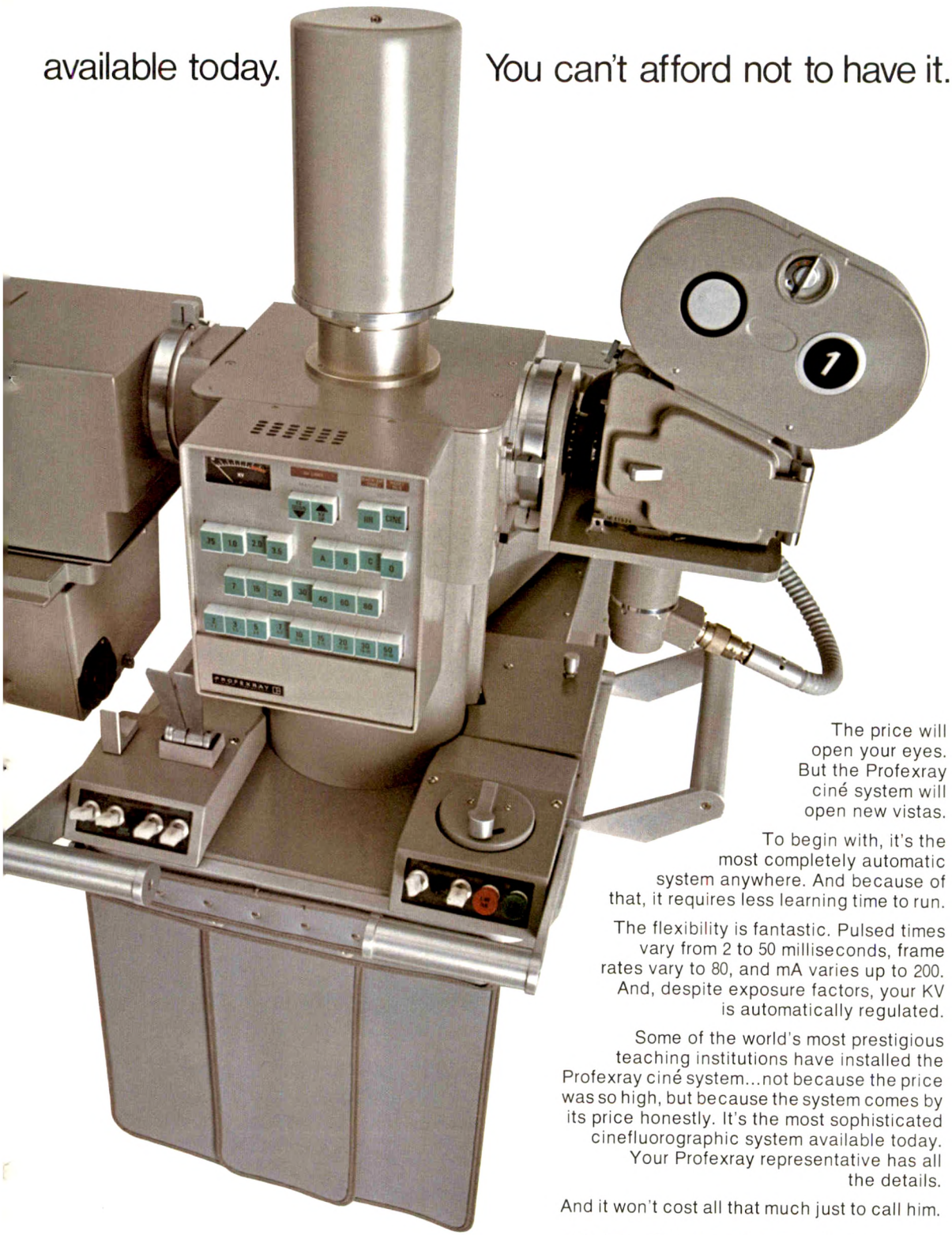
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No other company serving the medical community encompasses as broad a reach of products and services as does Picker. From sophisticated radiologic, radioisotope scanning, and ultrasound diagnostic instruments to patient gowns. From cobalt therapy equipment to catheters. From intensive cardiac care monitoring equipment to radiopaques.

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But what does such a product panorama mean to the user? Is this just diversity for diversity's sake?

Hardly. Product diversity leads to more than a thick catalog: it makes *systems* possible. Individual products can begin to coalesce into interrelated, coordinated, compatible *systems.* This voids the traditional user problem of integrating separate products of dubious compatibility. Likewise, it voids the need for multiple servicemen working on multiple products with no one accepting the total responsibility for the *only* thing the user really cares about: an efficient, functioning, problem-solving system. An unobtrusive system, if you will.

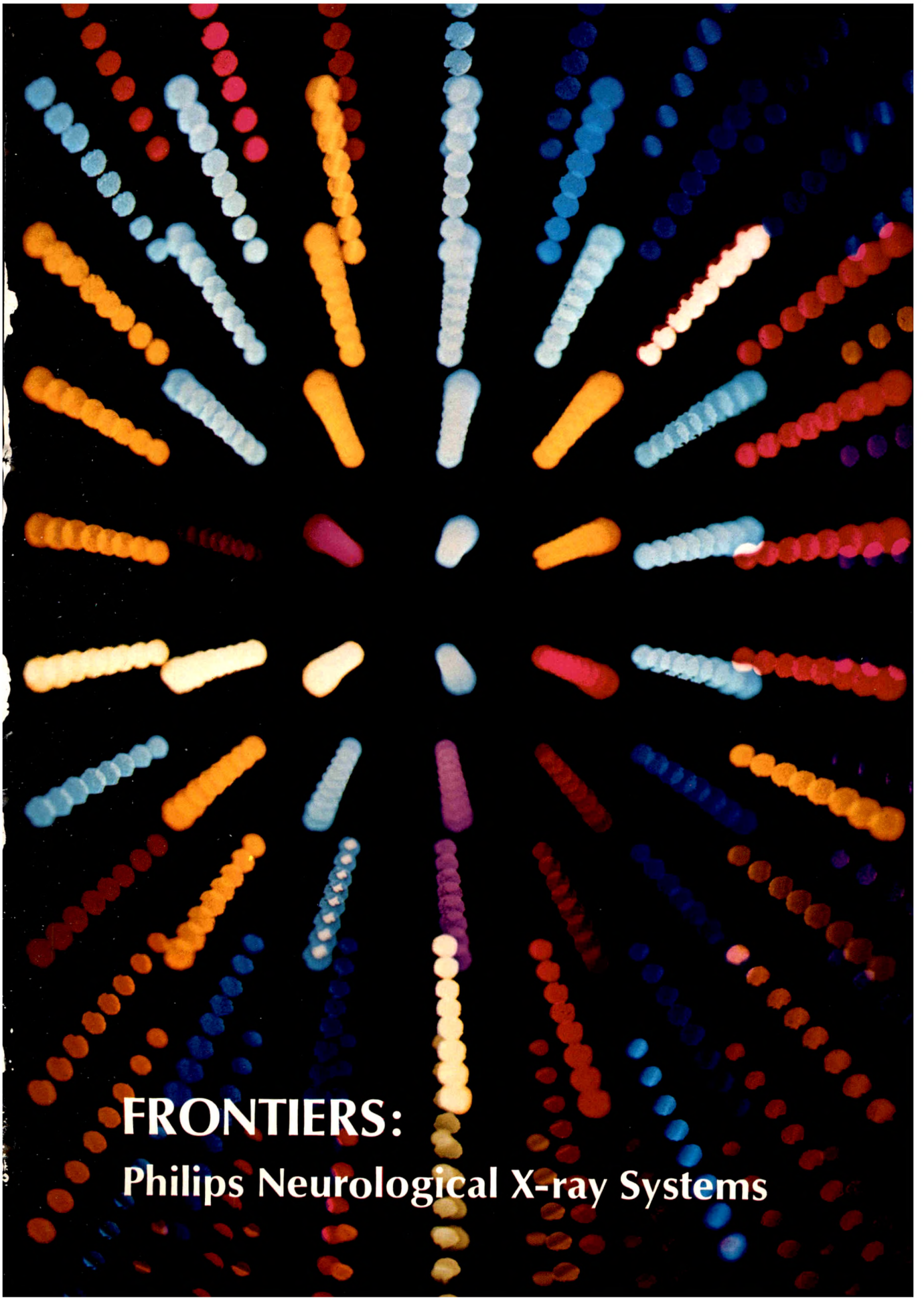
So we acknowledge the user's desire for *total* solutions to problems rather than for a miscellany of individual products. Accordingly, we point ourselves in this direction:

> *To design and build complete systems that will satisfy the user's needs for information, simplicity, practicality, economy, and reliability.*
> *To test those systems as systems*
> *To install and to instruct as needed.*
> *And, finally, to take the sole responsibility for servicing everything we sell.*

Picker, as the "single source responsibility" company, doesn't back away from guaranteeing your *total* satisfaction.

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FRONTIERS:
Philips Neurological X-ray Systems

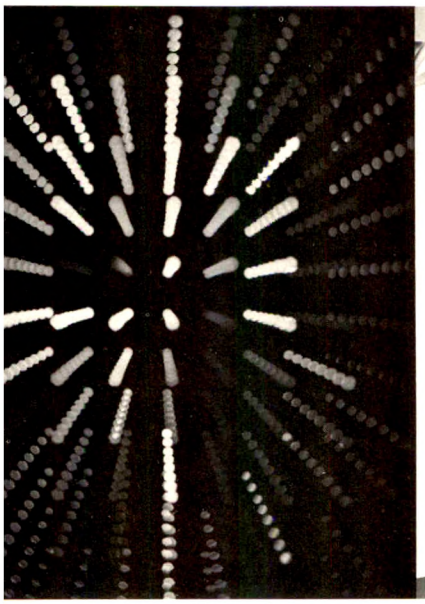
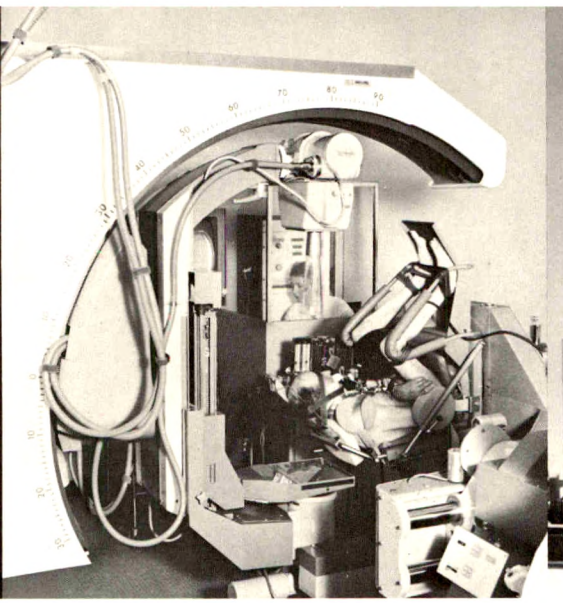
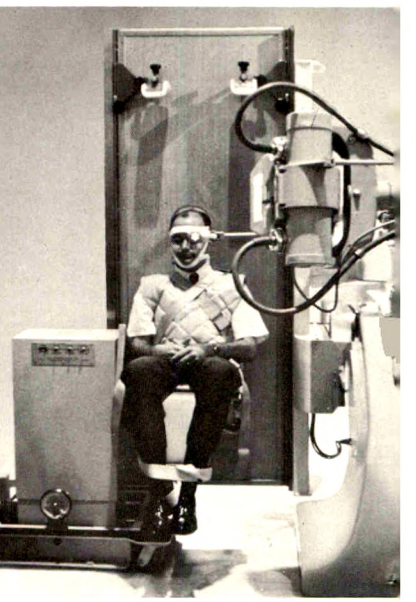


Photo by Erich Hartmann



Diagnost N



Polytome U

A MATTER OF LIFE AND TIME

Brain damage can irreversibly change the course of a human life. Recognizing that the physician requires fast, full information in planning a course of treatment, Philips developed the Diagnost N and Polytome U, for precise, radiographic examination. Each of these systems can help determine the precise extent of damage suffered after a neurological accident.

The Diagnost N is a completely self-contained unit for study of the skull and associated neurological procedures. It can be used for TV fluoroscopy, standard radiography, linear tomography, cinefluorography and video recording. The versatile neuro chair completes an isocentric system allowing examinations in any patient position and at any x-ray beam angle. These capabilities afford ease of patient handling and, in many instances, significant reduction in procedure time.

The Polytome U offers unlimited tomographic choice—linear, elliptical, circular or hypocycloidal—as well as zonography. It minimizes unwanted detail by blurring and gives precision graphic recording down to 1mm 'cut' of a specific area of study.

Both the Diagnost N and Polytome U are extremely high-speed units of great versatility. Each are of incalculable aid when detailed information, and speed in getting it, are vital in determining treament.

To find out more about how these systems can be of benefit to you, contact your Philips Medical Systems dealer or write:

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introduced by Squibb...
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■ Reduced Protein Content

Squibb has reduced the amount of protein by 50% while maintaining good lung scans.

■ Reduced Supernatant Activity

Squibb has sharply reduced the amount of radioactivity in the supernatant, decreasing the possibility of liver interference with the lung scan.

■ Reduced Unbound Iodine

Squibb has substantially reduced the amount of unbound iodine 131, effectively reducing the problem of blood background radioactivity.

Albumotope—LS—a good example of Squibb leadership in radiopharmaceutical research and development. Some people won't leave "well enough" alone.

Contraindications: Radiopharmaceuticals should not be administered to pregnant women or to persons under the age of 18 years unless the indications are very exceptional. Because iodide is excreted in human milk, aggregated radioalbumin should not be administered to nursing mothers.

Side Effects and Precautions: There have been no reported cardiovascular or other untoward effects attributable to Albumotope—LS. Extensive clinical use of Albumotope—LS has not borne out the hypothetical possibility that particles of large size might induce deleterious cardiovascular or cerebrovascular effects. The product appears to possess no antigenic properties. One patient with a known history of angioneurotic edema, who had been given Lugol's solution in conjunction with aggregated radioalbumin similar to Albumotope—LS, developed urticaria.

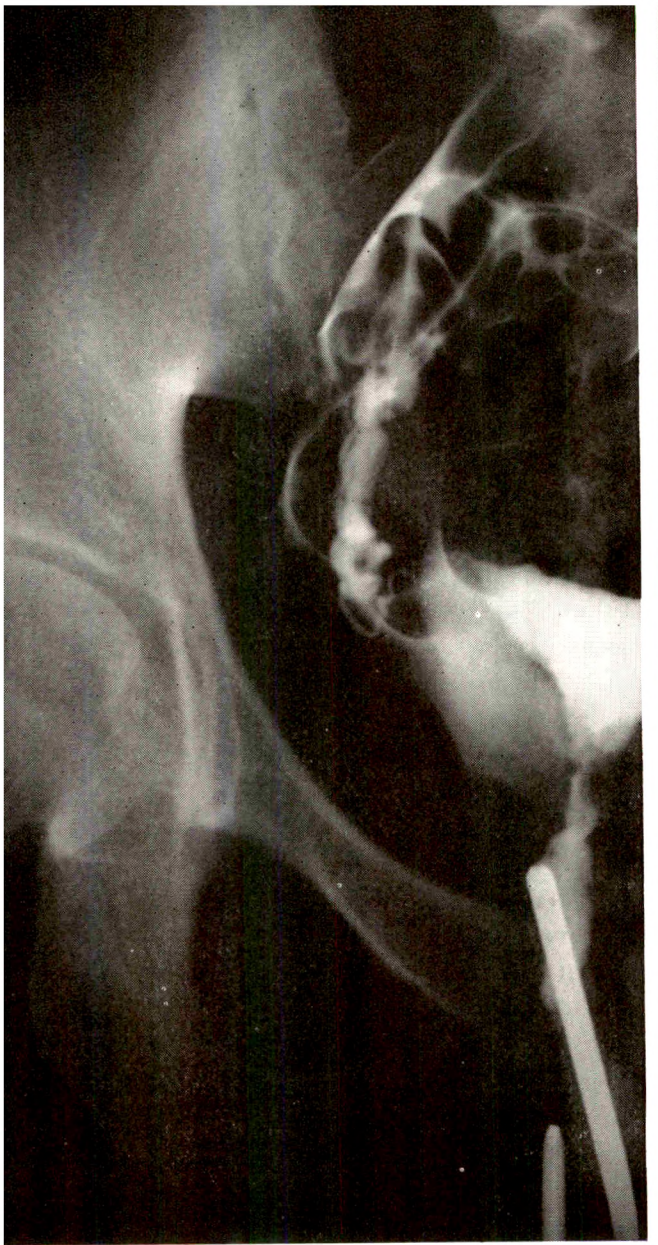
For full prescribing information, see package insert.

Available: As a sterile, nonpyrogenic, aqueous suspension. Each cc. contains approximately 0.5 mg. aggregated human serum albumin labeled with iodine 131. Not less than 90% of the aggregates are between 10 and 90 microns and none are more than 150 microns in size. The preparation also contains 0.9% (w/v) benzyl alcohol as a preservative. The potency ranges from 250 to 450 microcuries per cc. on date of assay.

Medotopes®



Squibb Division of Nuclear Medicine
New Brunswick, New Jersey 08903



Hysterosalpingogram with Salpix Contrast Medium.



24-hour postinjection x-ray of same patient.

now you see it

now you don't

With Salpix as your contrast medium, your gynecologic patient needn't undergo the 24-hour postinjection x-ray usually required with iodized oils. Water-soluble Salpix leaves virtually no irritating radiopaque residue to produce foreign-body reactions, yet it provides the viscosity needed for adequate visualization.

Contraindications: Contraindications to hysterosalpingography include the presence of severe vaginal or cervical infections, existing or recent pelvic infection, marked cervical erosion or endocervicitis, and pregnancy. The procedure is contraindicated during the immediate pre- or post-menstrual phase.

Sensitivity Test: If indicated in the patient's history, an intracutaneous skin test or sublingual absorption observation may be done with 0.1 cc Salpix.

Warning: Not for intravenous use.

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CONTRAST MEDIUM

Salpix

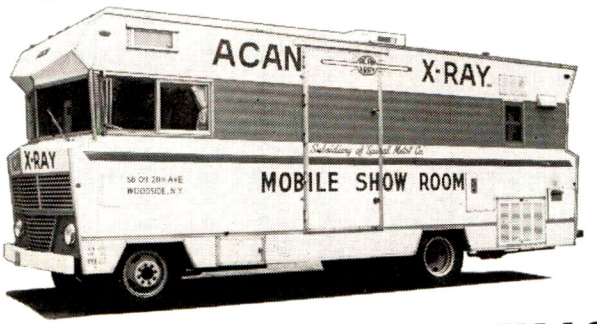
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Acan's Mobile Show Room is traveling around the United States to demonstrate the Acan Model RC 1100 Mobile Type Condenser Discharge X-Ray Unit. This unique piece of equipment is designed to assure high speed, high quality radiographs.

Rather than talk about it, however, Acan is sending its Mobile Show Room on a tour of the nation to demonstrate the unit. You'll have an opportunity to see the RC 1100 in operation, and to have all your questions answered at first hand.

In addition to the Condenser Discharge X-Ray Unit, the Mobile Show Room will feature other Acan products including the Dri-Glov Protective X-Ray Glove Dryer, the Naca Self-Threading Compact Film Processor and the Acan Collimator.

Look for the Acan Mobile Show Room in your community. Meanwhile, if you'd like information about any Acan product or service, please let us know.

ACAN X-RAY

a subsidiary of

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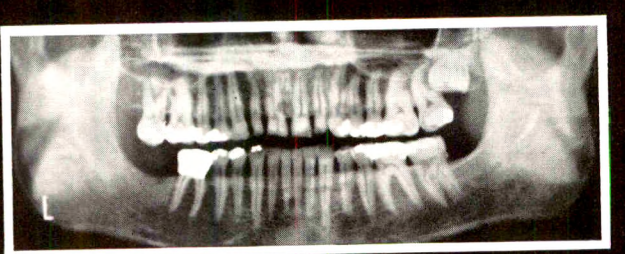




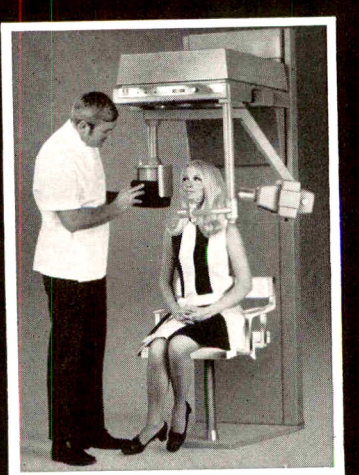
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Ask your GE representative for details or w General Electric Company, Medical System Department, Milwaukee, Wisconsin 53201.

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All macroaggregated serum albumins are not the same. Macroscan-131 offers all 5 of these benefits:

- **Uniformity of particle size distribution**
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Macroscan-131 is aseptically prepared and non-pyrogenic. It is ready to use and should not be heated prior to use.

INDICATIONS: For scintillation scanning of the lungs to evaluate total, unilateral, and regional arterial perfusion of the lungs.

WARNINGS: Radio-pharmaceutical agents should not be administered to pregnant or lactating women, or to persons less than 18 years old, unless the information to be gained outweighs the hazards. There is a theoretical hazard in acute cor pulmonale, because of the temporary small additional mechanical impediment to pulmonary blood flow. The possibility of an immunological response to albumin should be kept in mind when serial scans are performed. If blood is withdrawn into a syringe containing the drug, the injection should be made without delay to avoid possible clot formation.

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MACROSCAN®-131 AGGREGATED RADIO-IODINATED (I^{131}) ALBUMIN (HUMAN)

Each milliliter contains 1 to 3 mg. aggregated human serum albumin labeled with iodine 131, with benzyl alcohol, 0.9%, as preservative. Radioactivity is usually between 800 and 1300 microcuries per ml. on first day of shipment. For full prescribing information, see package insert.

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Yes, we *guarantee* to prove new 3M Brand Type R Medical X-Ray Film can give you better detail than the film you are now using. Radiologists who have evaluated Type R agree that "you see more on Type R." And you see it with 90-second processing. In fact, many radiologists tell us that Type R gives them significantly *better* detail than the 3½ minute films they were using. The secret is in our new synthetic polymer that minimizes loss in detail normally experienced in rapid processing. Type R provides the same superior performance in all processing cycles. Make us prove it really gives you *better* detail than your present film. Send this "guarantee" with your name and address to your local Profexray Representative or to 3M. We'll arrange an evaluation that will convince you.

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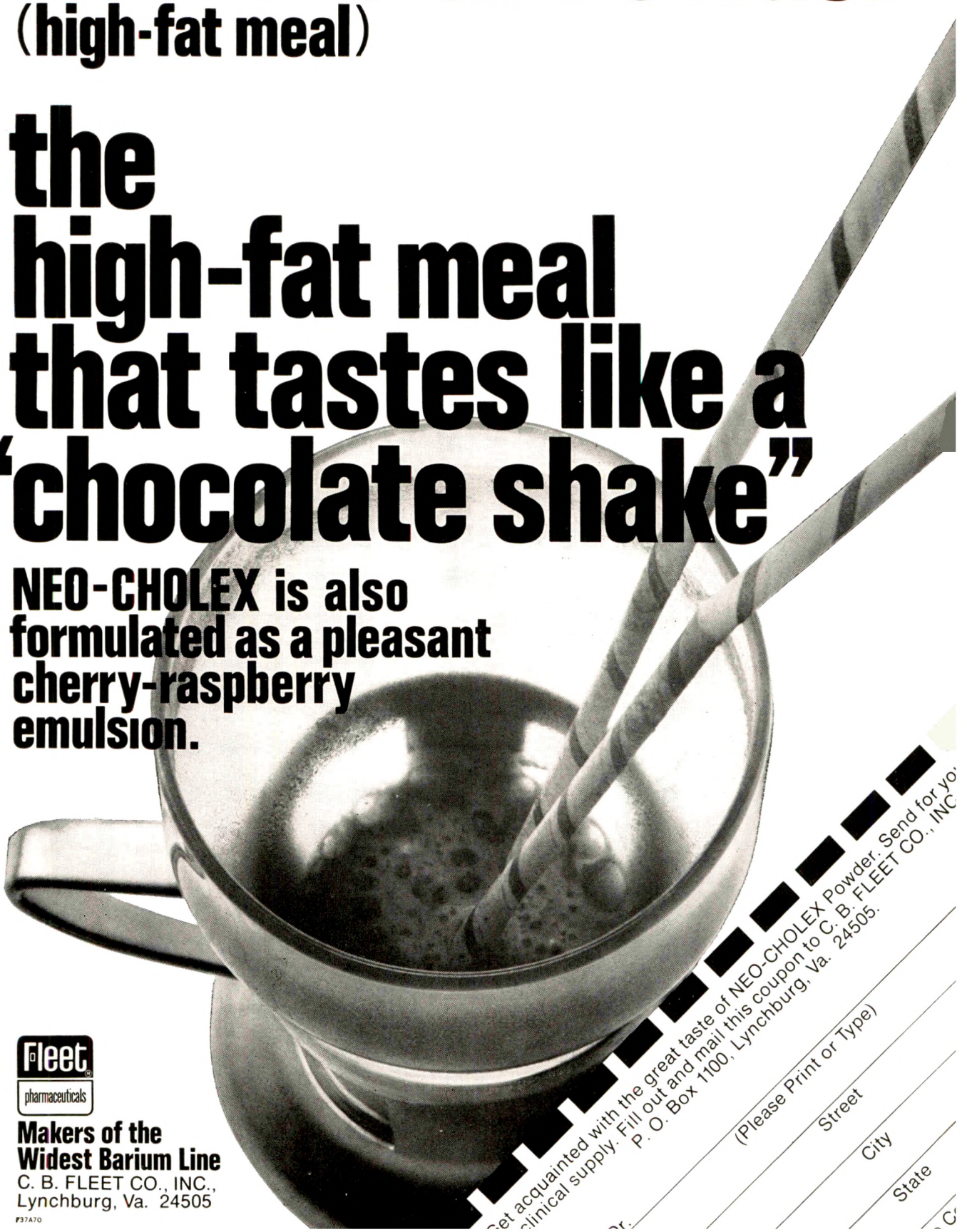
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the high-fat meal that tastes like a "chocolate shake"

NEO-CHOLEX is also formulated as a pleasant cherry-raspberry emulsion.



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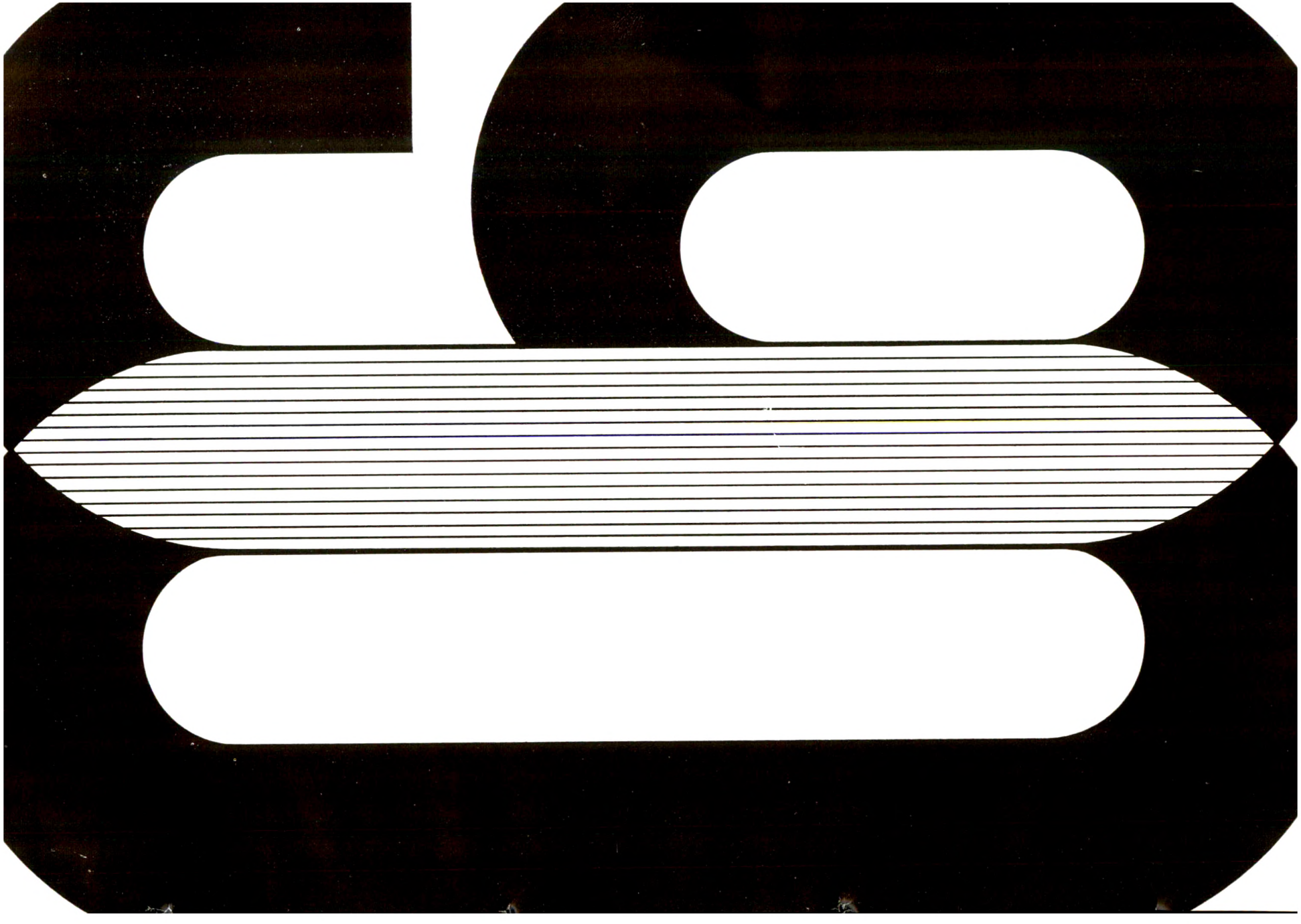
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WHAT A FANTASTIC DEMAND FOR AGFA-GEVAERT 90 SECOND X-RAY FILM ON **POLYESTER!**

Now that AGFA-GEVAERT Rapid Processing Film is available on polyester base as well as on tri-acetate, it is winning new friends all over the country. Maybe it's because of the optimum quality radiographs that are produced, regardless of processing cycle. Maybe it's because of the unusually bright, fog-free images that result. Maybe it's because this universal film is suitable for any processing, automatic or manual.

Why not find out for yourself? Call your LXC Representative.

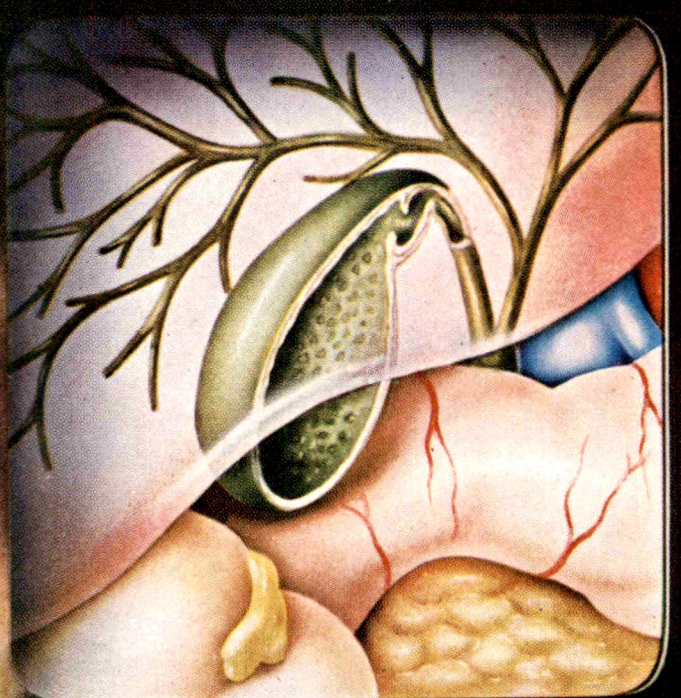
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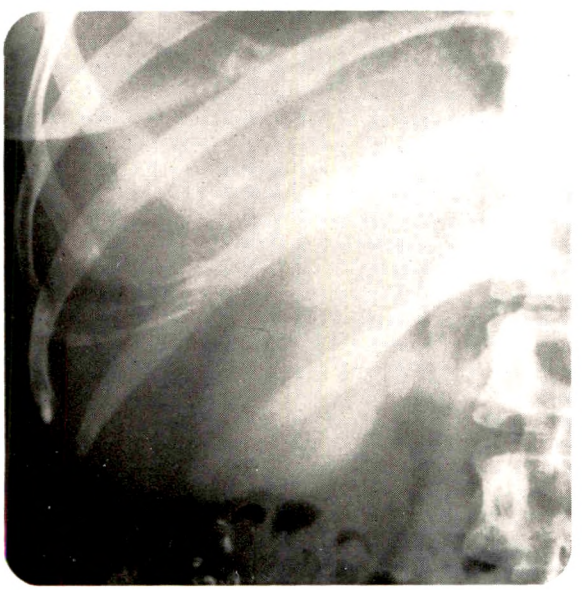
LOW X-RAY CORPORATION
161 Sixth Avenue, New York, N. Y. 10013. Phone 212-255-3700

A nonvisualized gallbladder...

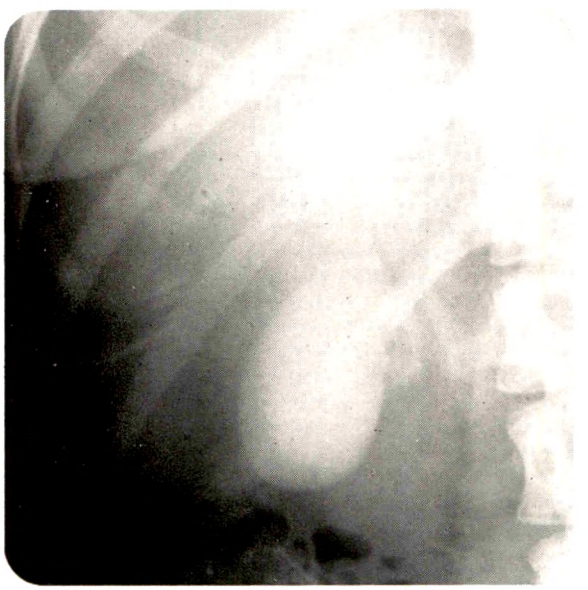


can often be visualized with Oragrafin® Calcium Granules

Calcium Ipodate



Cholecystogram 12 hours after routine dose of iopanoic acid.[1]



Cholecystogram 3 hours later after dose of 6 Gm. of calcium ipodate granules. Note filling of the common duct—from Root[1], discussion on the following page.

When surgery hangs on diagnostic films...

Calcium and sodium ipodate are rapidly absorbed from the gastrointestinal tract and begin to appear in the bile with striking rapidity. The abundant delivery to the liver and biliary system permits visualization even in a poorly functioning gallbladder. In addition, use of calcium ipodate granules permits same-day re-examination, should a second visualization be deemed necessary.

See next page for "Brief Summary."

Recent clinical study[1] demonstrated improved diagnoses

"A group of 114 patients with poorly filled gallbladders after routine administration of iopanoic acid were given 6 gm. of [calcium] ipodate granules immediately following their routine cholecystography. In 66 per cent of these patients opacification of the gallbladder was obtained within 3 hours, allowing a definite diagnosis. Of the remaining 33 per cent, one-half were ultimately proven diseased."[1] For one of the patients in this group, see x-ray pictures on previous page.

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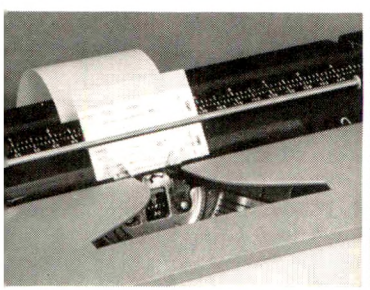
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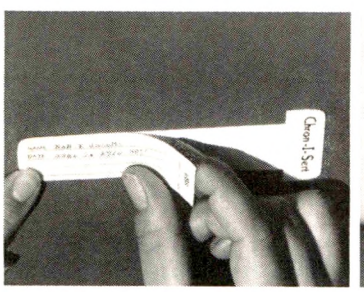
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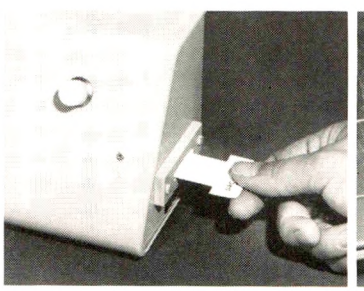
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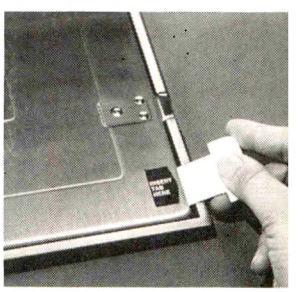
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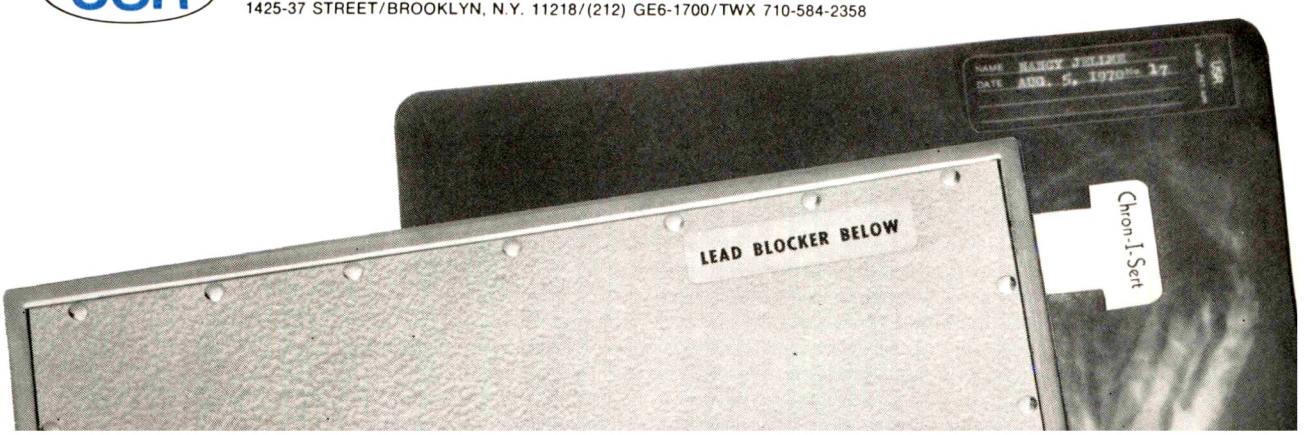
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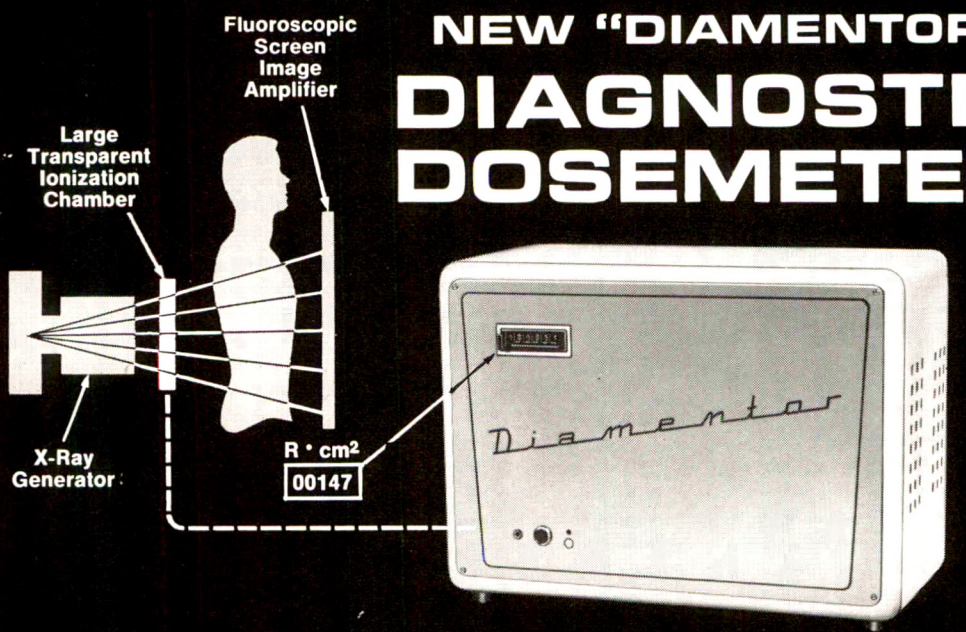
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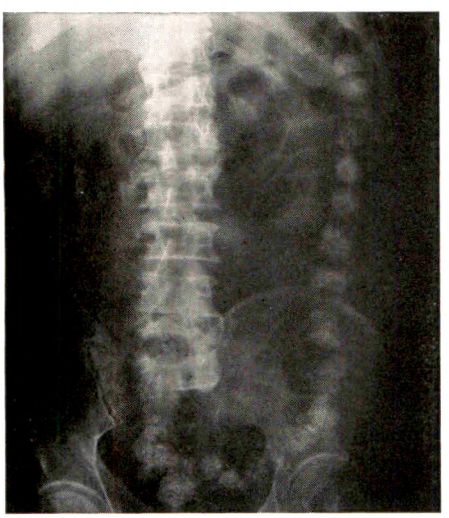
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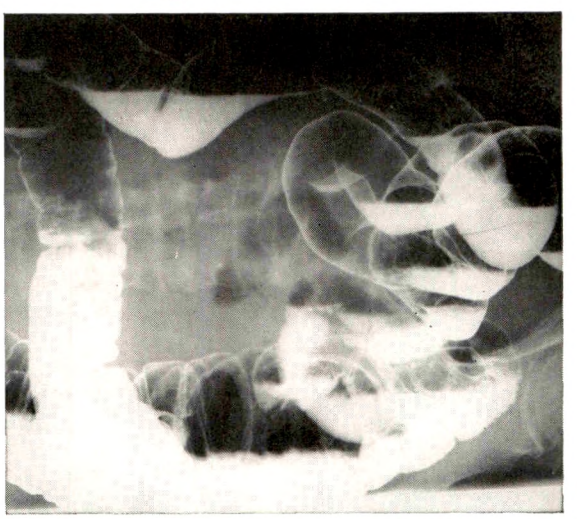
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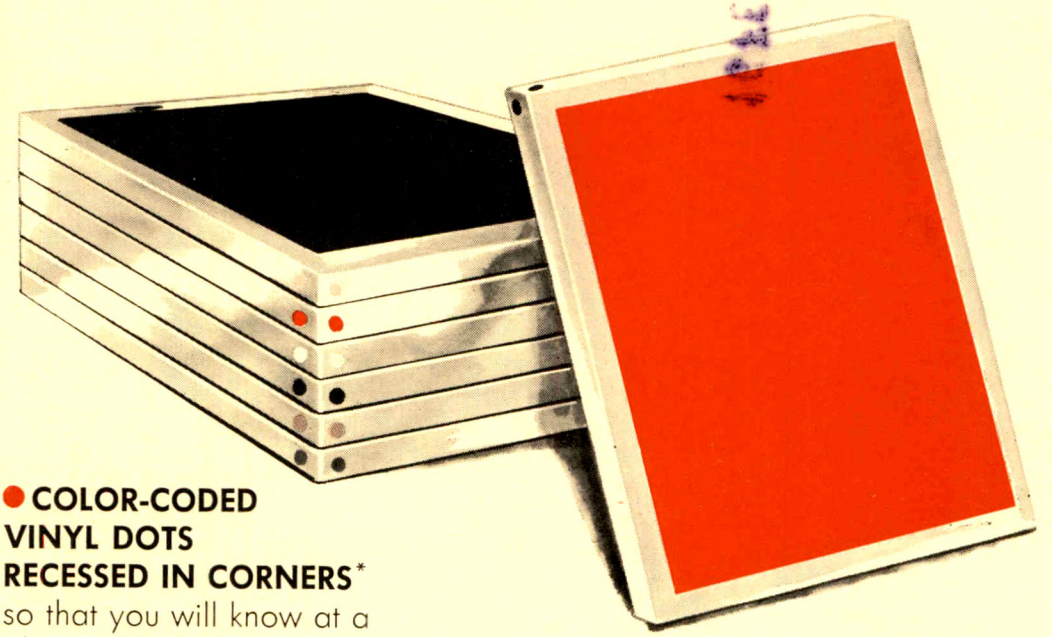
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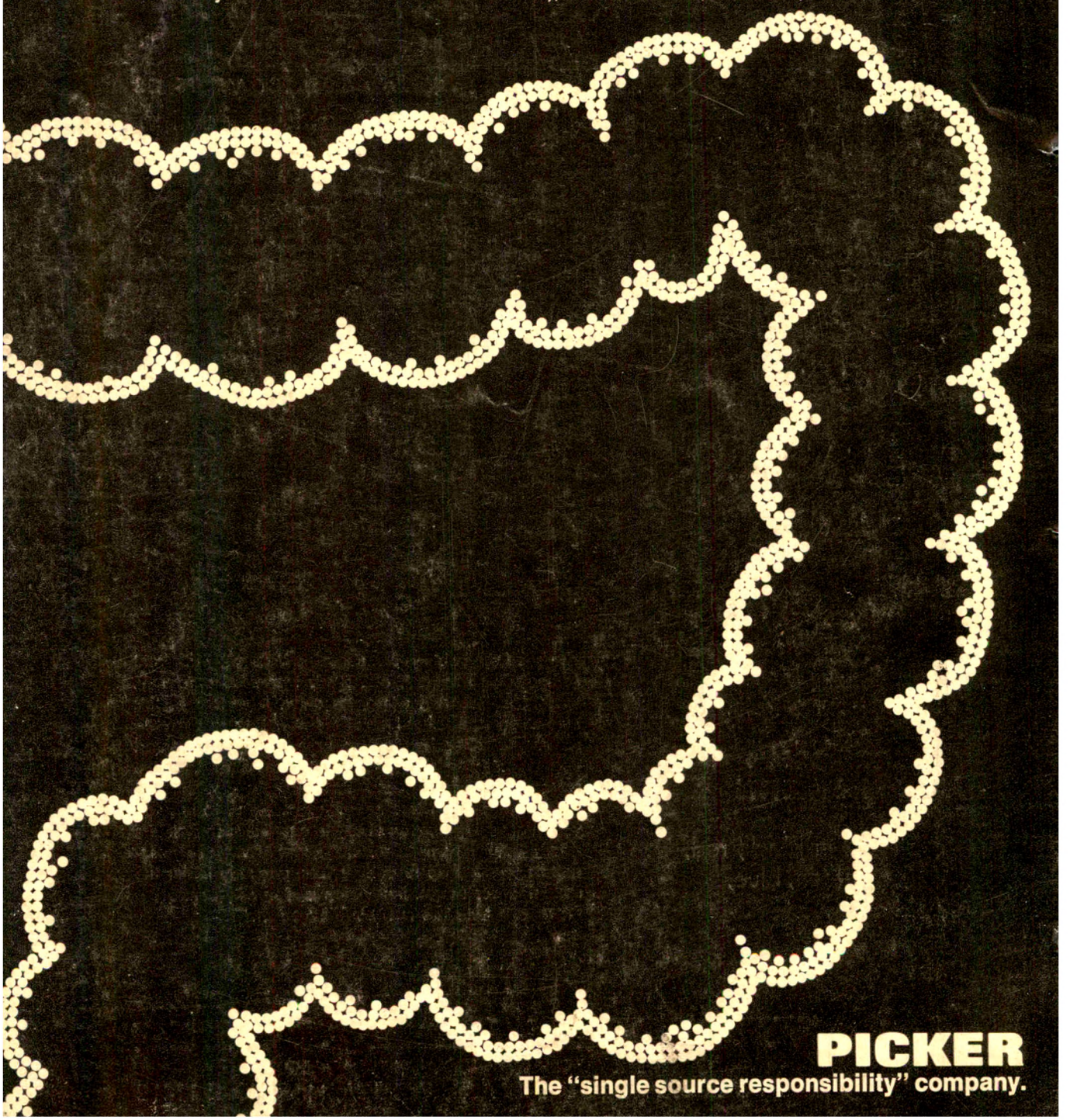
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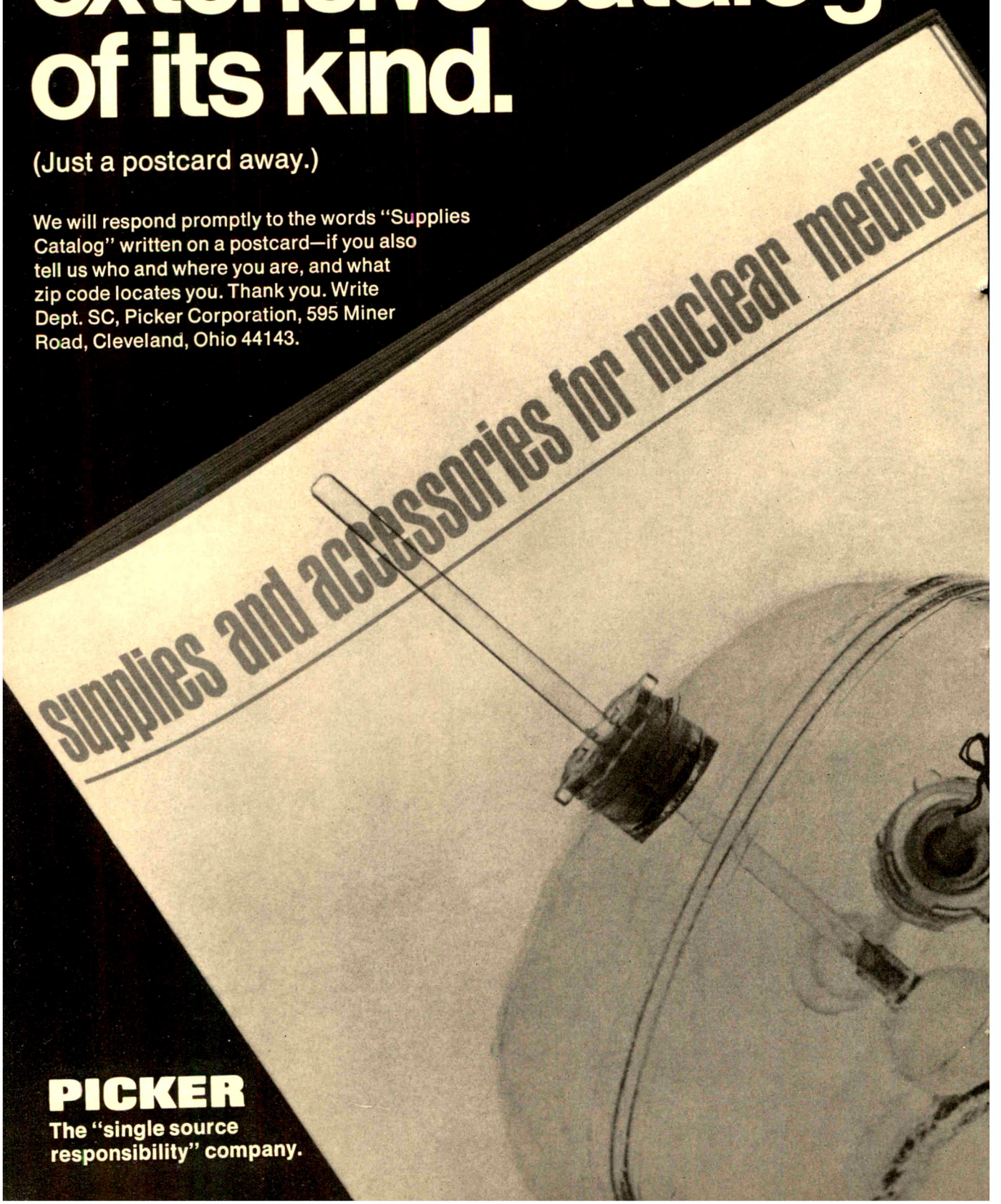
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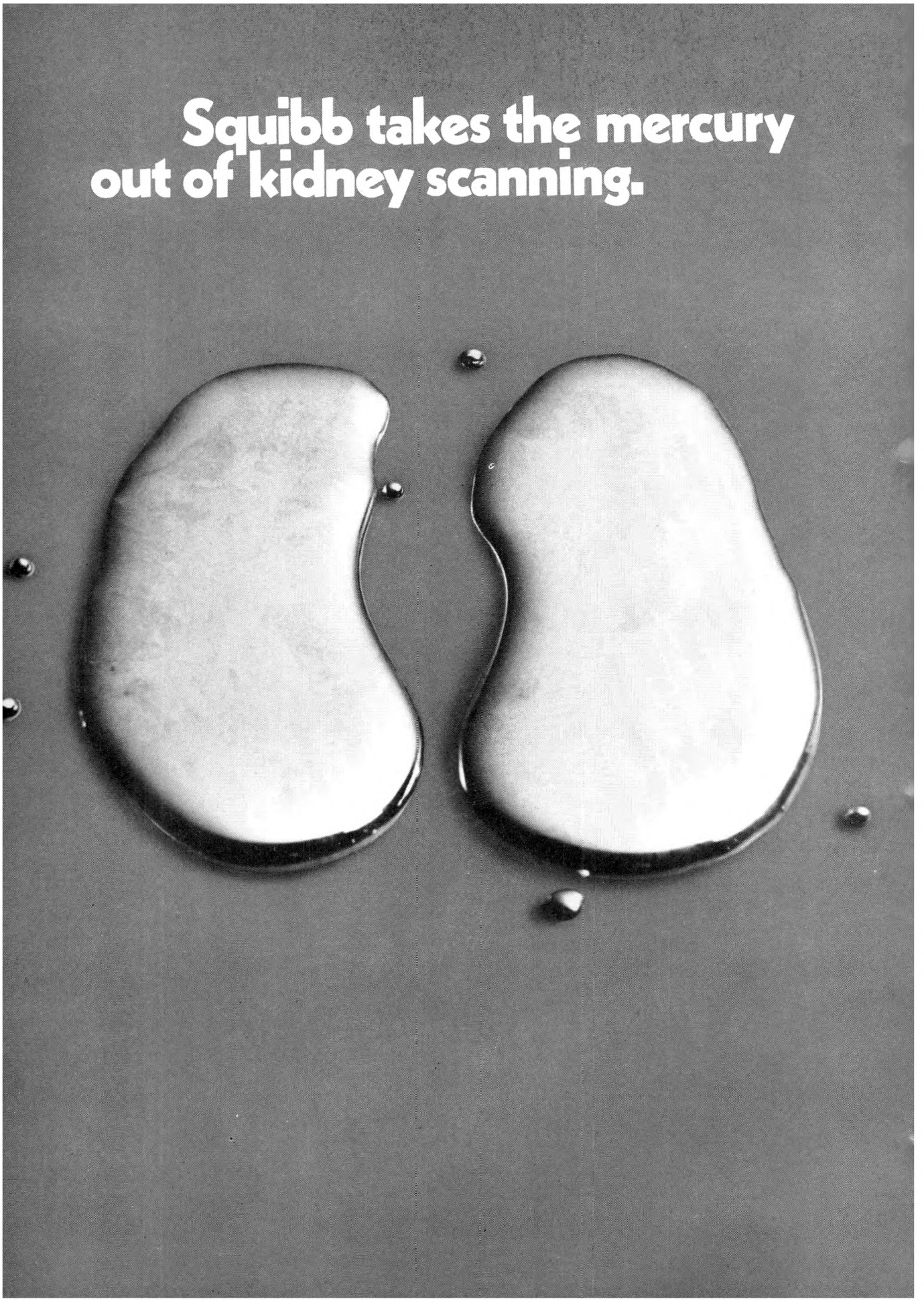
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See next page for brief summary.

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The non-mercurial renal scan.

The RENOTEC (Technetium 99m-Diethylenetriamine Pentaacetic Acid [DTPA]) Kit includes: 1) 5 vials (2 cc. each) of Sterile Reaction Solution providing 5 mg. ferric chloride per cc. and 2.5 to 5 mg. ascorbic acid per cc.; 2) 5 Unimatic® Disposable Syringes (2 cc. each) containing Sterile 0.07N Sodium Hydroxide Solution providing 2.8 mg. sodium hydroxide per cc.; and 3) 5 Unimatic Disposable Syringes (2 cc. each) containing Sterile DTPA Solution providing 2.5 mg. diethylenetriamine pentaacetic acid per cc.

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Warnings: The contents of the syringes in the Renotec Kit and the Tesuloid Kit should **not** be injected directly into a patient.

Usage in pregnancy—These agents should not be administered to women who are pregnant or who may become pregnant and during lactation unless the indications are exceptional and the need for the agent outweighs the possible potential risk from the radiation exposure involved.

Since sodium pertechnetate ^{99m}Tc may be taken up by the fetus and excreted in human milk, administration of the preparation during pregnancy and lactation is not recommended.

Formula feedings should be substituted for breast feedings if these agents must be administered to the mother during lactation.

^{99m}Tc-DTPA, ^{99m}Tc-S colloid, and sodium pertechnetate ^{99m}Tc should not be administered to persons less than 18 years of age unless the expected benefit outweighs the hazards. It should be noted that although radiopharmaceuticals are not generally used in individuals under 18, procedures using ^{99m}Tc-DTPA or ^{99m}Tc-S colloid are occasionally necessary in such patients. The low internal radiation dosage of ^{99m}Tc-DTPA makes it a very satisfactory agent when scans of the kidney, brain, or blood vessels are necessary in young patients. The low internal radiation dosage of ^{99m}Tc-S colloid makes it a very satisfactory agent when liver or spleen scans are necessary in young patients.

Radiopharmaceuticals, produced by nuclear reactor or cyclotron, should be used only by physicians who are qualified by specific training in the safe use and safe handling of radioisotopes and whose experience and training have been approved by the appropriate federal or state agency authorized to license the use of radioisotopes.

When obtaining elutions from Technetope II (Technetium 99m) Sterile Generator, proper radiation safety precautions should be maintained at all times. The column containing ^{99}Mo need not be removed from the lead shield at any time. There is a high radiation field surrounding an unshielded column. Solutions of sodium pertechnetate ^{99m}Tc withdrawn from the generator should always be adequately shielded. The early elutions from the generator are highly radioactive. **Important:** Since material obtained from the generator may be intended for intravenous administration, aseptic technique must be strictly observed in all handling. The stoppers of the eluent bottle, of the elution tube, and of the collecting vial, as well as both rubber closures in the generator column, should be swabbed with a suitable germicide before each entry. All entries into the generator column must be made aseptically with sterile needles. Only the eluent provided should be used to elute the generator. Use a fresh milking tube and collecting vial for each elution; sufficient equipment is provided for this purpose. All equipment used to collect or administer sodium pertechnetate ^{99m}Tc must be sterile. Do not administer material eluted from the generator if there is any evidence of foreign matter. NOTE: The Renotec Kit and the Tesuloid Kit are not radioactive. However, after the eluted ^{99m}Tc is added, adequate shielding of the resulting preparation should be maintained.

Precautions: When using radioactive material, care should be taken to insure minimum radiation exposure to the patient (*i.e.*, by using the smallest dose of radioactivity consistent with safety and validity of data) as well as to all personnel directly or indirectly involved with the patient. Before a test is repeated in the same patient, the need should be carefully evaluated; this is especially true in younger patients.

Each elution from Technetope II (Technetium 99m) Sterile Generator should be assayed before use for ^{99m}Tc activity and for the possible presence of ^{99}Mo. Material containing more than 5 microcuries of ^{99}Mo per dose of ^{99m}Tc pertechnetate exceeds Atomic Energy Commission limits and should not be administered. Poor gastrointestinal absorption of an oral dose of pertechnetate and resultant low blood radioactivity levels have been observed in the postprandial state, in seriously ill patients, and in a small number of normal, fasting individuals. Since pertechnetate is concentrated by the gastric mucosa and the salivary glands, secretions of the digestive tract are radioactive and may cause artifacts on the cranial scan. Therefore, all possible care should be taken to avoid extracranial contamination, not only for the protection of patients and of hospital personnel but also to avoid obtaining a falsely positive scan due to extracranial radiation. Any condition which alters the blood-brain barrier or the normal cranial vasculature may cause abnormal areas of increased radioactivity. The brain scan with sodium pertechnetate ^{99m}Tc is therefore likely to be abnormal in patients with scalp contusions or acute head injuries. Following a craniotomy, uptake of radioactivity is increased throughout the operative field, usually for only a few weeks but in some instances for prolonged periods. Since cerebral radiographic techniques temporarily affect the blood-brain barrier, brain scanning with sodium pertechnetate ^{99m}Tc should precede cerebral angiography when possible, or should be postponed for several days thereafter. A negative brain scan does not rule out the possibility of a lesion and should therefore never be considered diagnostically conclusive. Because the normal vascular structures are more apparent on a ^{99m}Tc pertechnetate scan than on a radiochlormerodrin scan, and because the choroid plexus may be visible, it is particularly important to recognize the appearance of a normal brain scan when ^{99m}Tc pertechnetate is used, in order to avoid incorrect interpretation.

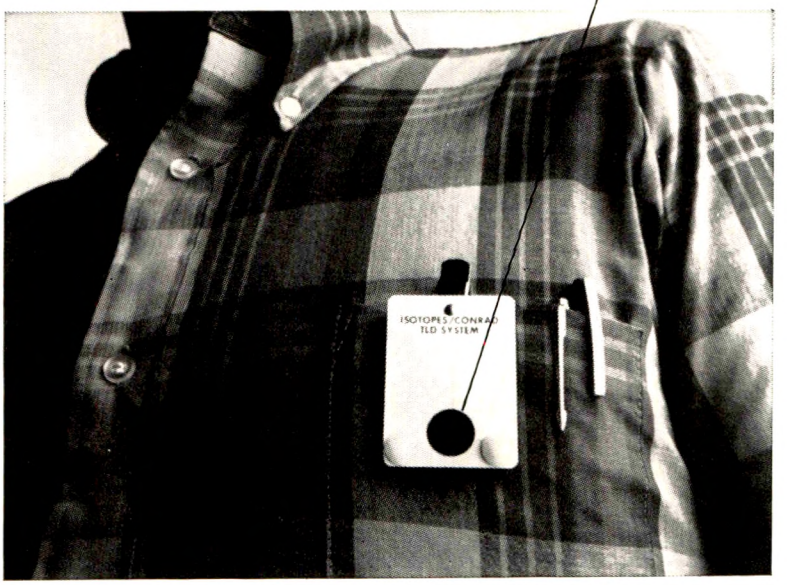
NOTE: The Renotec Kit and the Tesuloid Kit were designed for use with the sodium pertechnetate eluate obtained from a Technetope II Sterile Generator. It is recommended that only Technetope II be used as the source of sodium pertechnetate with the Renotec Kit and the Tesuloid Kit unless the user has demonstrated that other sources of ^{99m}Tc are consistently compatible and meet the standards of Technetope II.

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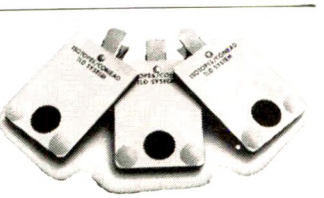
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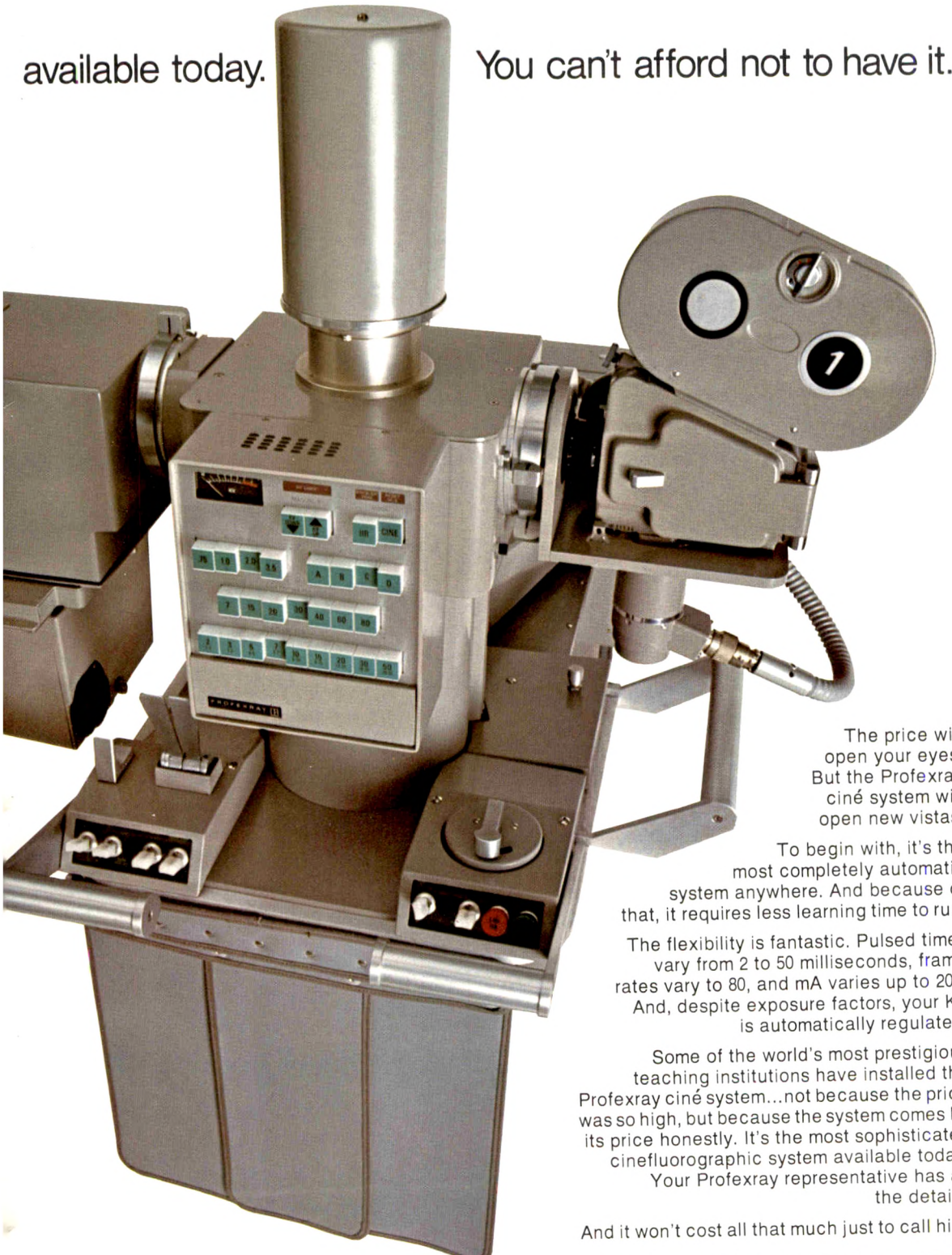


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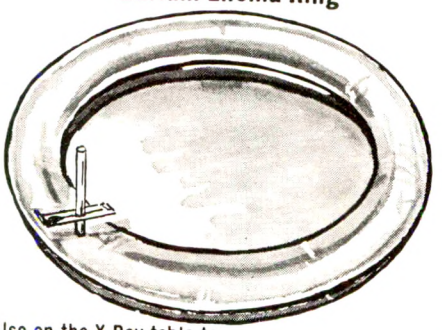
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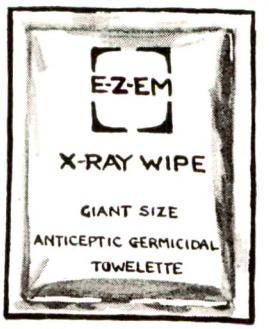
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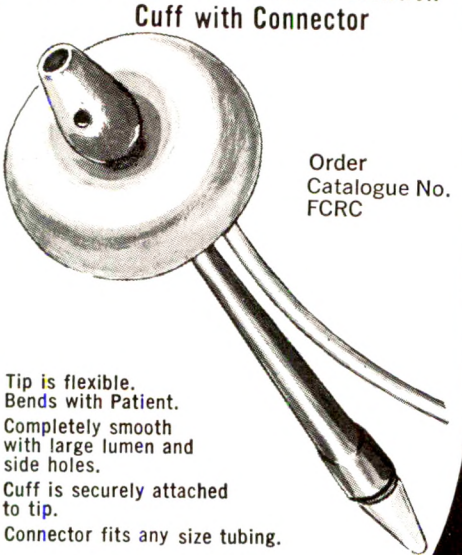
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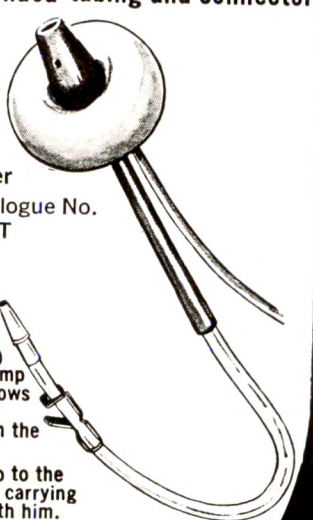
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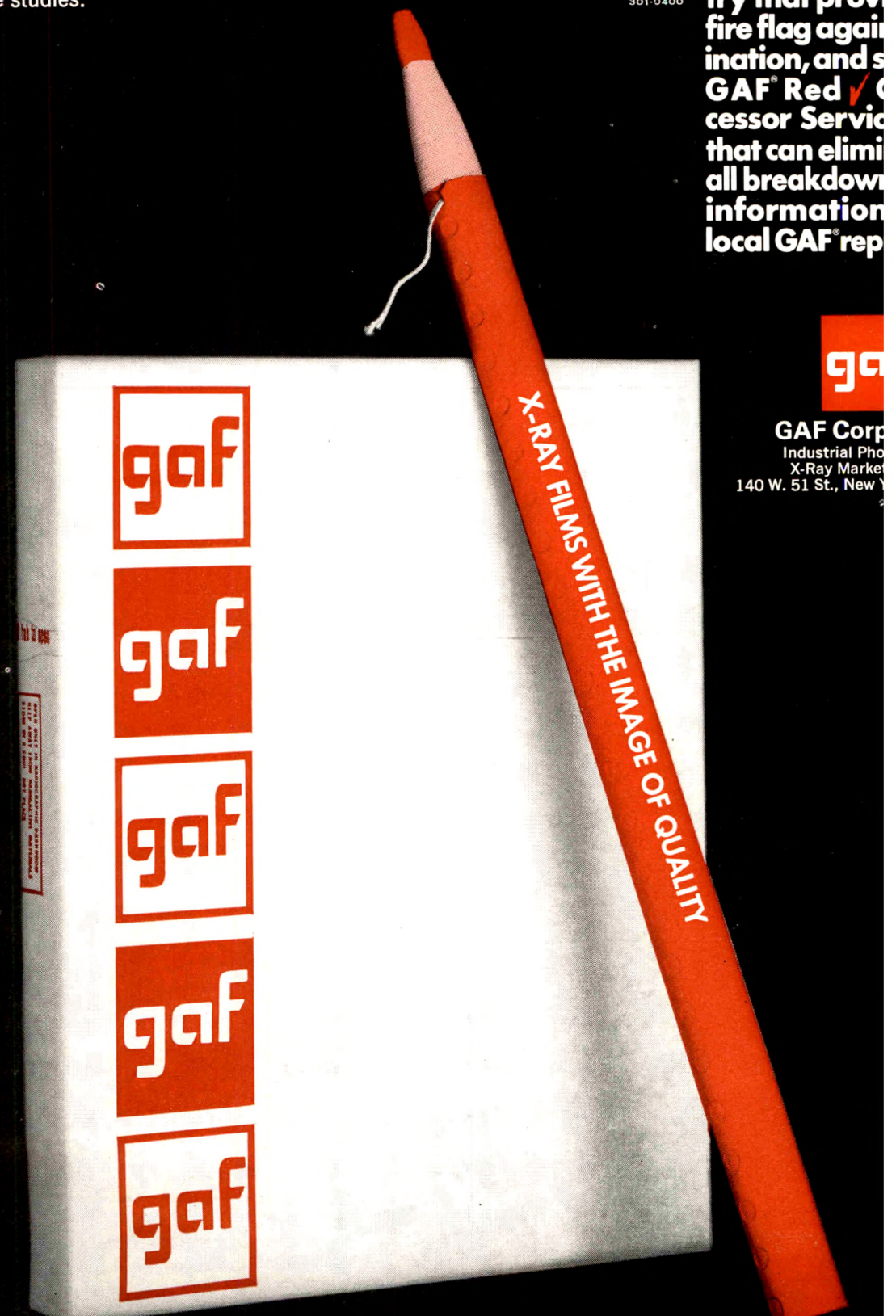
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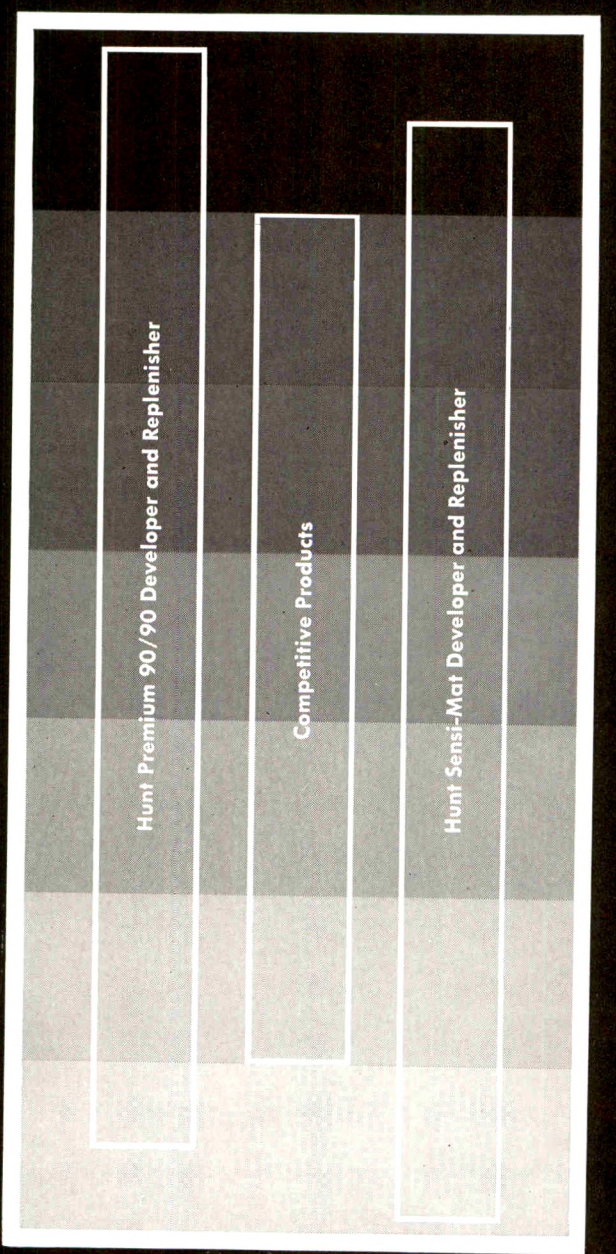
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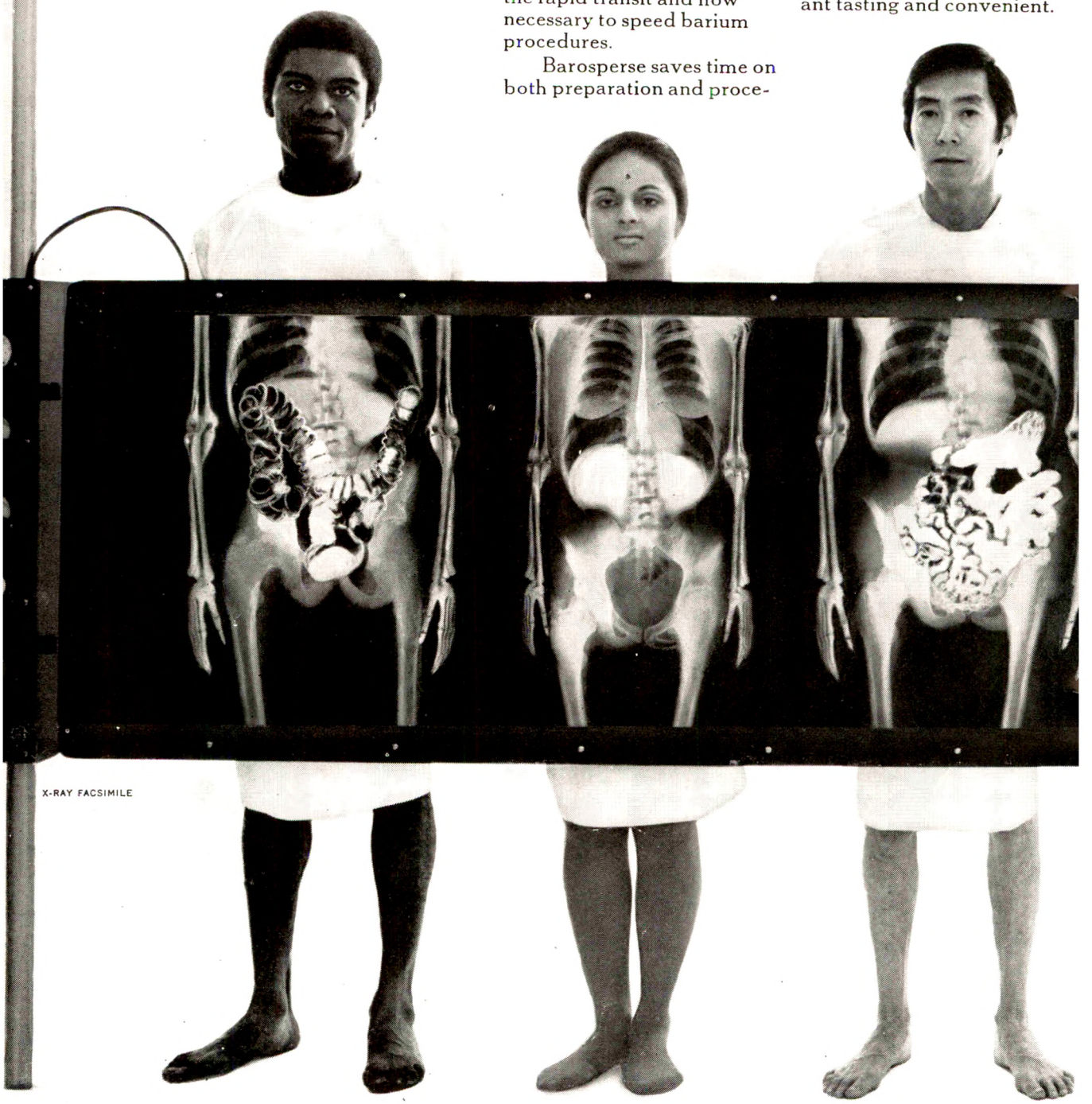
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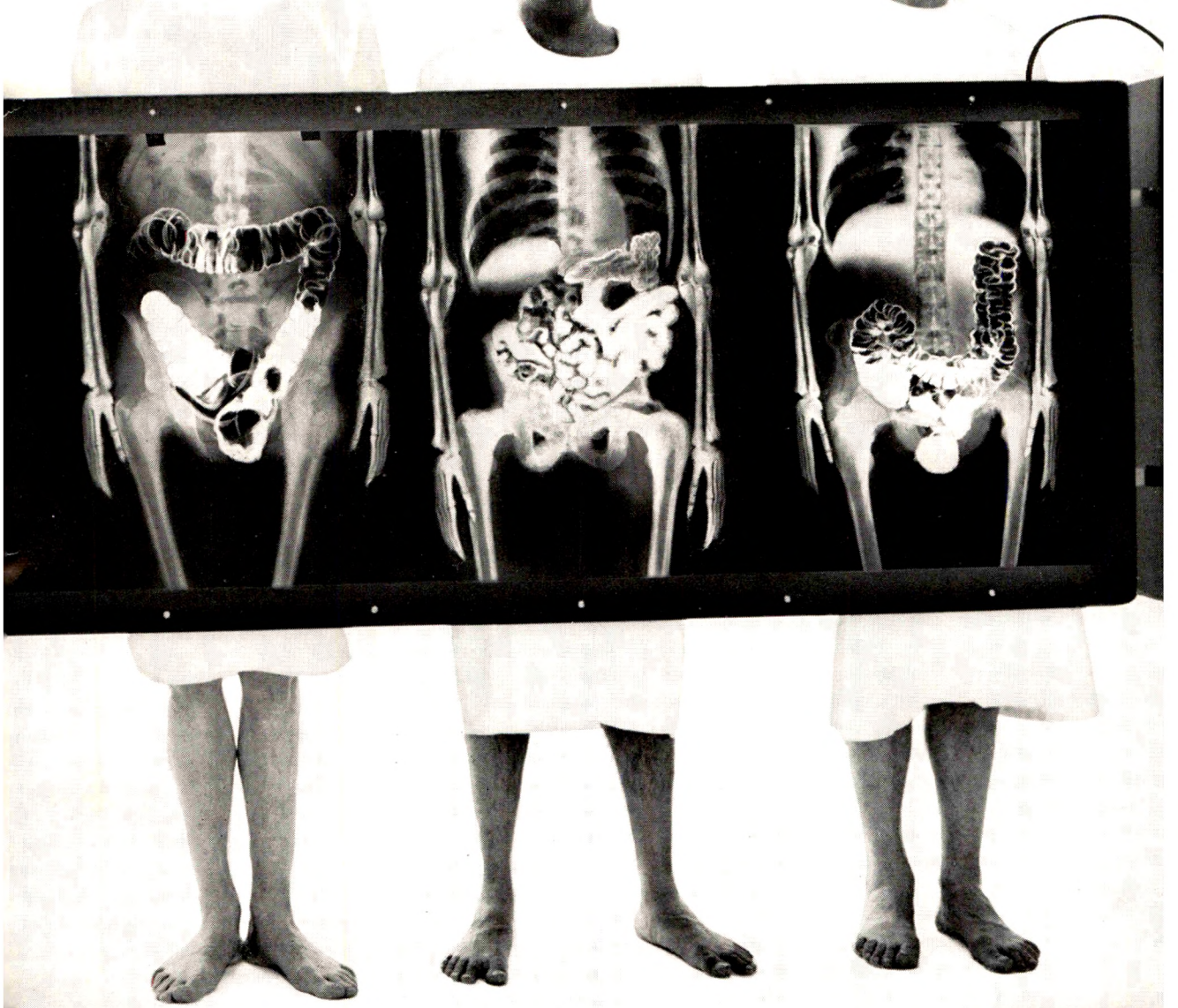
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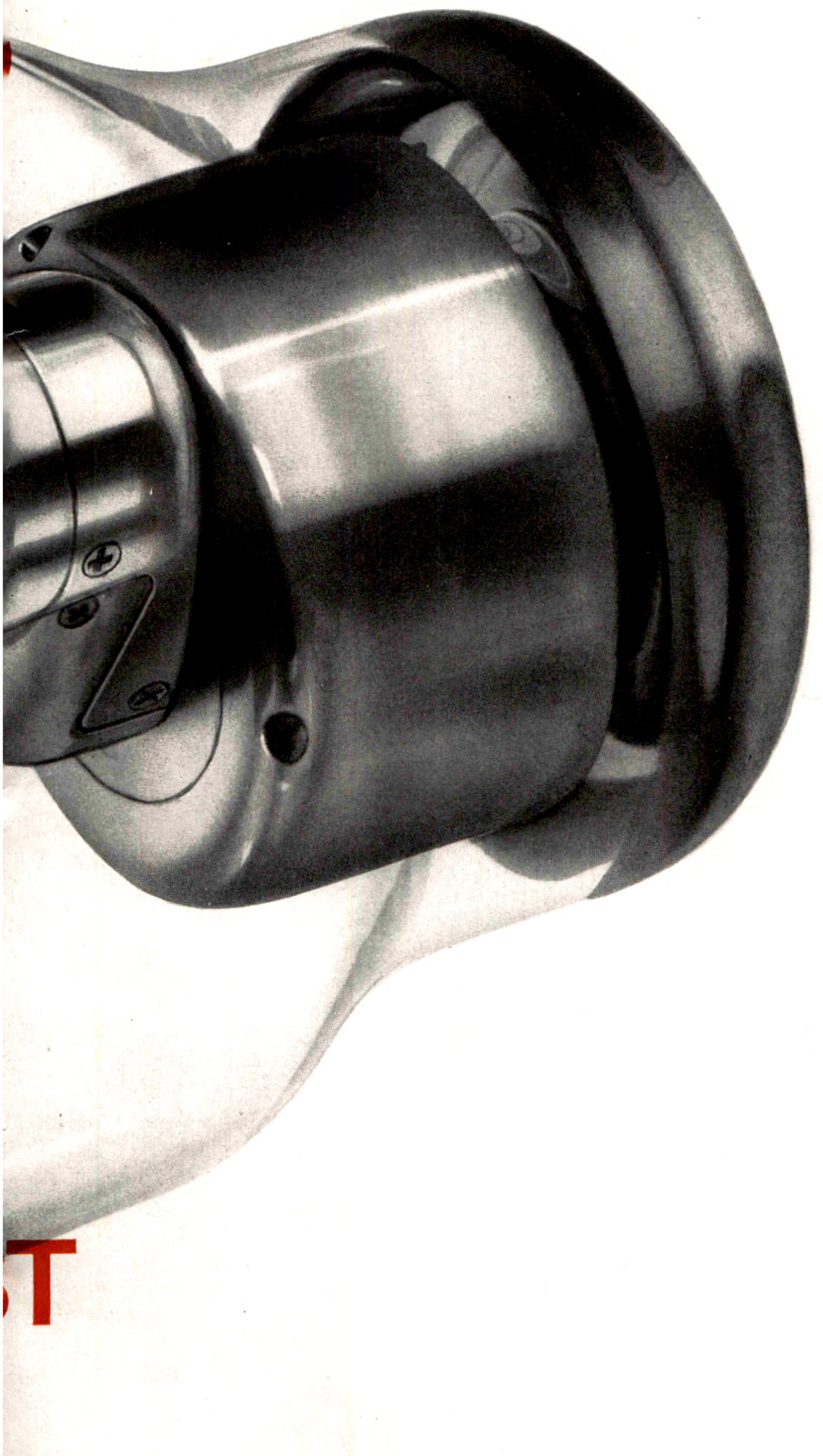
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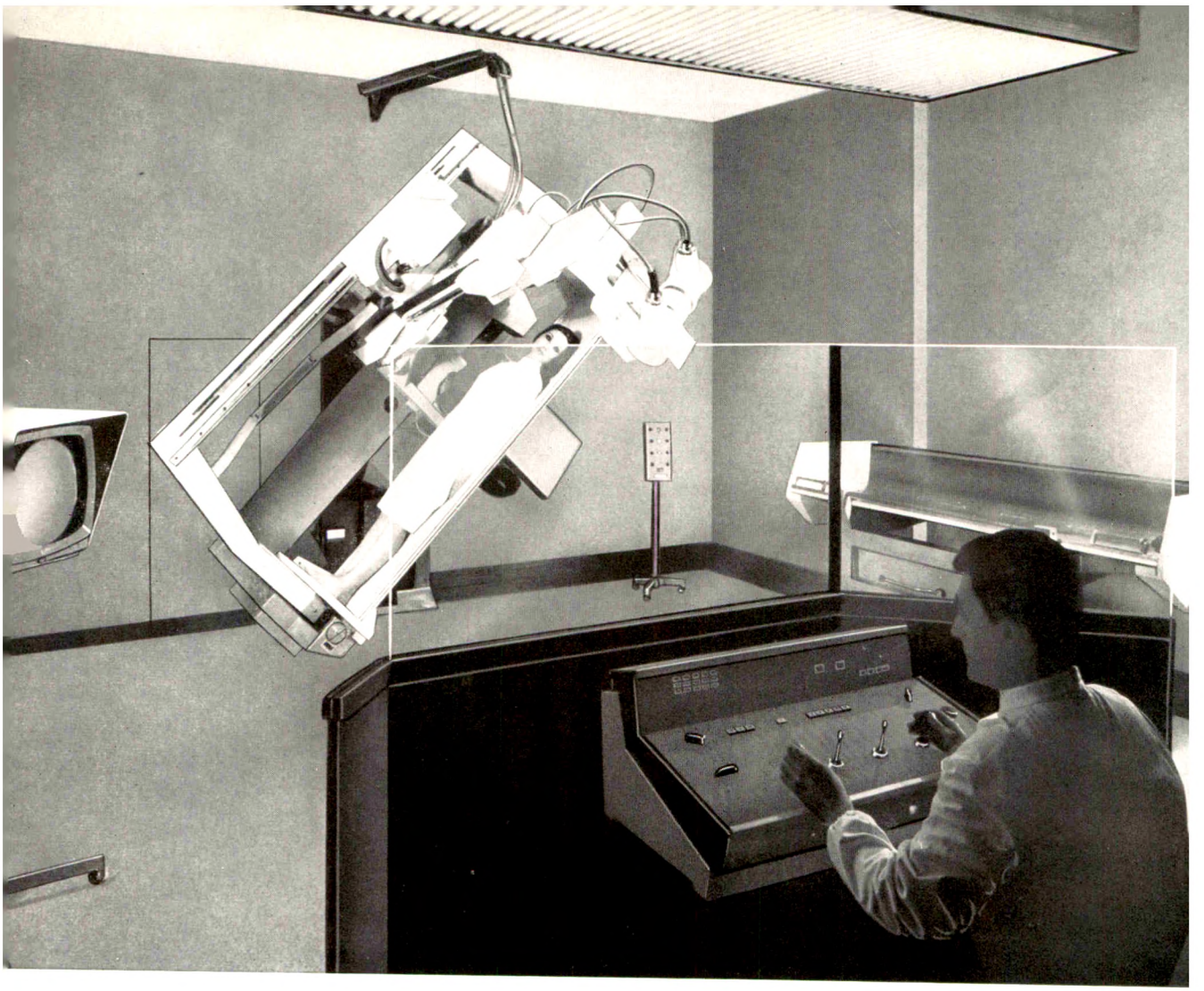
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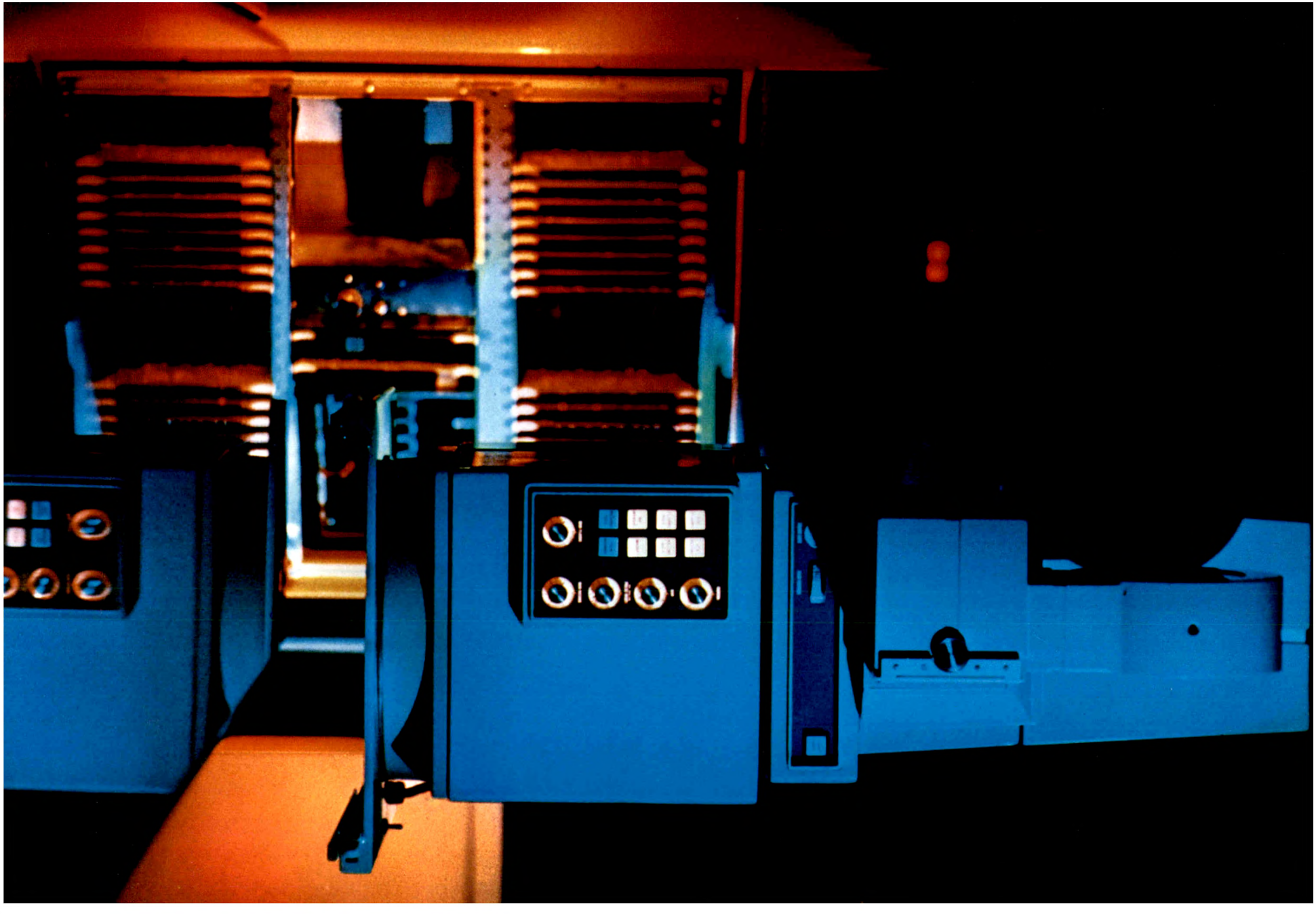
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You can see many of these GE answer systems, ask questions, even put them through their paces. They'll be at the 1970 meeting of RSNA in Chicago. See you there.
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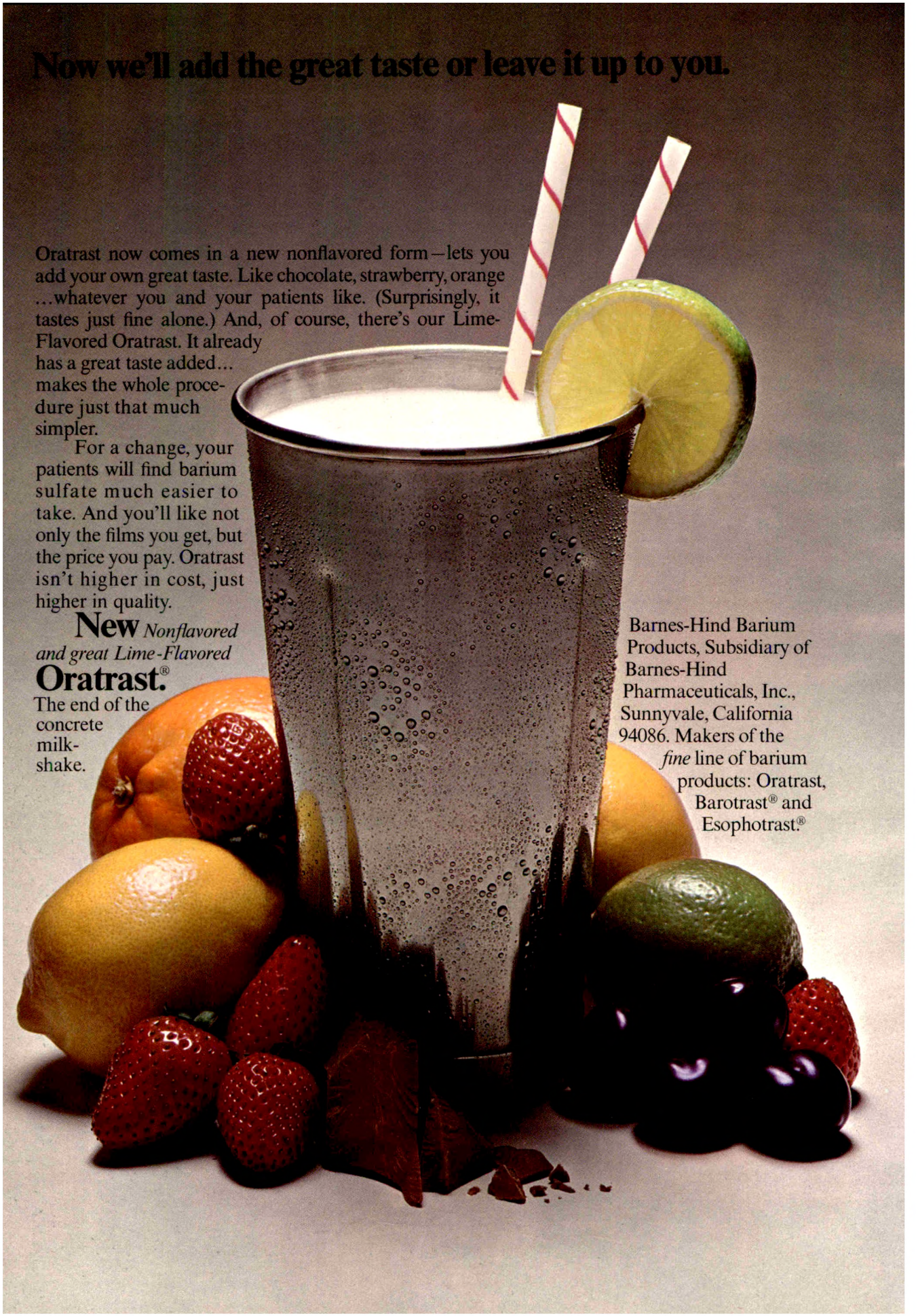
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Barnes-Hind Barium Products, Subsidiary of Barnes-Hind Pharmaceuticals, Inc., Sunnyvale, California 94086. Makers of the *fine* line of barium products: Oratrast, Barotrast® and Esophotrast.®

The great visual-izer

The great visualizer

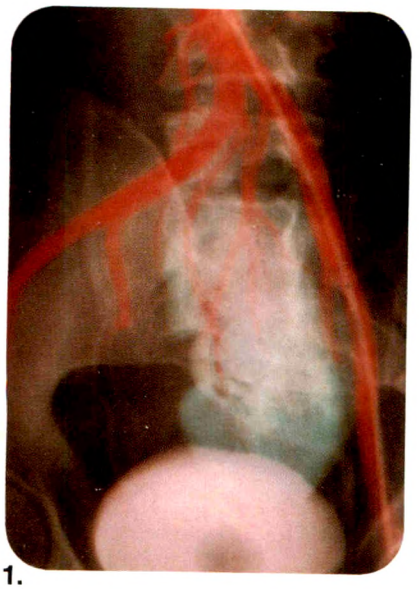
Hypaque® 50%

sodium

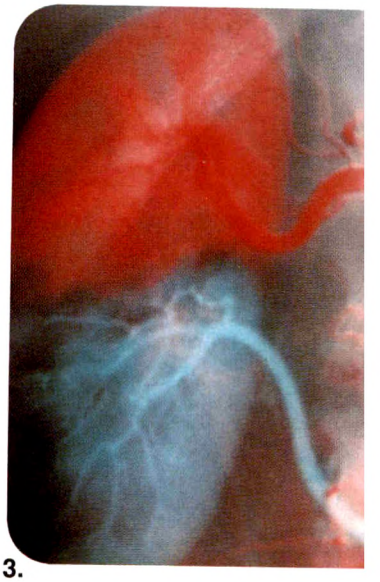
Brand of **sodium diatrizoate, USP**

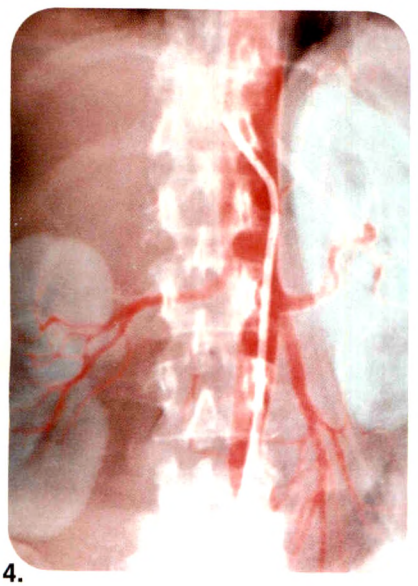
1. **Aortogram:**
   Ectopic left kidney
2. **Selective Renal Arteriogram:**
   Adenocarcinoma of kidney
3. **Aortogram:**
   Dual Renal Blood Supply
4. **Aortogram**
5. **Excretory Urogram**
6. **Selective Renal Angiogram:**
   Renal cyst

To brighten your black, white, and gra world, we present these x-rays in colc

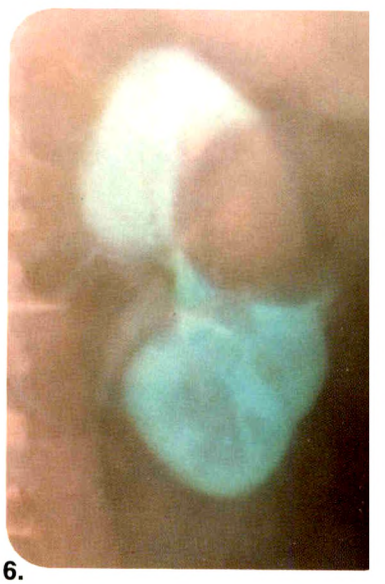

1.


2.


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5.


6.

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Hypaque® 50%
sodium

Brand of **sodium diatrizoate, USP**

Winthrop

Summary of Full Prescribing Information

HYPAQUE® SODIUM (brand of **sodium diatrizoate, USP**) **50%** Sterile Aqueous Injection (Weight/Volume)

EXCRETORY UROGRAPHY AND DRIP INFUSION PYELOGRAPHY

Description: Hypaque sodium is sodium 3,5-diacetamido-2,4,6-triiodobenzoate, a radiopaque medium used to delineate internal structures. Hypaque sodium 50 per cent contains 300 mg. iodine per ml. Contains calcium disodium edetate 1:10,000 as stabilizer. Na_2CO_3, NaOH, and HCl added to adjust pH between 6.5 and 7.5. Not for multiple dose use. Should be protected from strong light.

Warning: Do not use Hypaque sodium for myelography. Injection of even a small amount into the subarachnoid space may produce convulsions and result in fatality.

General and Specific Precautions: Caution is advised in patients with a history of bronchial asthma, other allergic manifestations, or of sensitivity to iodine; these conditions represent a special risk, though not an absolute contraindication. To avoid or minimize possible allergic reactions, premedication with antihistamines may be considered. **Benadryl® (brand of diphenhydramine hydrochloride) in the same syringe with Hypaque may result in precipitation.** A test dose of contrast medium is not entirely reliable. Severe reactions, including fatalities, have occurred with a test dose as well as with the full diagnostic dose.

Caution is advised in patients with severe cardiovascular disease. In patients with impaired renal function and cardiovascular disease, drip infusion pyelography imposes a sudden osmotic and sodium load which can precipitate congestive heart failure. Contrast media injected into arteries or veins can promote sickling of red cells in susceptible individuals. In patients with reduced renal function, repeat of excretory or retrograde pyelography should be delayed at least 48 hours to avoid temporary suppression of urine. In preparation for excretory urography of patients with multiple myeloma, partial dehydration is not recommended, since this may predispose to the precipitation of myeloma protein in the kidney tubules. When thyroid function studies (PBI and 24-hour radioiodine uptake levels) are indicated, they should be performed *prior* to radiographic studies or several days afterwards, to avoid inaccurate results.

General and Specific Adverse Reactions: Adverse reactions, usually of a minor nature, have occurred in 10-14 per cent of patients who have received Hypaque intravenously. Reactions due to *faulty technique* include hematomas and ecchymoses, following extravasation from the vein, and pyrogenic reactions. *Hemodynamic reactions* include vasodilatation with flushing, hypotension and, rarely, vein cramp or thrombophlebitis. Serious *cardiovascular reactions* include rare cases of cardiac arrhythmias (e.g., ventricular fibrillation), shock, and cardiac arrest. Transient proteinuria may occur occasionally following the injection of radiopaques and, rarely, oliguria and anuria have been reported secondary to a hypotensive reaction. *Allergic reactions* include asthmatic attacks, nasal and conjunctival symptoms, cutaneous reactions such as urticaria and, rarely, anaphylactic shock, sometimes with fatal outcome. Severe reactions may also be manifested by signs and symptoms relating to the *respiratory system* (dyspnea, cyanosis, pulmonary or laryngeal edema), or to the *nervous system* (restlessness, confusion, or convulsions). *Other reactions* include nausea, vomiting, excessive salivation, anxiety, headache, and dizziness. Infrequently, "iodism" (salivary gland swelling) from organic compounds appears two days after exposure and subsides by the sixth day. Reactions to drip infusion pyelography may not appear until some hours after the examination.

ANGIOGRAPHY (PERIPHERAL ARTERIOGRAPHY AND VENOGRAPHY)

Precautions *(See also General Precautions):* Extreme caution is advised in considering peripheral arteriography in patients suspected of having thromboangiitis obliterans (Buerger's disease). Any procedure (even needling or insertion of a catheter) may induce a severe arterial and/or venous spasm. Caution is also advisable in patients with severe ischemia associated with ascending infection.

Adverse Reactions *(See also General Adverse Reactions):* In addition to those listed under General Adverse Reactions, are those due to arterial trauma during the procedure (i.e., needling, insertion of catheter, subintimal injection, perforation, etc.) as well as to the hypertonicity or effect of the medium; also reported are transient arterial spasm, extravasation, hemorrhage, hematoma with tamponade, injury to nerves proximal to artery, thrombosis (rare in venography, if vein is irrigated following injection), dissecting aneurysm, arteriovenous fistula, (e.g., with accidental perforation of femoral artery and vein during needling), transient leg pain from contraction of calf muscles in femoral arteriography; transient hypotension after intraarterial (brachial) injection, and brachial plexus injury with axillary artery injections.

CEREBRAL ANGIOGRAPHY

Inasmuch as cerebral angiography is a highly specialized procedure requiring the use of special techniques, it is recommended that Hypaque sodium (brand of sodium diatrizoate) be used for this purpose only by persons skilled and experienced in carrying out the procedure.

Contraindication: Carotid angiography should be avoided during the progressive period of a stroke because of the increased risk of cerebral complications.

Precautions *(See also General Precautions):* Select patients for this procedure with care. Use the 50 per cent solution with caution in extreme senility (but not old age *per se*), advanced arteriosclerosis, severe hypertension, and cardiac decompensation.

The diagnostic value of the procedure, according to many authorities, when employed early enough in locating lesions amenable to surgery, may outweigh the added risk to patients who have recently experienced cerebral embolism or thrombosis (stroke syndrome). A small number of postangiographic fatalities, including progressive thrombo: curred in which the procedure did not appear to play a r with severe cerebrovascular disease may be examined indirect methods of angiography.

Care should be exercised to avoid contaminating cathete needles, and contrast media with glove powder or cotton fi

Angiography is hazardous in subarachnoid hemorrhage. In r procedure can be hazardous because of ischemic compli ticularly if performed during or soon after an attack.

Adverse Reactions *(See also General Adverse Reactions)* sources of complications are faulty technique, occlusive at vascular disease, repeated injections of the contrast media doses than those recommended.

Untoward reactions are mostly mild and transient, althoug visual field defects and deaths have been reported.

Vascular reactions: flushing, vessel spasm, thrombophlet taneous petechiae.

Neurologic complications: transient cerebral blindness, ne disorders, convulsions, coma, hemiparesis, unilateral visual field defects, aphasia, and respiratory difficulties.

AORTOGRAPHY

Precautions *(See also General Precautions):* Special cauti to avoid inadvertent intrathecal injection when using the technique. The incidence and severity of reactions or that may occur are influenced by: the care and experienc the procedure is performed; the amount and type of medi age and condition of the patient; and the premedication ar used. Since aortography is not without some danger, it sl formed only by those experienced in the technique. Wher repeated injections of the solution during a single stu avoided.

Adverse Reactions *(See also General Adverse Reactions)* age and shutdown; neurologic complications such a myelitis or paraplegia; cardiovascular complications su cardiac arrest or failure, coronary occlusion, arterial thr bolism, and perforation of vessels; extravasation or hem the puncture site or retroperitoneal catheter perforation the intestinal wall; acute pancreatitis; deaths; diffus petechiae; subintimal injection and aortic dissection.

INTRAOSSEOUS VENOGRAPHY

See General Precautions and Adverse Reactions. A gene is sometimes necessary since the method is painful. Oc travasation of the contrast medium from the needle into may occur.

DIRECT CHOLANGIOGRAPHY

Precaution *(See also General Precautions):* This proced used with caution in the presence of acute pancreatitis more than 5 to 10 ml. without undue pressure.

Adverse Reactions *(See also General Adverse Reaction* be attributed to injection pressure or excessive volume o resulting in overdistention. Such pressure may produce epigastric fullness, followed by moderate pain in the ba per abdominal quadrant, which will subside when injecti

Some of the medium may enter the pancreatic duct and sient serum amylase elevation 6 to 18 hours later, witho effects. Pancholangitis resulting in liver abscess has b Occasionally, nausea, vomiting, fever, and tachycardi observed.

HYSTEROSALPINGOGRAPHY

Contraindications: Menstruation, active or imminent; in part of the genital tract, including the external genital known or suspected. Use not advised for six months afte nancy, or 30 days after conization or curettement.

Precaution *(See also General Precautions):* Caution sh cised in patients with known or suspected carcinoma to spread of the lesion by the procedure.

SPLENOPORTOGRAPHY

Precautions *(See also General Precautions):* Procedure formed with caution on patients with blood dyscrasias, severe bleeding, or a spleen recently become tender an

Adverse Reactions *(See also General Adverse Reactio* hemorrhage with hemoperitoneum has been reported, instances, hemorrhage has required splenectomy.

How Supplied: Ampuls of 30 ml., boxes of 10 and 25. Box ampuls for testing sensitivity are available upon reque and 30 ml., rubber stoppered, boxes of 10 and 25. Vials ber stoppered, boxes of 10. Each vial contains sufficient mit withdrawal of 1 ml. for testing sensitivity.

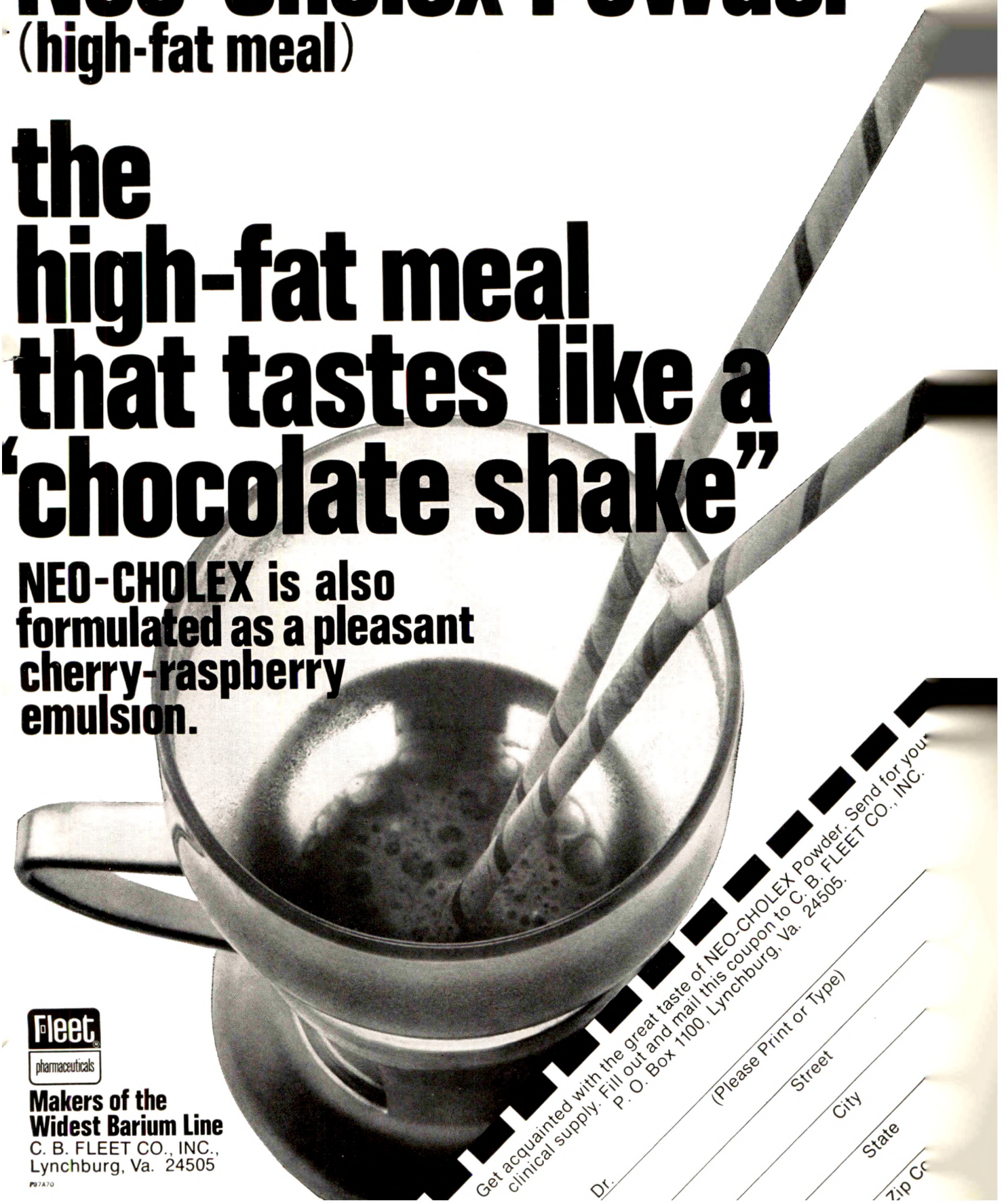
If the solution is chilled, crystals may form but will rea the ampul or vial is placed in moderately hot water befo body temperature before injecting.

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(high-fat meal)

the high-fat meal that tastes like a "chocolate shake"

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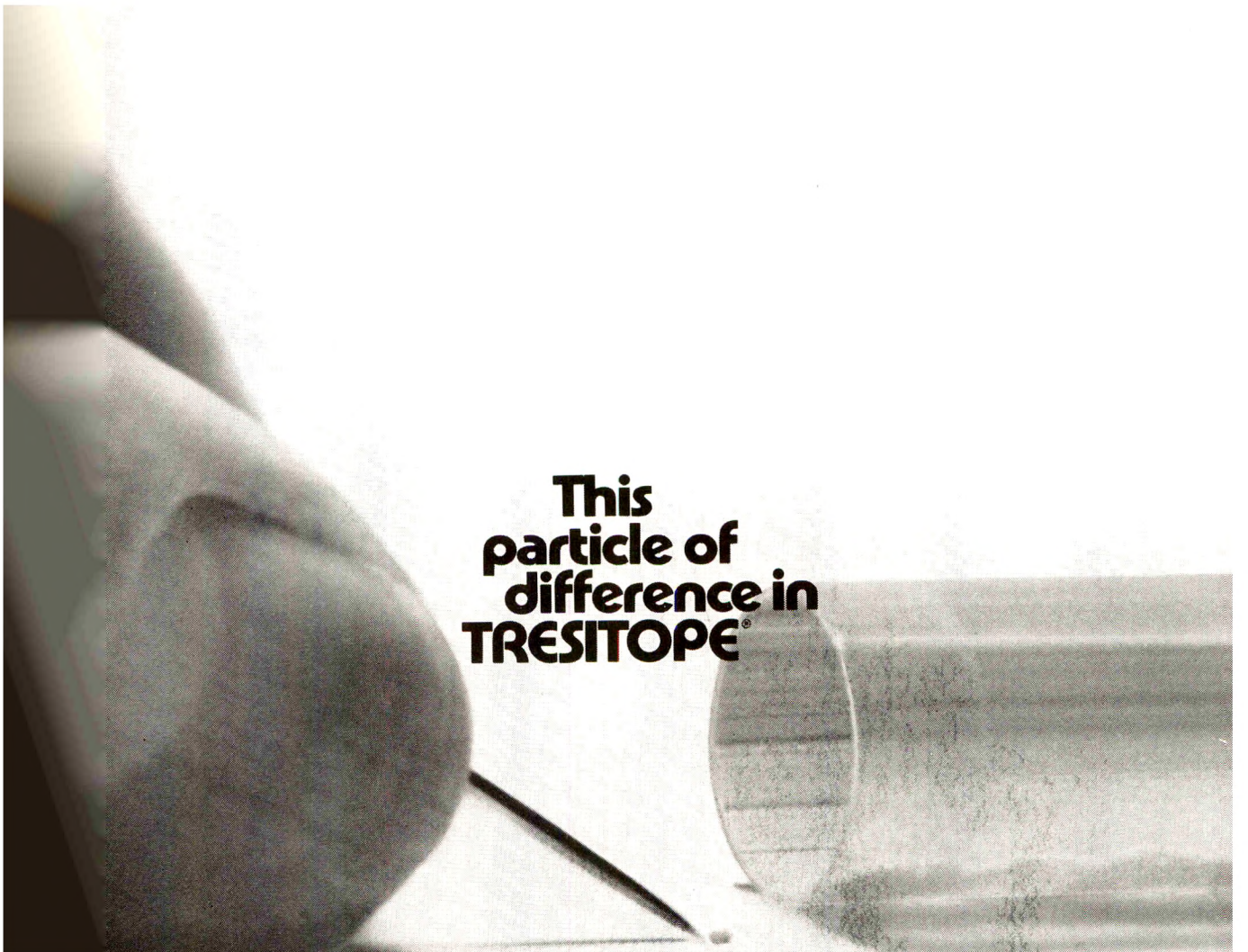
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This particle of difference in TRESITOPE®



makes a big difference in your in vitro thyroid function tests*



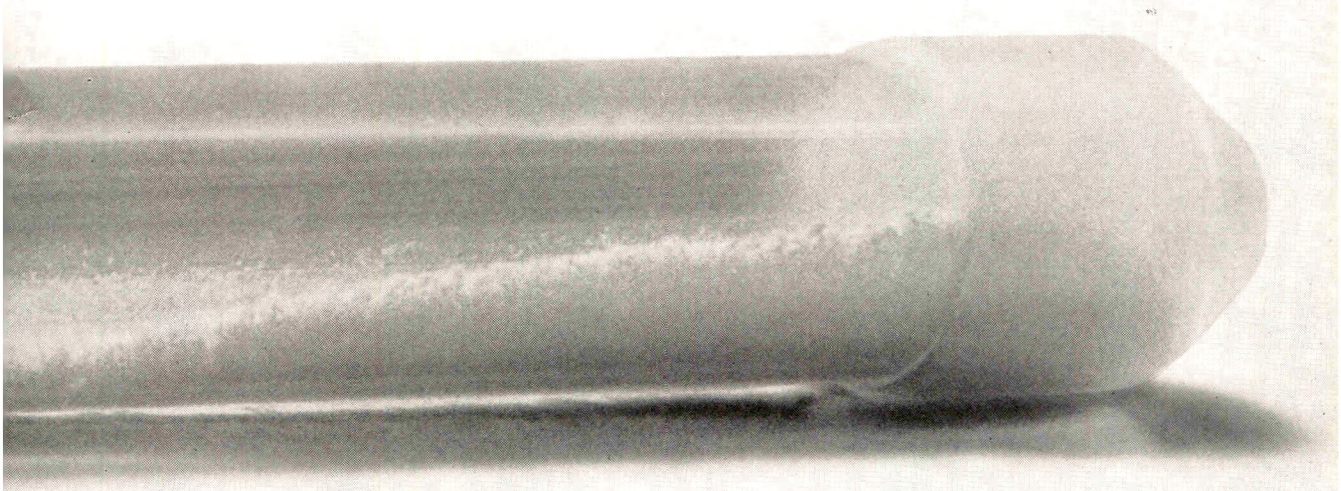
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Now the r powder is granulate more relic reproducil results tha ever befor

The new resin particles i Tresitope Diagnostic Kit vide a more effectual s dary binding site for t hormone.

The resin uptake der uniformly absorbs th rum-buffer solution, facil simplicity of test proce and is a key factor in yie reliable, reproducible re

***NOTE:** While the resi take test is a very useful the evaluation of thyroid



tion, it should not be used as the sole basis for such an evaluation. In any patient, the clinical state is probably the best indication of thyroid status, and *any* laboratory test must be interpreted with caution when test results do not agree with clinical evidence.

There is a Tresitope Diagnostic Kit to meet your needs. The 12-test kit containing 10 light-resistant (amber) vials of solution for serum testing, plus 2 vials for use with reference samples, is designed to save refrigerator space. The vials of radioactive test solution are packaged separately and are the only parts requiring refrigeration. A handy styrofoam platform holds the vials. One end of the platform is modified to facilitate suction washings of the resin powder.

The Tresitope Diagnostic Kit is also available as a 105-test kit and a bulk vial kit. The 105-test kit contains 100 light-resistant (amber) vials of solution for serum testing, plus 5 vials for use with reference samples. The vials of radioactive test solution are packaged separately with these two kits and are the only parts requiring refrigeration. Included is a sufficient supply of tubes of resin powder and individual droppers for each test.

The bulk vial kit contains a 60 ml. bottle of test solution with a sufficient number of plastic tubes of resin powder to perform at least 105 tests.

IMPORTANT

Use appropriate radiation precautions in handling, identifying and discarding all radioactive material. Remember that minute amounts of radioactivity remain on components used in the test, including the styrofoam platform when it is used in performing the test, and particularly when the Tresitope Suction Method is used for a number of tests.

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Division of Nuclear Medicine
New Brunswick,
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Medotopes®

TRESITOPE® DIAGNOSTIC KIT

Resin Uptake Kit with Liothyronine I 125 Buffer Solution

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FIGURE 1. SERIAL SCINTIPHOTOS. ANTERIOR VIEW.

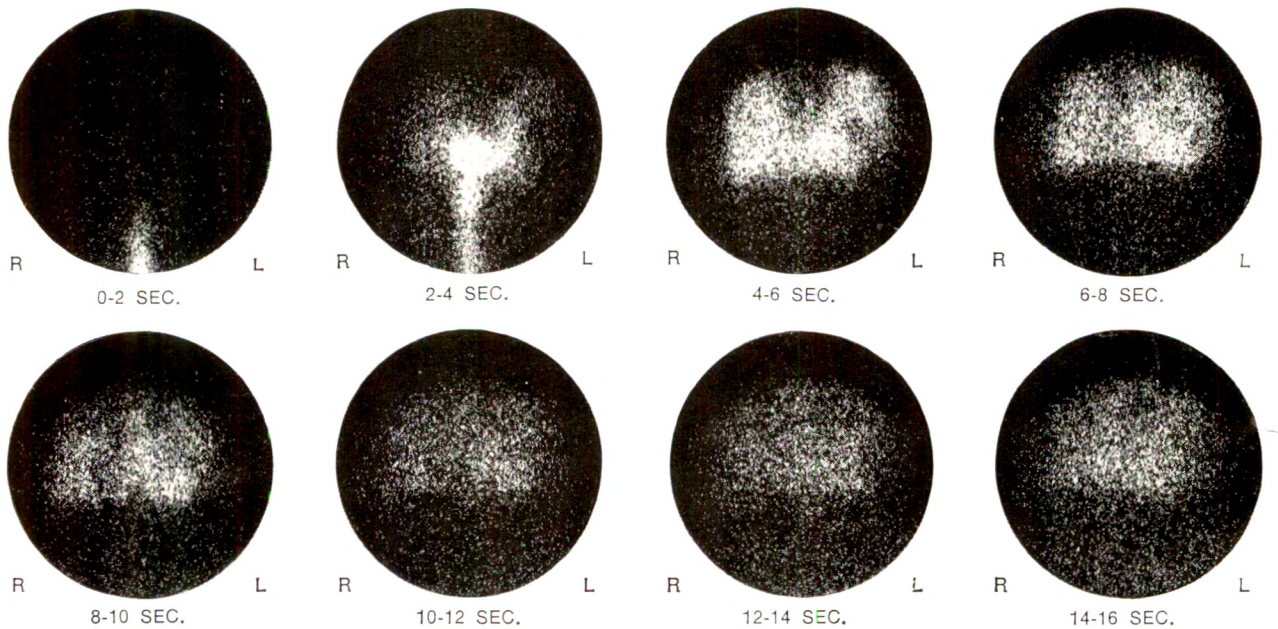


FIGURE 2.
AREAS-OF-INTEREST.
ANTERIOR VIEW.

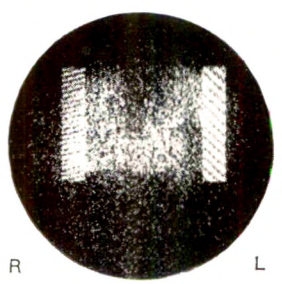


FIGURE 3. PULMONARY DILUTION CURVES, ABNORMAL.
Traced from original chart recordings for clarity of reproduction.

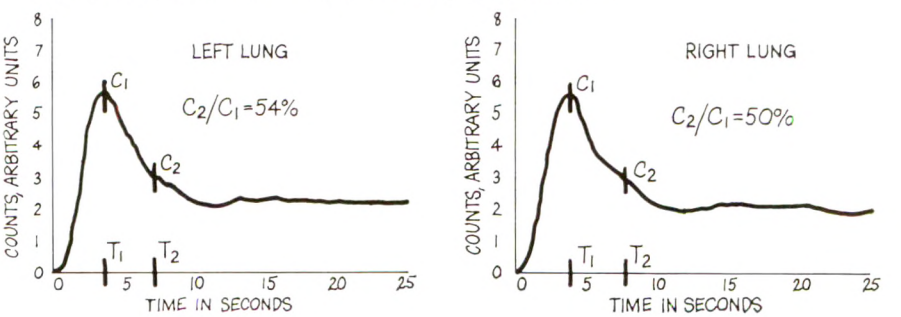
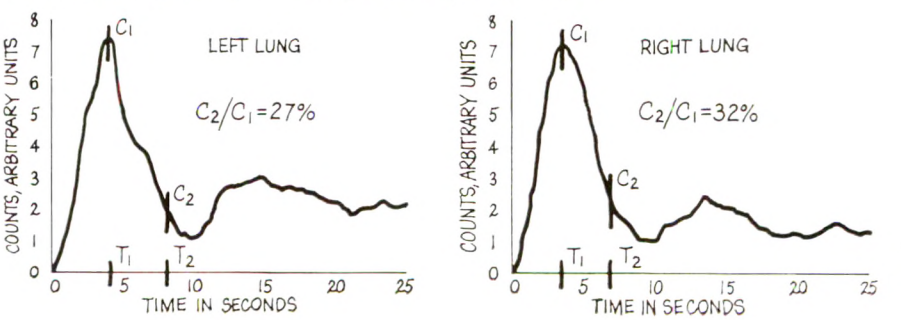


FIGURE 4. PULMONARY DILUTION CURVES, NORMAL.
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Th Cardiac Dynamic Study

A Dynamic Technique Using the Nuclear-Chicago Pho/Gamma® Scintillation Camera and Data-Store/Playback System

This study combines serial scintiphotos of the circulation of ^{99m}technetium pertechnetate through the heart and lungs, photographed from the Pho/Gamma Scintillation Camera, with a time-concentration curve of the pulmonary circulatory dynamics using the Data-Store/Playback Accessory and a dual-channel ratemeter/dual-pen chart recorder.

SETTING UP. The patient is positioned beneath the Pho/Gamma detector so that the heart and lungs are included within the field of view. For adults, a central venous catheter is inserted and the tip is advanced to the superior vena cava. For children, a percutaneous femoral venous puncture is performed.

ISOTOPE AND DOSE. 50 microcuries/lb. of ^{99m}Tc pertechnetate are injected as a bolus. This is followed by a sterile saline "flush." It is imperative that the tracer be administered as a bolus for proper interpretation of the pulmonary dilution curve.

DATA ACCUMULATION. Since the ^{99m}Tc pertechnetate is injected so close to the heart, serial hand-pulled scintiphotos are started immediately. Each exposure is for 1-2 seconds and no more than eight films are necessary. Alternatively, the automatic-sequencing 35mm camera may be used to obtain precisely timed sequential images.

The Data-Store/Playback Accessory plays an important role in the examination. The entire sequence is recorded in a high-resolution digital format (256 x 256 matrix) on the magnetic tape recording system. Subsequent replay of the tape allows reconstitution of the serial images at any desired frame rate and permits correction of film exposure factors to provide excellent scintiphotos. The study may be viewed on the system's variable-persistence oscilloscope during both original recording and upon tape replay.

The pulmonary dilution curves are obtained by choosing two separate areas-of-interest, one corresponding to the right lung field, the other to the left lung field. With this system's variable controls, these areas-of-interest may be rectangular or oval in shape. It is important, however, that these areas-of-interest correspond only to the lung fields, and no portion of the heart or great vessels should be included. Time-activity curves are generated with the dual ratemeter/recorder with a time constant of 0.5 seconds and a chart speed of 12 inches/minute.

CASE HISTORY. The clinical study on the opposite page is that of a seven-year-old child suspected of having a small left-to-right intercardiac shunt based on the characteristics of a systolic murmur. The child was not cyanotic. Following the diagnostic nuclear-medicine procedure, the patient was catheterized. A ventricular septal defect with a 1.2-to-1 left-to-right shunt was revealed as determined by standard dye dilution curves. In addition, there was a supervalvular obstruction of the pulmonary artery. Systemic pressures were observed in the right ventricle suggesting the diagnosis of an "Acyanotic Tetralogy of Fallot."

EVALUATION. The serial two-second images (Fig. 1) were produced upon replay of the Data-Store/Playback Accessory. The bolus of ^{99m}Tc pertechnetate is clearly seen in the inferior vena cava (0-2 sec.), having been injected into the right femoral vein. The tracer, thereafter, flows into the right atrium (2-4 sec.), then into the right ventricle and out through the pulmonary artery into both lung fields (4-6 sec.). Later frames show the return of the tracer to the left atrium, the left ventricle, and then out the aorta.

The pulmonary dilution curves were produced by adjusting the area-of-interest controls of the Data-Store/Playback Accessory, causing the areas-of-interest to correspond to the right and left lungs as indicated by the intensified areas seen on the representative scintiphoto (Fig. 2). The resulting pulmonary dilution curves (Fig. 3) show a rapid rise in count rate to a peak count rate C_1 at time T_1. $T_1 - T_0$ is the interval from time of rise onset to time of peak activity. At time T_2 ($T_2 - T_1 = T_1 - T_0$), count rate C_2 is determined from the curve. As shown, C_2 is 50 - 54% (C_2/C_1) of count rate C_1. These curves are abnormal and suggest the possibility of a left-to-right shunt. Normally, C_2/C_1 is less than 40% as shown by normal curves (Fig. 4).

CONCLUSIONS. The diagnosis of a left-to-right shunt was confirmed in this case, both at cardiac catheterization and at surgery.

An abnormal pulmonary dilution curve, it should be noted, does not indicate the anatomical location of the defect, nor does it indicate the severity of the left-to-right shunt. This cardiac dynamic study should be considered only as a screening procedure. In the event of an abnormal radionuclide pulmonary dilution curve, further diagnostic procedures are indicated.

G-241

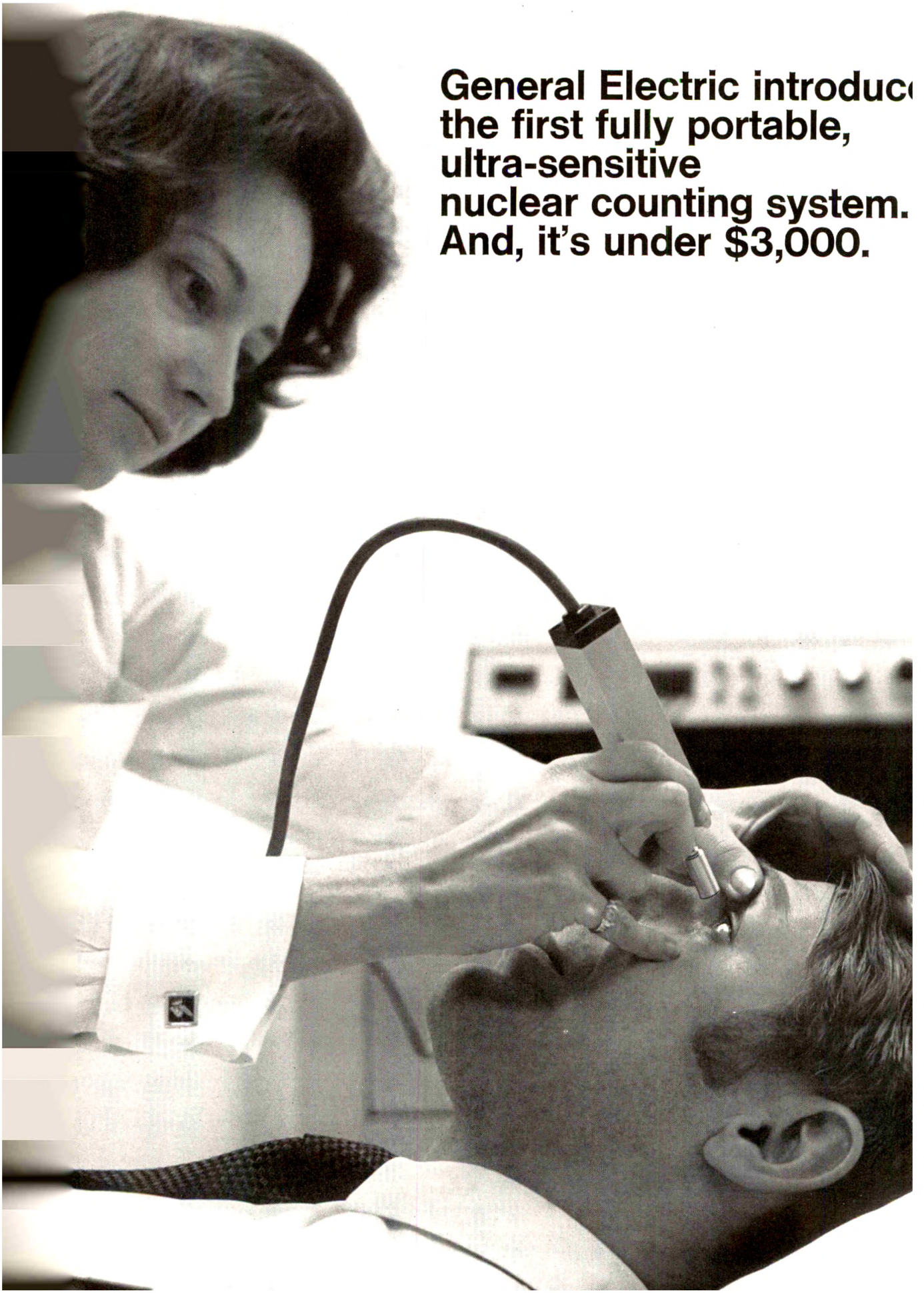
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And, it's under $3,000.

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You can now think of using ^{125}I for organ scanning, for example. For the first time, use low-energy emitting isotopes like ^{35}S, ^{55}Fe and ^{45}Ca in *in vivo* experimental work. X-ray fluorescence scanning and analysis. Tumor detection and measurement of tumor dynamics. Detection of ^{55}Fe x-rays in blood measurements and ^{51}Cr x-rays in spleen scanning. Carbon-14 research.

A patient's body heat creates no problem. The NUCLE EYE Monitor maintains its unique low-background counting capability from room temperature to 85°C. Without cooling.

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The eight-pound NUCLE EYE Monitor is fully portable —take it with you from laboratory to laboratory and even to patient bedside. Nickel-cadmium batteries give five hours of continuous operation before recharging.

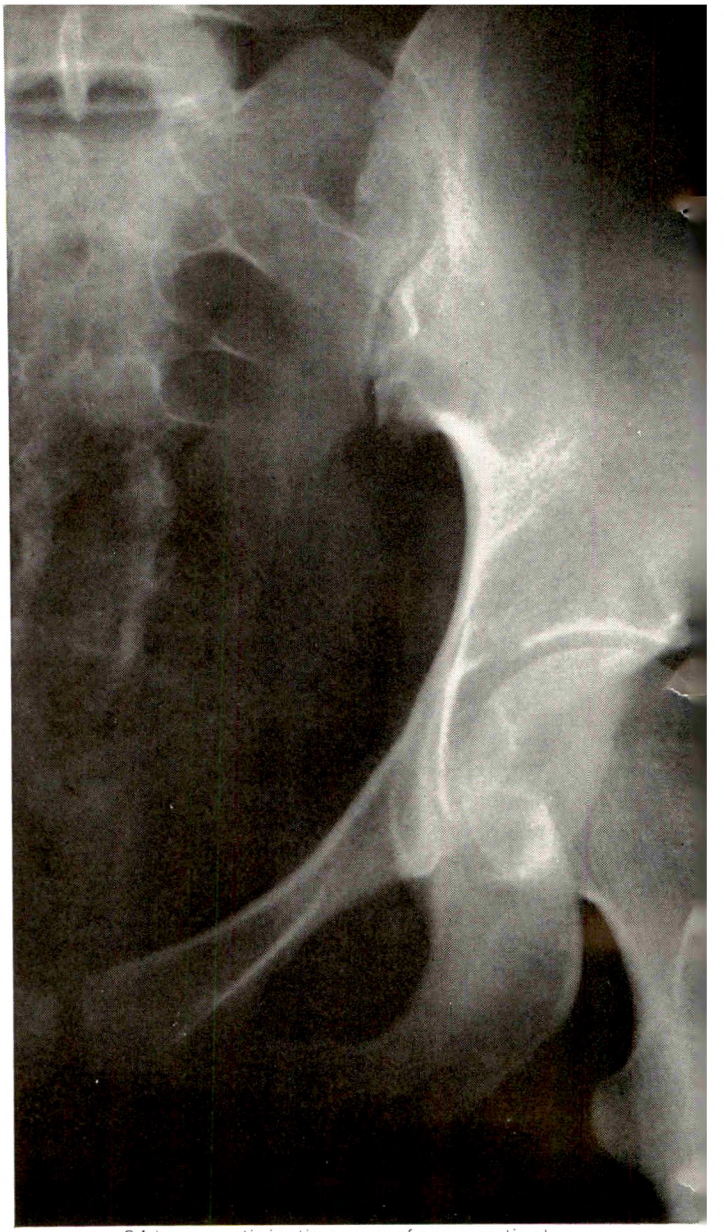


GENERAL ELECTRIC

162-51A



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now you see it

now you don't

With Salpix as your contrast medium, your gynecologic patient needn't undergo the 24-hour postinjection x-ray usually required with iodized oils. Water-soluble Salpix leaves virtually no irritating radiopaque residue to produce foreign-body reactions, yet it provides the viscosity needed for adequate visualization.

Contraindications: Contraindications to hysterosalpingography include the presence of severe vaginal or cervical infections, existing or recent pelvic infection, marked cervical erosion or endocervicitis, and pregnancy. The procedure is contraindicated during the immediate pre- or post-menstrual phase.

Sensitivity Test: If indicated in the patient's history, an intracutaneous skin test or sublingual absorption observation may be done with 0.1 cc Salpix.

Warning: Not for intravenous use.

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CONTRAST MEDIUM

Salpix

TRADEMARK

EACH CC CONTAINS:
sodium acetrizoate 0.53 g and
polyvinylpyrrolidone 0.23 g

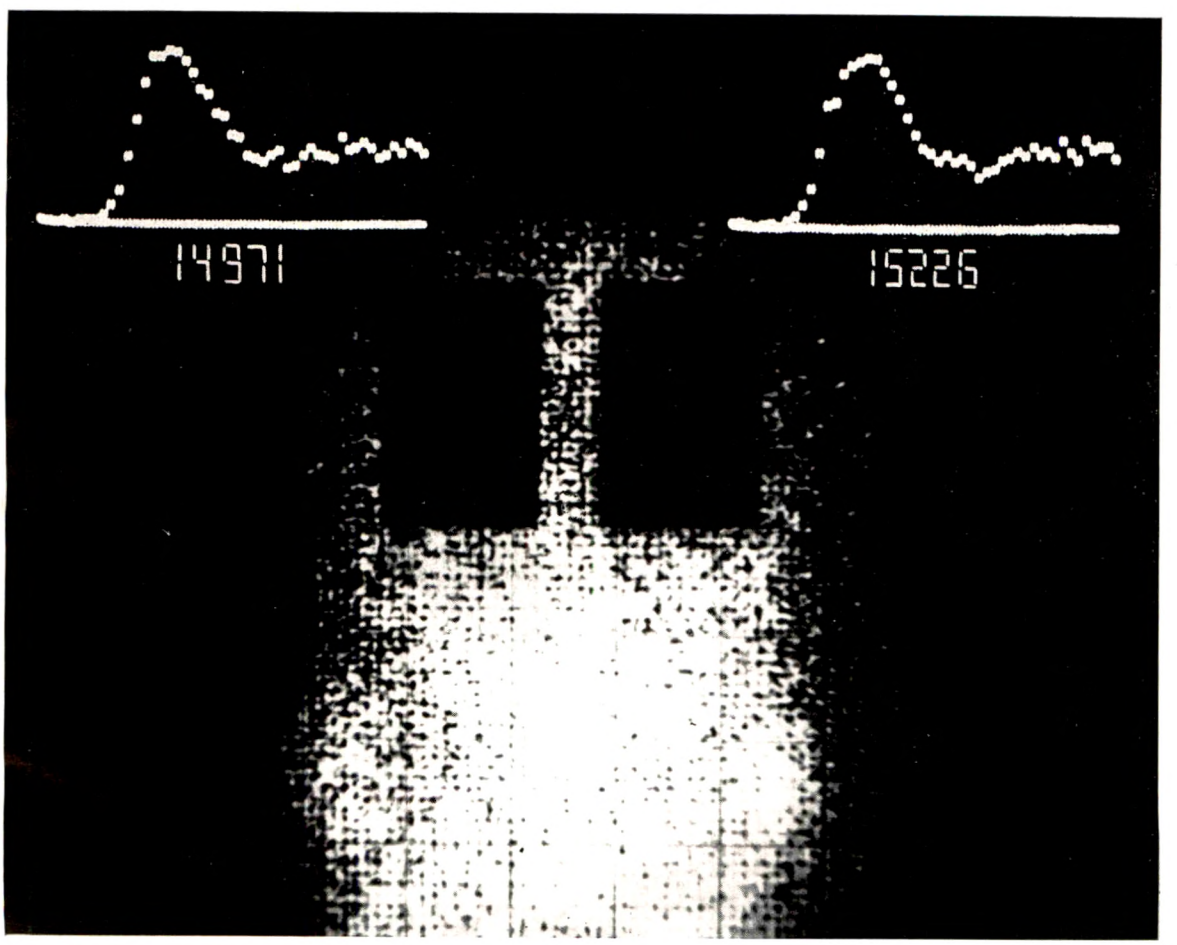
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The Picker Dynacamera 2:

The scintillation camera which permits the selection of dual regions of interest and simultaneously displays count rate vs. time and total integrated counts in both regions.

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Soon, patients at 49 hospitals will respond to the visible sign of progress in radiotherapy.



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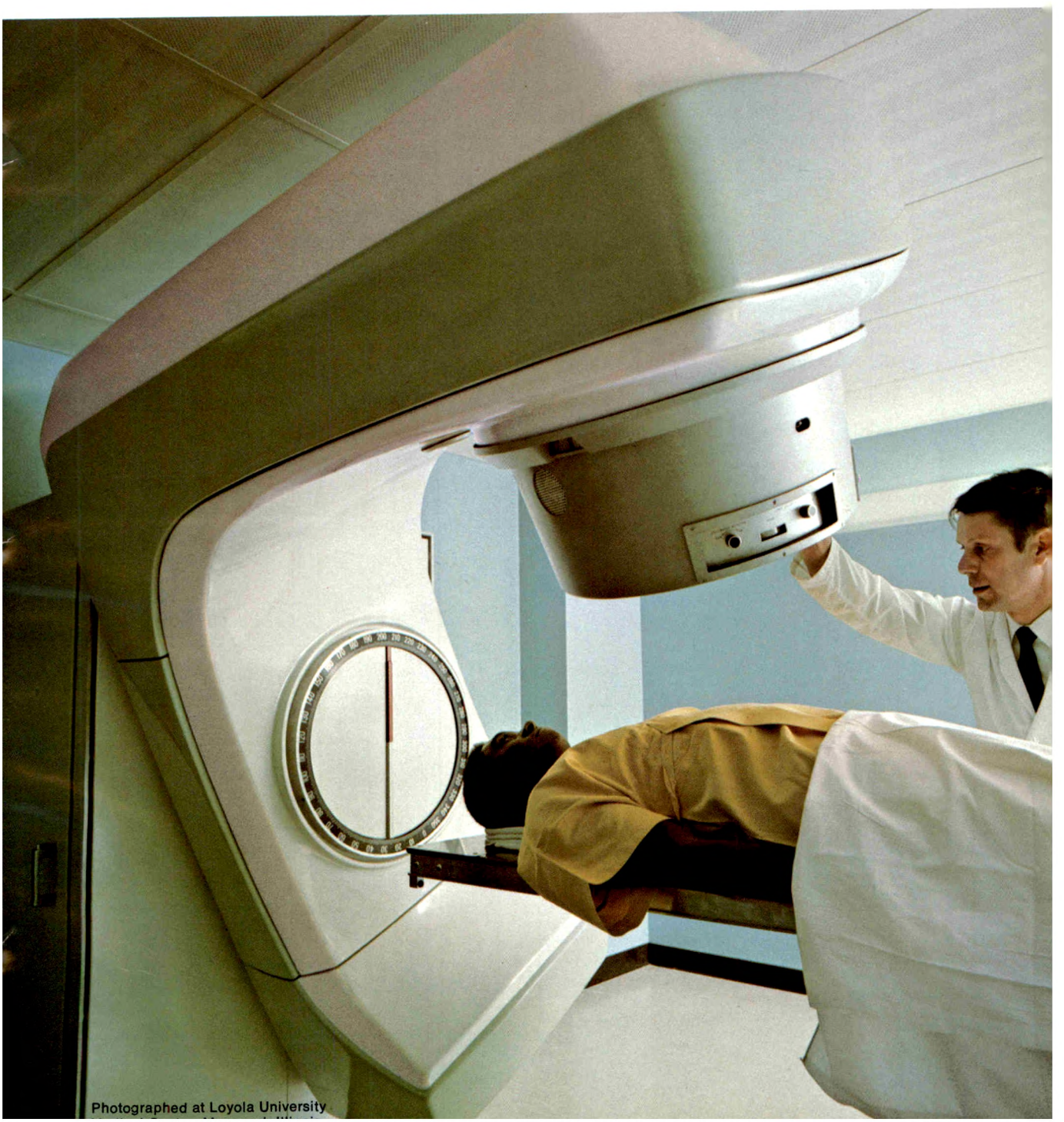
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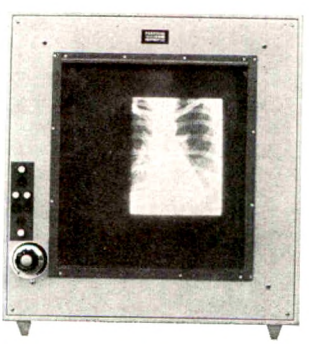
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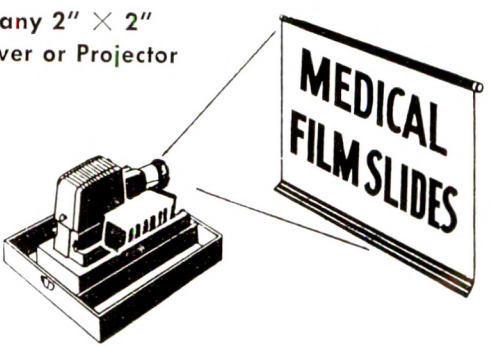
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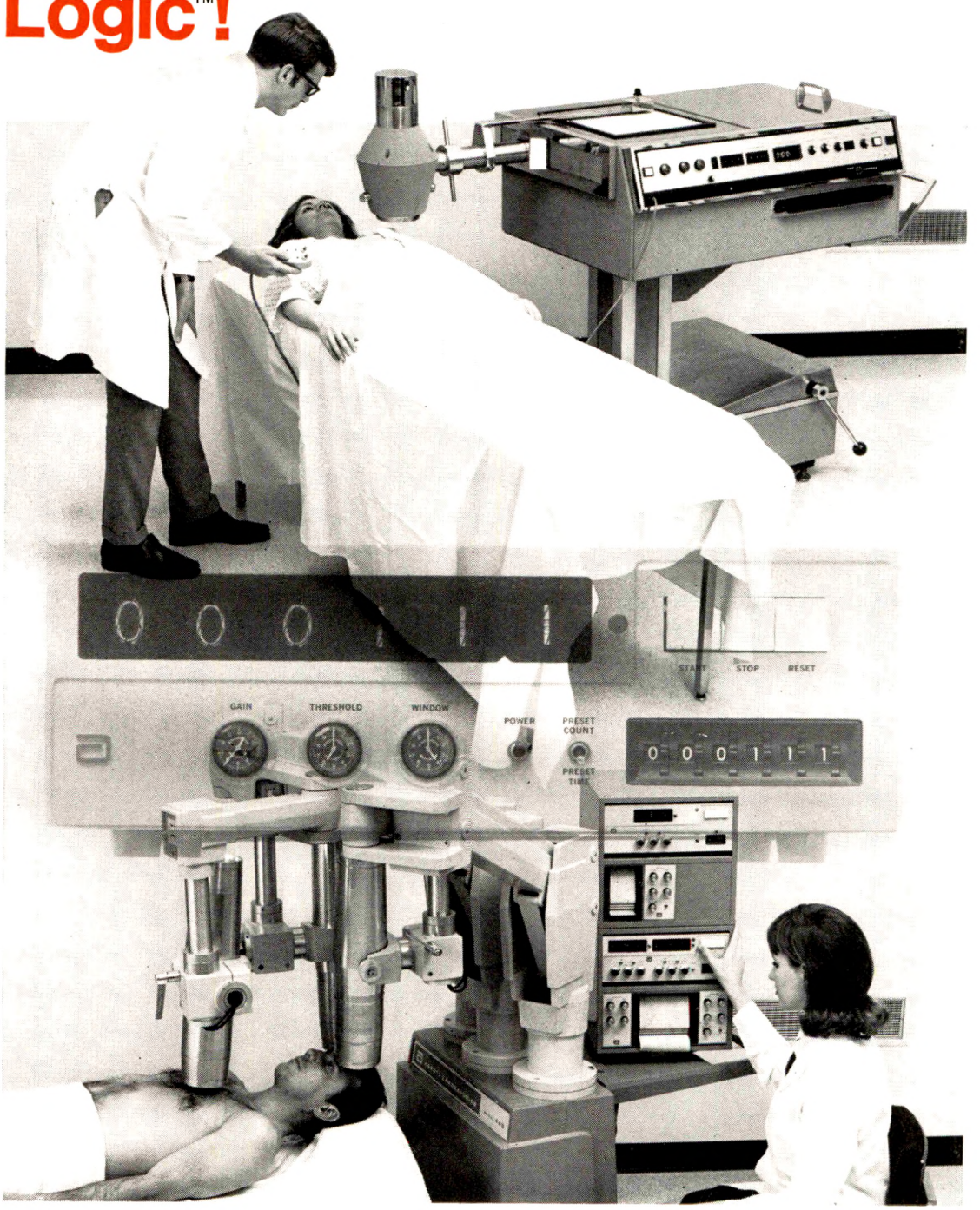
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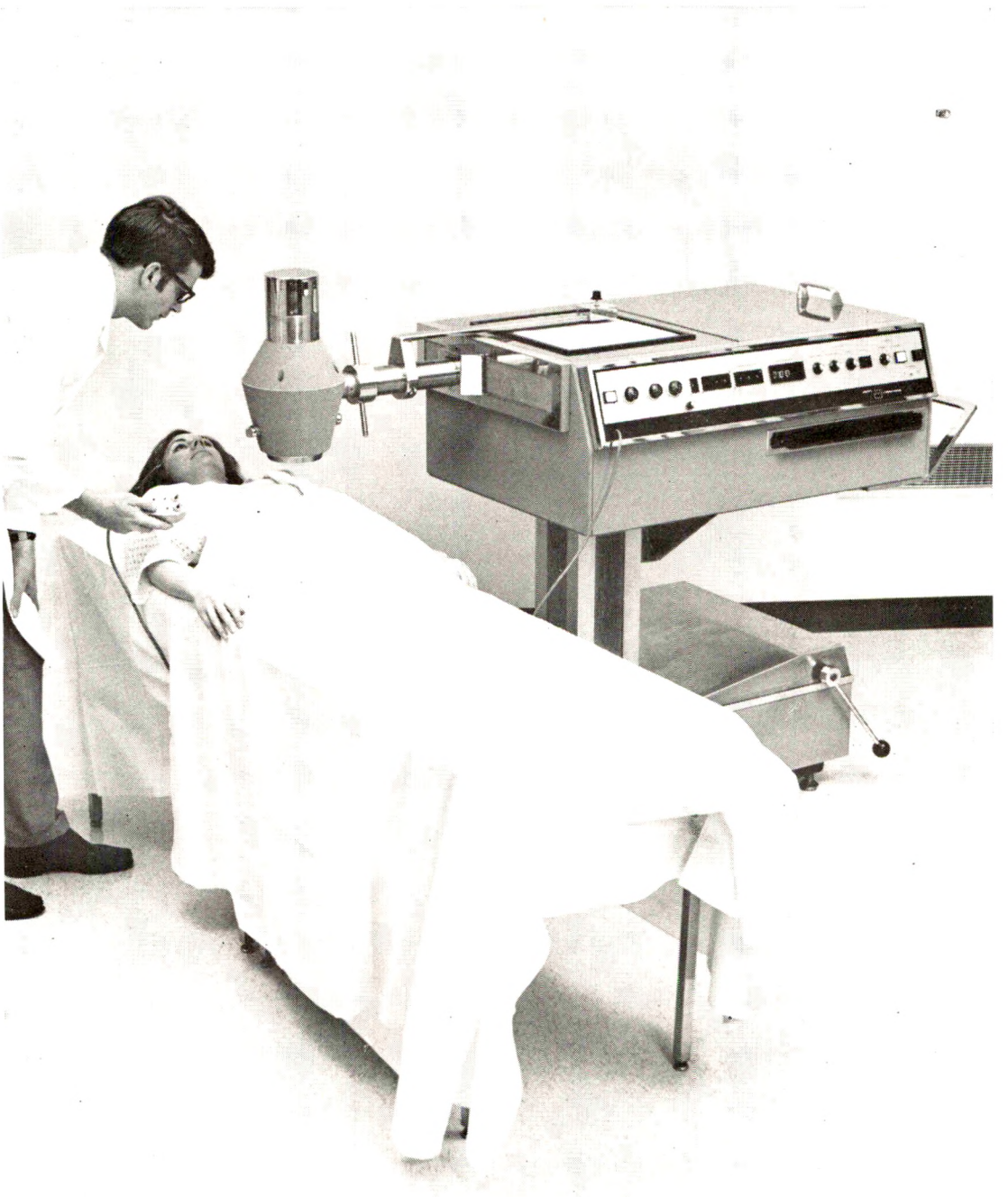
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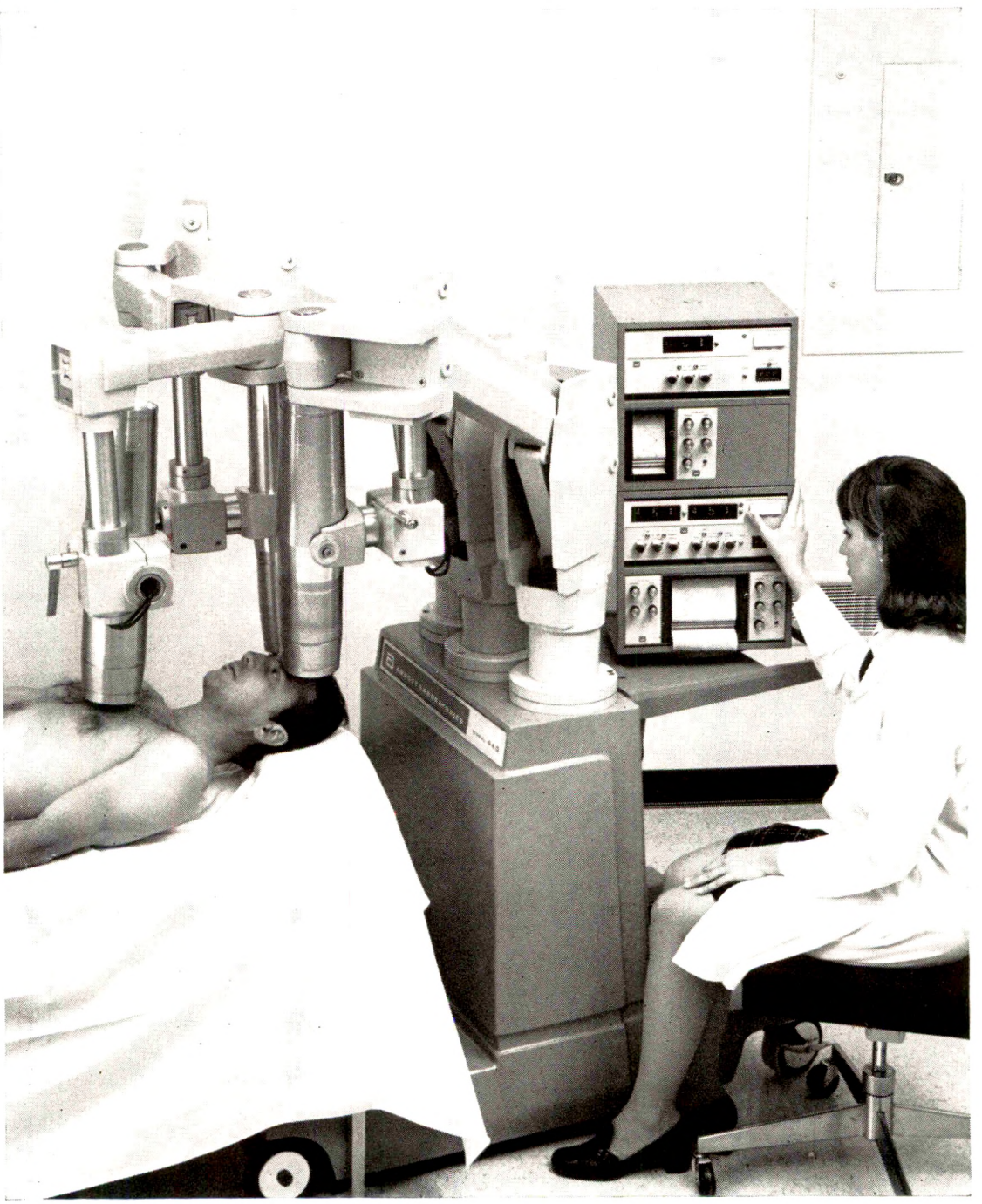
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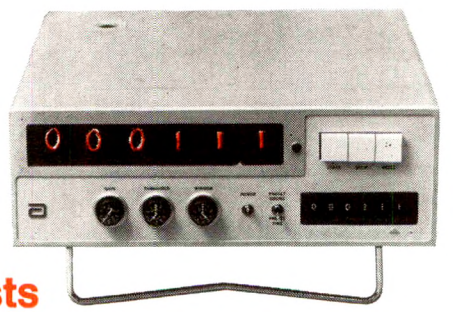
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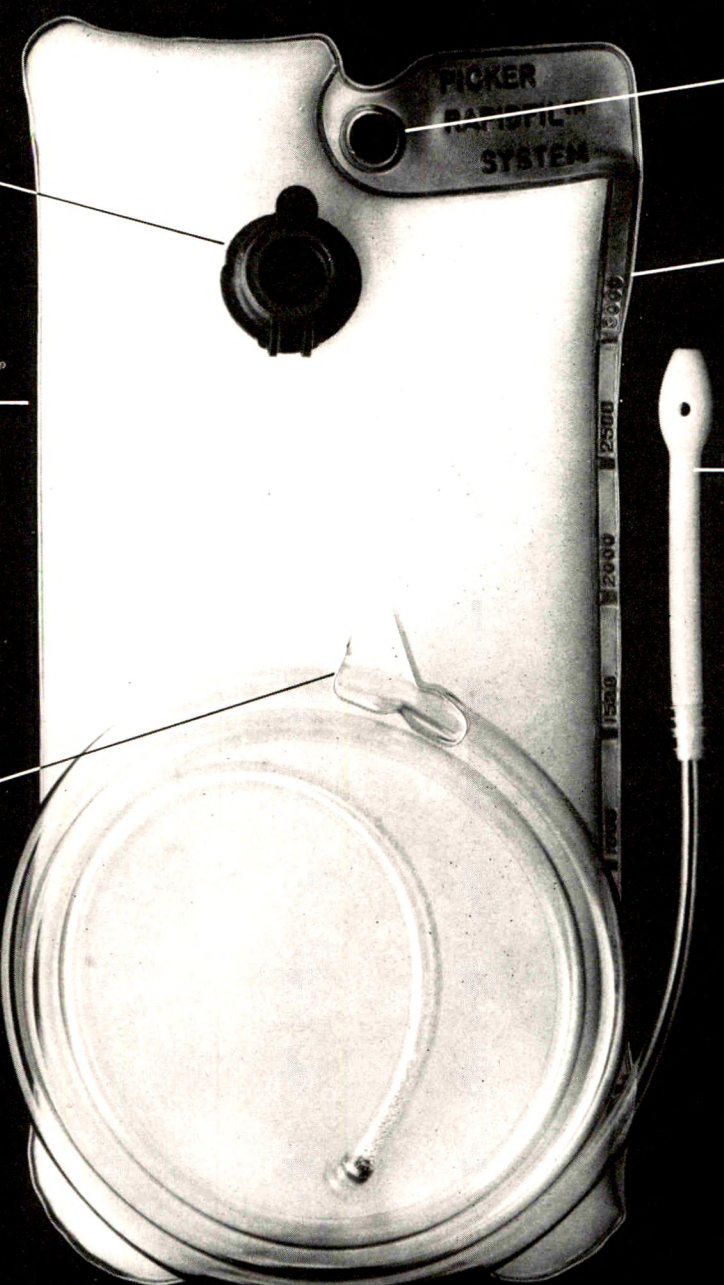
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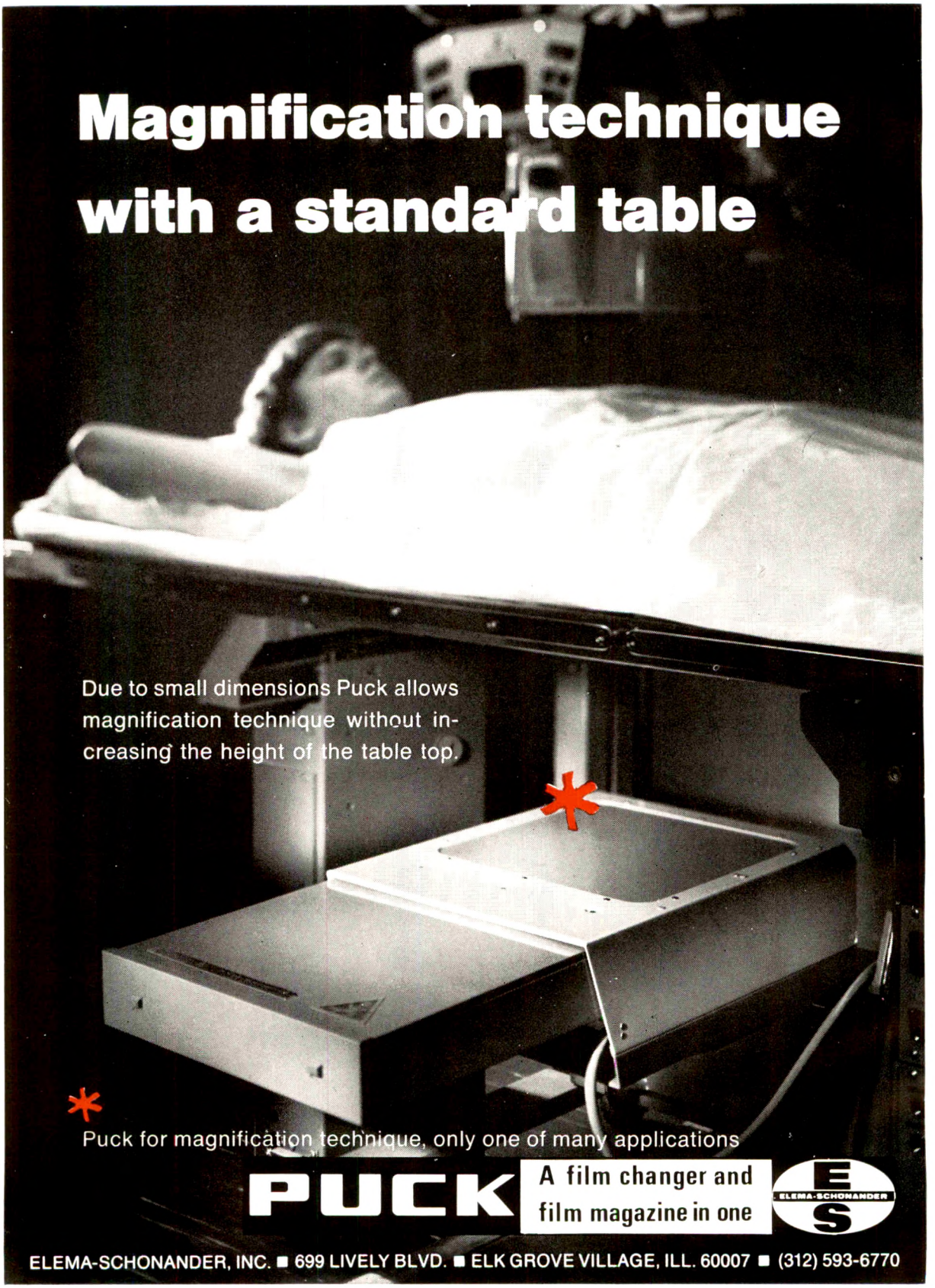
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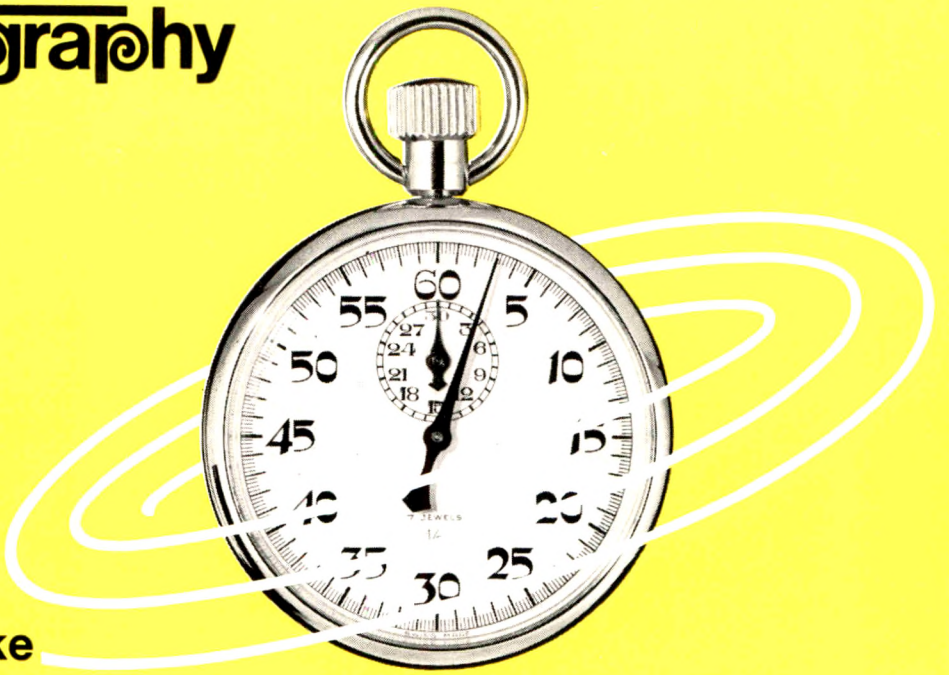
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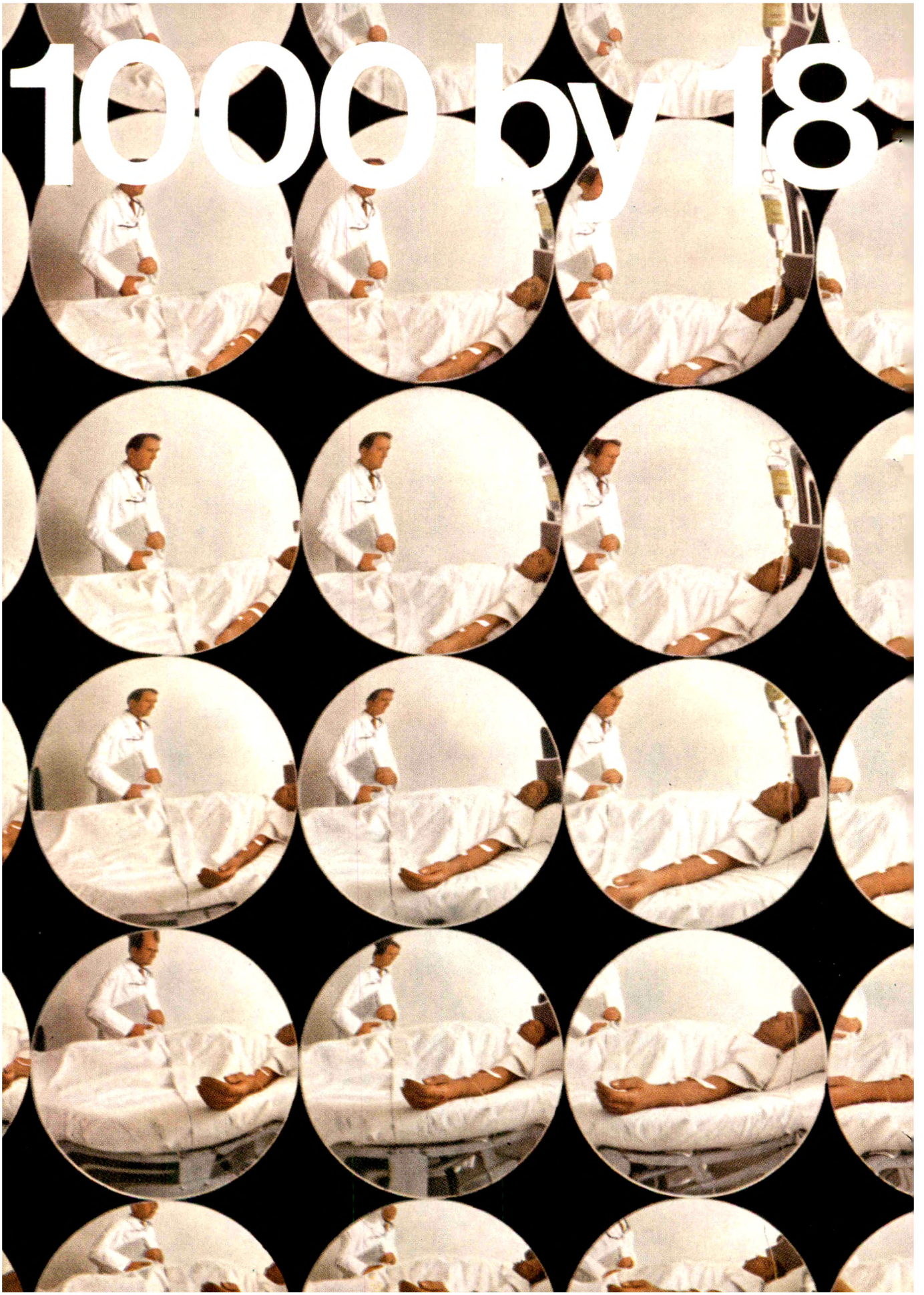


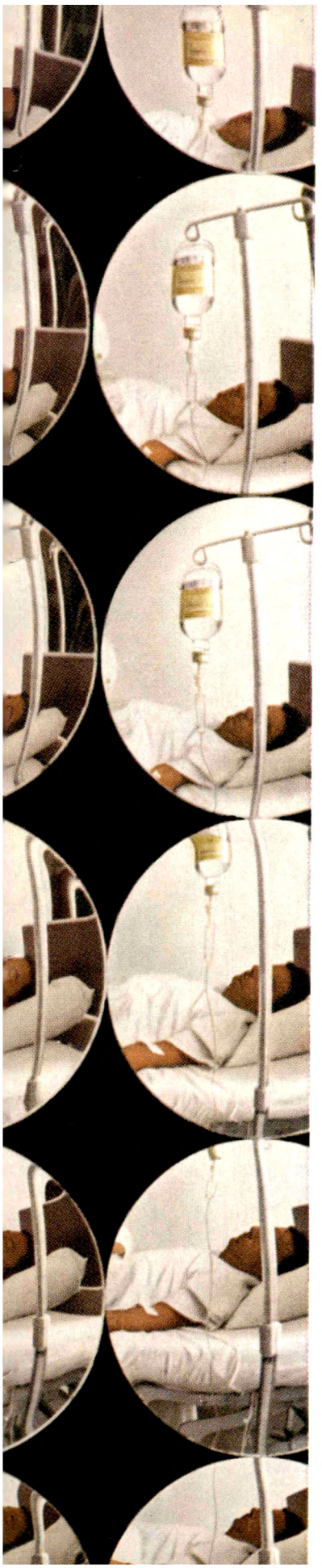
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Better visualization

Improvement over regular I.V. pyelography was shown specifically in 89.5% of 180 cases by 8 of the 18 investigators.[1]

Adverse reactions

Adverse reactions were generally mild and occurred in 6.7% of the cases. The most frequent reactions were nausea and urticaria (see brief summary on next page). (All Renografin solutions used were equivalent to 30% concentration.)

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The bottle containing Renografin-DIP is calibrated, to make dosage measurement easy. It has a standard screw-neck that accommodates all the usual attachments. It will take any top—screw-top, needle or spike.

Drip infusion pyelography is a simple, reliable, and relatively safe procedure for diagnosis of urologic disease process. Use Renografin-DIP whenever drip infusion pyelography is indicated.

1. Data on file at The Squibb Institute for Medical Research.

See next page for brief summary.

Whenever drip infusion pyelography is indicated

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Renografin-DIP® (Meglumine Diatrizoate Injection U.S.P.) for drip infusion pyelography provides a sterile, aqueous solution of 30% meglumine diatrizoate which contains approximately 14% (42.3 grams per 300 cc.) bound iodine and 0.04% disodium edetate as a sequestering agent. *The solution contains approximately 0.054 mg. (0.002 mEq.) sodium per cc. (16.2 mg. per 300 cc.).*

Contraindications: Contraindicated in persons hypersensitive to salts of diatrizoic acid. Urography is contraindicated in patients with anuria.

Warnings: A definite risk exists with the use of contrast agents in excretion urography in patients with multiple myeloma. There has been anuria with progressive uremia, renal failure and death. This risk of the procedure in these patients is not a contraindication; however, partial dehydration in preparation for study is not recommended since it may predispose for precipitation of myeloma protein in renal tubules. No therapy, including dialysis, has been successful in reversing this effect. Myeloma should be considered in persons over 40 before undertaking urographic procedures.

In cases of known or suspected pheochromocytoma, if the physician feels that the possible benefits outweigh the considered risks, radiopaque materials should be administered with extreme caution; however, an absolute minimum of material should be injected, the blood pressure should be assessed throughout the procedure, and measures for treating a hypertensive crisis should be available.

Contrast media may promote sickling in homozygous individuals when injected I.V. or intra-arterially. Although a history of sensitivity to iodine *per se* or to other contrast media is not an absolute contraindication, administration of meglumine diatrizoate requires extreme caution in such cases. Meglumine diatrizoate should be used in pregnant patients only when the physician deems its use essential to the welfare of the patient since safe use during pregnancy has not been established. Perform thyroid function tests prior to administration of meglumine diatrizoate since iodine-containing contrast agents may alter the test results. Perform urography with extreme caution in persons with severe concomitant hepatic and renal disease.

Precautions: Diagnostic procedures involving use of contrast agents should be performed under the direction of personnel with prerequisite training and a thorough knowledge of the particular procedure. Appropriate facilities should be available for coping with situations which may arise as a result of the procedure and for emergency treatment of severe reactions to the contrast agent itself; competent personnel and emergency facilities should be available for at least 30 to 60 minutes after I.V. administration since delayed reactions have been known to occur. These severe life-threatening reactions suggest hypersensitivity to the contrast agent. A personal or family histor of asthma or allergy or a history of a previous reaction to contrast agent warrants special attention and may predic more accurately than pretesting the likelihood of a reactio although not the type nor severity of the reaction in the in dividual. The value of any pretest is questionable. Th pretest most performed is the slow injection of 0.5—1.0 cc of the preparation which may be given through the needl to be used for the full dose. If no reaction occurs within 1 minutes, the full dose may be given; however, this does nc preclude the possibility of reaction. Should the test dos produce an untoward response, the necessity for continu ing the examination should be reevaluated. If deemed es sential, examination should proceed with all possibl caution. In rare instances, reaction to the test dose may b extremely severe; therefore, close observation and facil ities for emergency treatment are indicated.

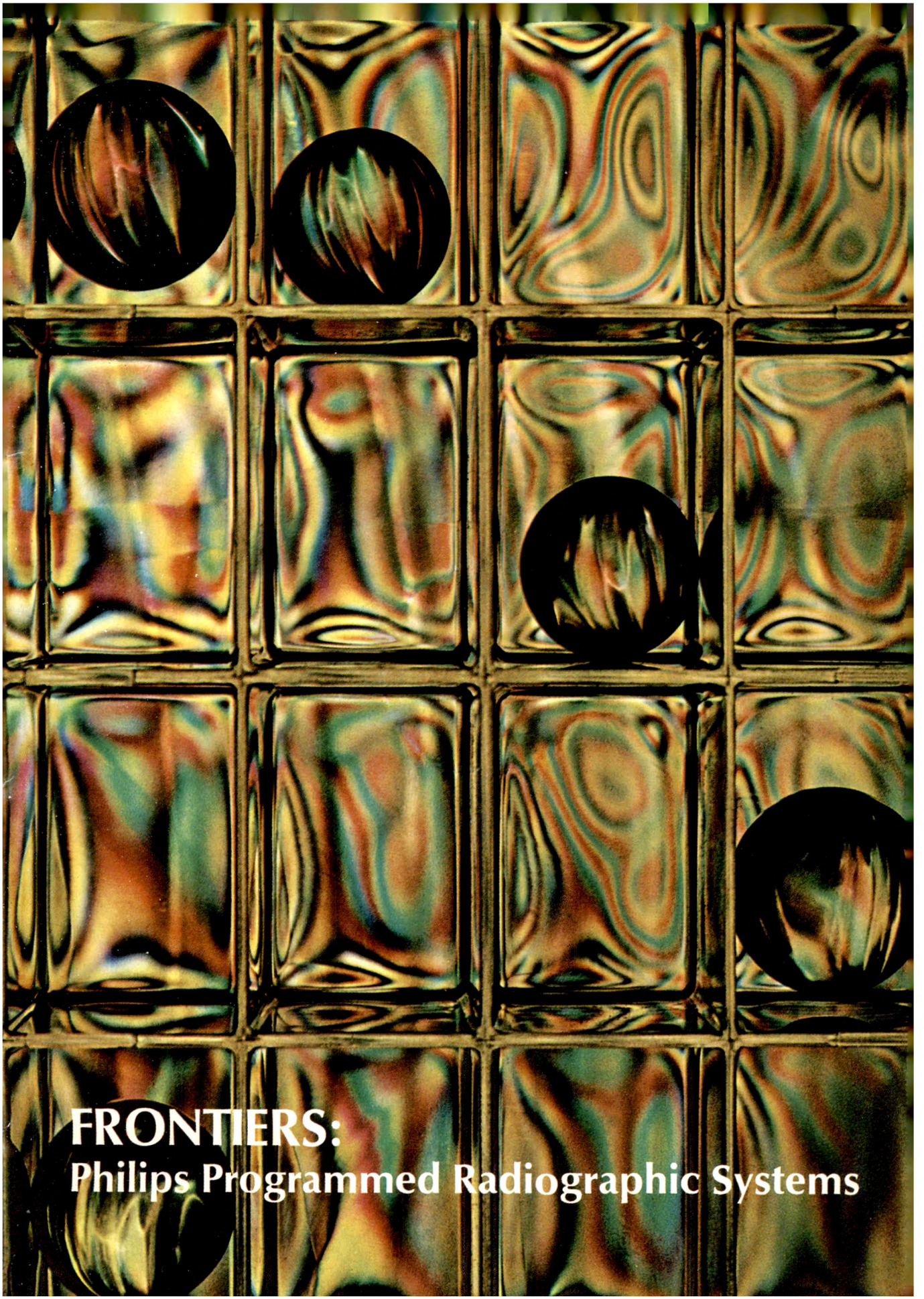
Renal toxicity has been reported in a few patients wit liver dysfunction who were given oral cholecystographi agents followed by urographic agents; therefore, if know or suspected hepatic or biliary disorder exists, administra tion of meglumine diatrizoate should be postponed follow ing the ingestion of cholecystographic agents. Conside the functional ability of the kidneys before injecting meglu mine diatrizoate.

The recommended rate of infusion should not be ex ceeded. The diuretic effect of the drip infusion procedure may hinder an assessment of residual urine in the bladder Adequate visualization may be difficult or impossible ir uremic patients or others with severely impaired rena function (see Contraindications).

Adverse Reactions: Reactions most frequently encoun tered with drip infusion pyelography are nausea and urti caria. Chills, metallic taste, vomiting, dizziness, a rise o fall in blood pressure, itching, flushing, or generalized feel ing of warmth, sneezing, etc. may occur and, rarely, may be severe enough to require discontinuation of dosage Severe reactions which may require emergency measures (see Precautions) are a possibility and include cardiovas cular reaction characterized by peripheral vasodilatation with hypotension and reflex tachycardia, dyspnea, confu sion, and cyanosis progressing to unconsciousness. Ar allergic-like reaction ranging from rhinitis or angioneurotic edema to laryngeal or bronchial spasm or anaphylactoic shock may occur. Temporary renal shutdown or other nephropathy may occur. Intravenous injection of meglu mine diatrizoate in a more concentrated formulation has produced a few instances of a burning or stinging sensa tion and of venospasm or venous pain.

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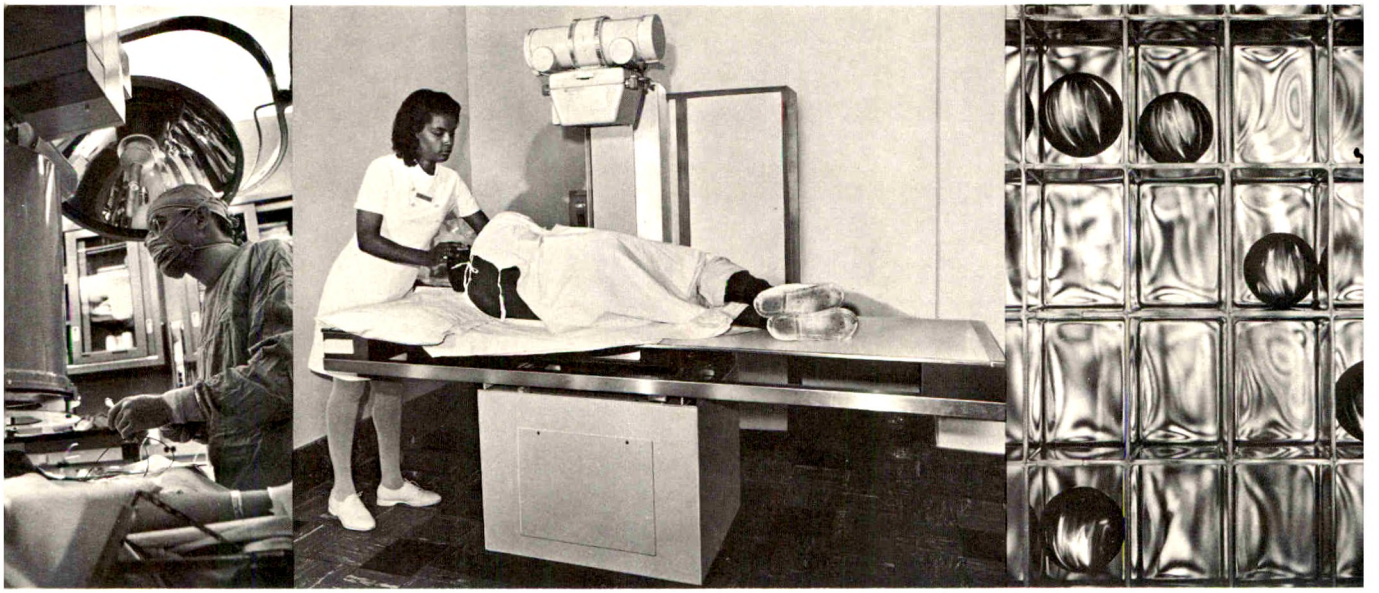


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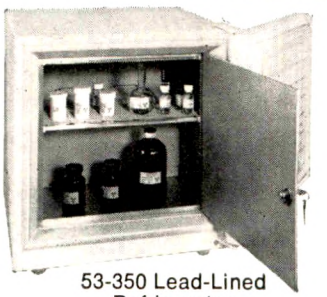
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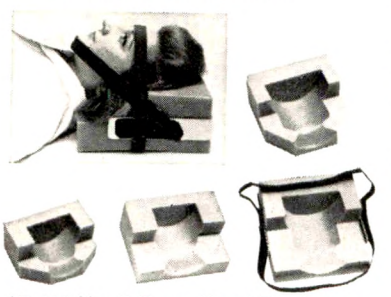


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PNEUMONIA OF ATYPICAL MEASLES*

RESIDUAL NODULAR LESIONS

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CHILDREN who have been immunized with inactivated virus vaccine may develop an atypical measles illness when exposed to "wild" measles virus.[5,13–15] This atypical form of measles is characterized by a 2 to 3 day prodrome of high fever, cough, headache, myalgia, and abdominal pain, followed usually by a peripheral maculopapular rash on the limbs including the palms and soles (Fig. 1*A*). The rash spreads centripetally (Fig. 1*B*), may become vesicular and petechial, and often is pruritic. Limb edema is frequently present. Moderate eosinophilia may occur. Pneumonia, heralded by cough and suggested by physical signs is confirmed by roentgenography which usually also demonstrates hilar lymphadenopathy and pleural effusion.[5] Our interest in this illness was stimulated by Cases I and II (summarized below) which suggest a nodular pulmonary residuum to the pneumonia. Case III indicates that the illness may occur with pneumonia but without skin rash.

REPORT OF CASES

CASE I. S. B. (SMH 65-04-87). This 9 year old girl was admitted by her physician to Strong Memorial Hospital on January 9, 1967 following 2 days of 104° F. fever, abdominal pain, anorexia, vomiting, and dry cough. She developed a maculopapular rash on her limbs and had dullness and absent breath sounds over her left chest. Roentgenograms showed left lower lobe segmental pneumonia, left subpulmonary pleural fluid, and left hilar lymphadenopathy (Fig. 2, *A* and *B*). The rash spread centripetally to the trunk and neck and its character changed during the course of the illness with the appearance of vesicles and later petechiae (Fig. 3, *A* and *B*). The erythrocyte sedimentation rate was 37 mm./hr. and a 7 per cent eosinophilia was noted by differential count of the blood smear. Urinalysis, stool examination, Tine test and *Proteus* OX-19

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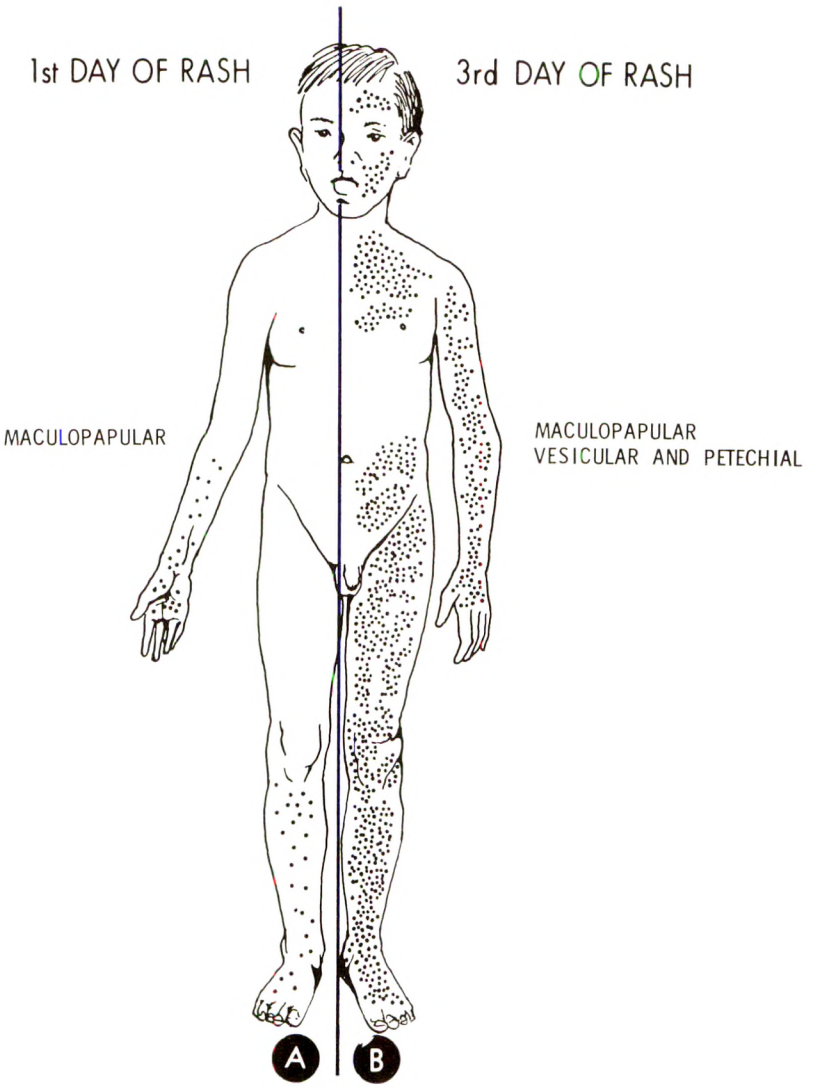


FIG. 1. Atypical measles illness. Schema of rash distribution. (*A*) First day. (*B*) Third day.

agglutination test were normal. Chest roentgenograms 3 days later showed an increase in left pleural effusion, and subsequent thoracentesis yielded 250 cc. of cloudy straw colored fluid. A smear of the fluid sediment contained eosinophils, polymorphonuclear leukocytes and atypical reticulum cells. No fungi or pathogenic bacteria grew from cultures of pleural fluid, throat, blood, urine or stool. She was less febrile and the rash was fading by the 5th hospital day. Follow-up roentgenograms on the 7th day showed partial clearing of pleural fluid and of left lower lobe pneumonia (Fig. 4, *A* and *B*). There was no specific diagnosis at the time of hospital discharge, but subsequently an exposure to clinical measles was established. She had received a 0.5 cc. subcutaneous injection of formalin inactivated (K) Edmondson B measles vaccine 2 months in succession and 1 live (L) attenuated Edmondson B measles vaccine the following month (KKL monthly sequence) $3\frac{1}{2}$ years before this illness. Retrospectively, a diagnosis of atypical measles illness was made based upon a rise in both the hemagglutination inhibition and complement fixation titers of late convalescent sera.

Chest roentgenography 9 months later showed a 5 cm. nodular lesion in the left lower

lobe (the site of the previous pneumonia) and at 20 months the nodular lesion was slightly smaller (Fig. 5, *A* and *B*). This nodule was thought to be either a pre-pneumonia lesion or a residuum to the pneumonia. The differential diagnosis for a pre-pneumonia pulmonary nodular lesion included granuloma, pulmonary sequestration, hamartoma, arteriovenous malformation, hemangioma, solitary neoplastic metastasis and hematoma. Several of these possibilities were excluded by history and clinical findings, but others could not be excluded. Since no previous roentgenograms of the patient's chest had been obtained, a pre-existent lesion had to be ruled out. Therefore the patient was admitted to Strong Memorial Hospital for re-evaluation almost 2 years after the acute illness, even though she appeared healthy and was asymptomatic. Hematocrit, complete blood cell count, urinalysis and skin tests for tuberculosis and histoplasmosis were again normal. A thoracic aortoarteriogram was normal (Fig. 6*A*) as was a left bronchogram (Fig. 6*B*). In view of her healthy status, she was discharged and has remained asymptomatic.

Case II. J. B. (SMH 68-06-17). This 10 year old boy was seen by his physician February 12, 1968, because of headache, painful cough, fever and skin rash. The character of the rash was maculopapular and it involved the upper and lower limbs, including the palms and soles. Four years previously he had been immunized against measles with 2 formalin inactivated Edmondson B and 1 live attenuated Edmondson B measles vaccines (KKL monthly sequence). A right lower lobe pneumonia was noted in the chest roentgenogram (Fig. 7*A*). No bacterial pathogens were isolated from the throat culture. About 8 days after presentation the rash had become partly petechial. Convalescent complement-fixing antibody titer for measles was obtained and showed a greater than nine-fold rise over the initial serum antibody titer ($<1:8$ to $>1:4{,}096$) confirming the diagnosis of atypical measles illness. Chest roentgenography at 3 months and at 10 months showed a nodular lung residuum at the site of the previous pneumonia (Fig. 7, *B* and *C*).

Case III. M. K. (SMH 45-54-94). This 9 8/12 year old girl was seen February 16, 1968, with the following symptoms and signs of 4 days' duration: malaise, fever of 102°F., chills, cough, and chest pain on deep breathing. She had been exposed 10 days previously to measles in school and had been immunized with measles vaccines (KKL monthly sequence) 4 years previously. Dullness to percussion was noted over her right upper chest. She had no skin rash. Roentgenogram of the chest con-

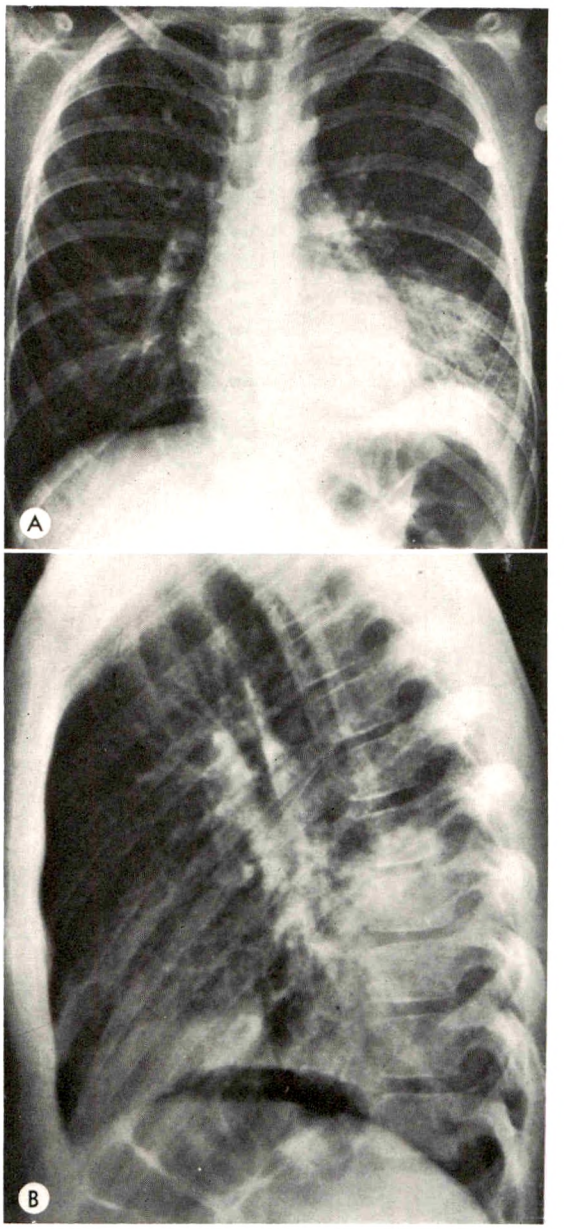


Fig. 2. Case 1. Left lower lobe segmental pneumonia, subpulmonary effusion and hilar lymphadenopathy. (*A*) Posteroanterior roentgenogram. (*B*) Lateral roentgenogram.



FIG. 3. Case 1. (*A*) Limb distribution of rash. (*B*) Close-up view.

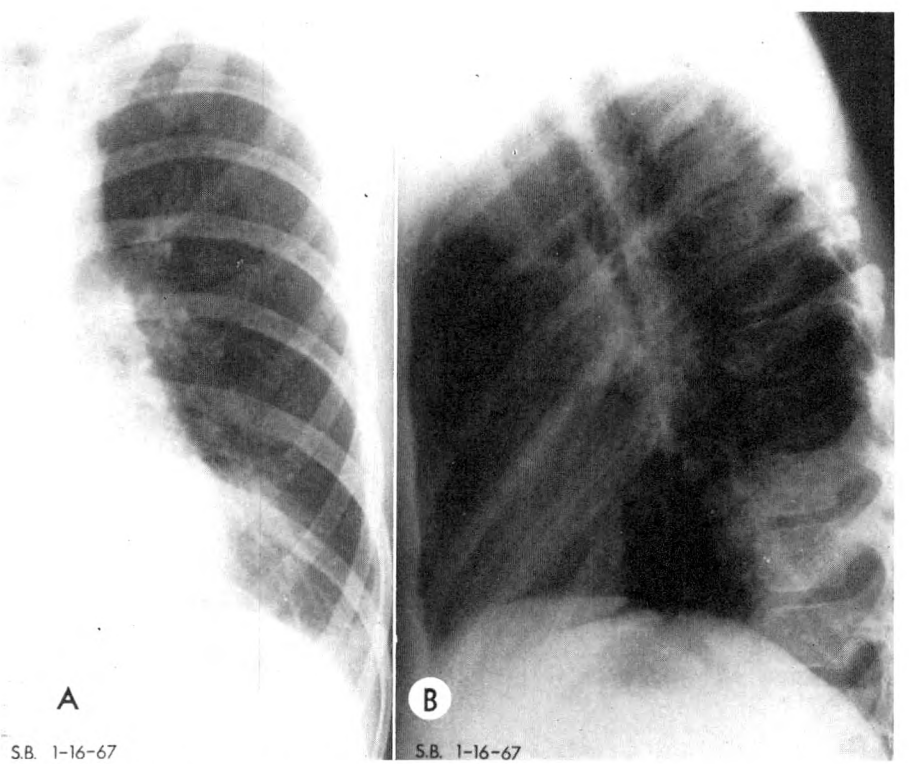


FIG. 4. Case 1. Partial clearing of pleural fluid and pneumonia 7 days after initial roentgenograms. (*A*) Posteroanterior roentgenogram. (*B*) Lateral roentgenogram.

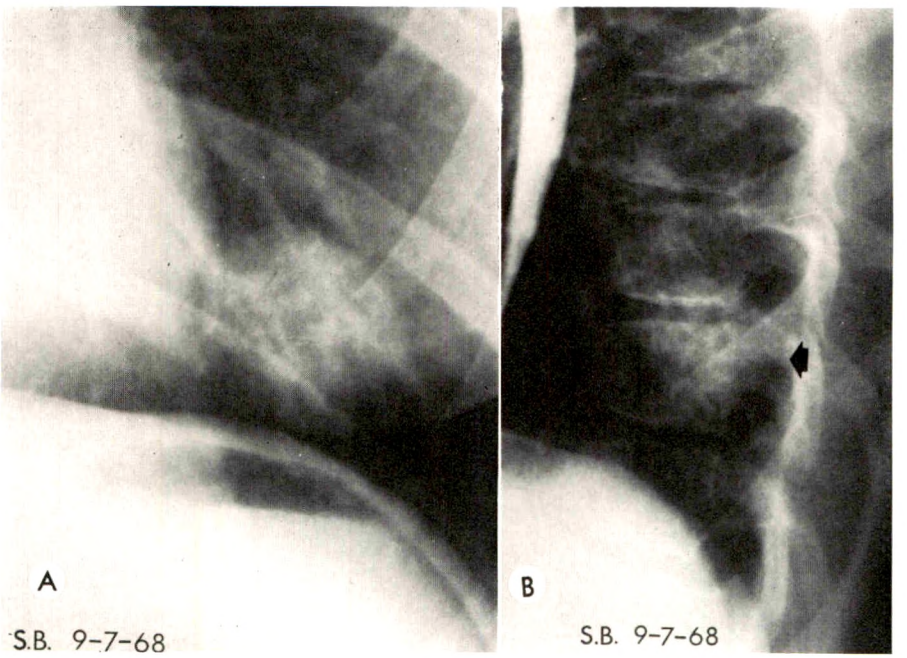


Fig. 5. Case 1. Nodular residuum at 22 months. (*A*) Posteroanterior roentgenogram—slightly magnified. (*B*) Lateral roentgenogram.

firmed a lobular pneumonia of the right upper lobe (Fig. 8). No bacterial pathogens were isolated from the throat culture. A presumptive diagnosis of primary atypical pneumonia was made. Three days later she was improving and 2 weeks later chest roentgenograms showed minimal residuum to pneumonia and she felt well. At no time during the illness was a rash evident.

Acute and convalescent sera were obtained for cold agglutinin titers and for determination of antibody response to measles virus and *Mycoplasma pneumoniae*. The cold agglutinin and *Mycoplasma* titers were negative, while there was a nine-fold rise in the measles complement fixation antibody titer ($<1:8$ to $>1:4,096$) confirming the presence of atypical measles illness. Follow-up chest roentgenog-

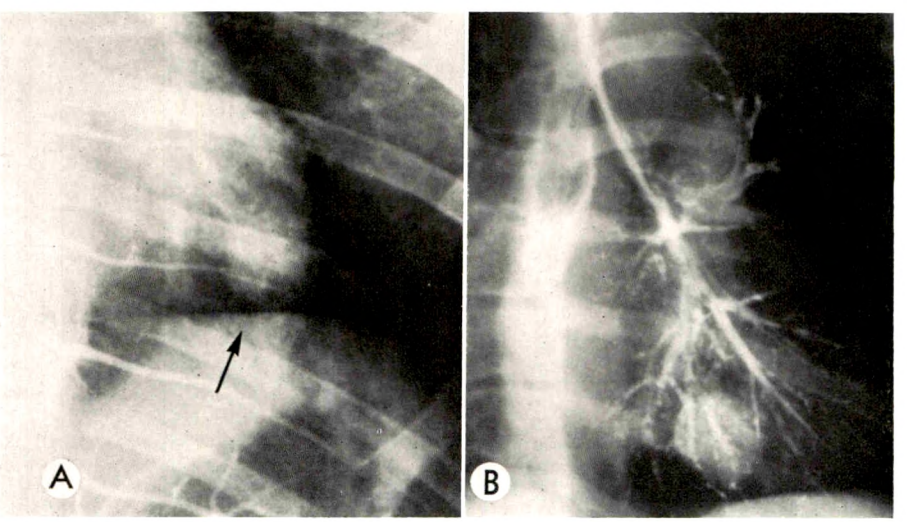


Fig. 6. Case 1. (*A*) Normal thoracic aortoarteriogram. (*B*) Normal left bronchogram. Note nodular residuum.

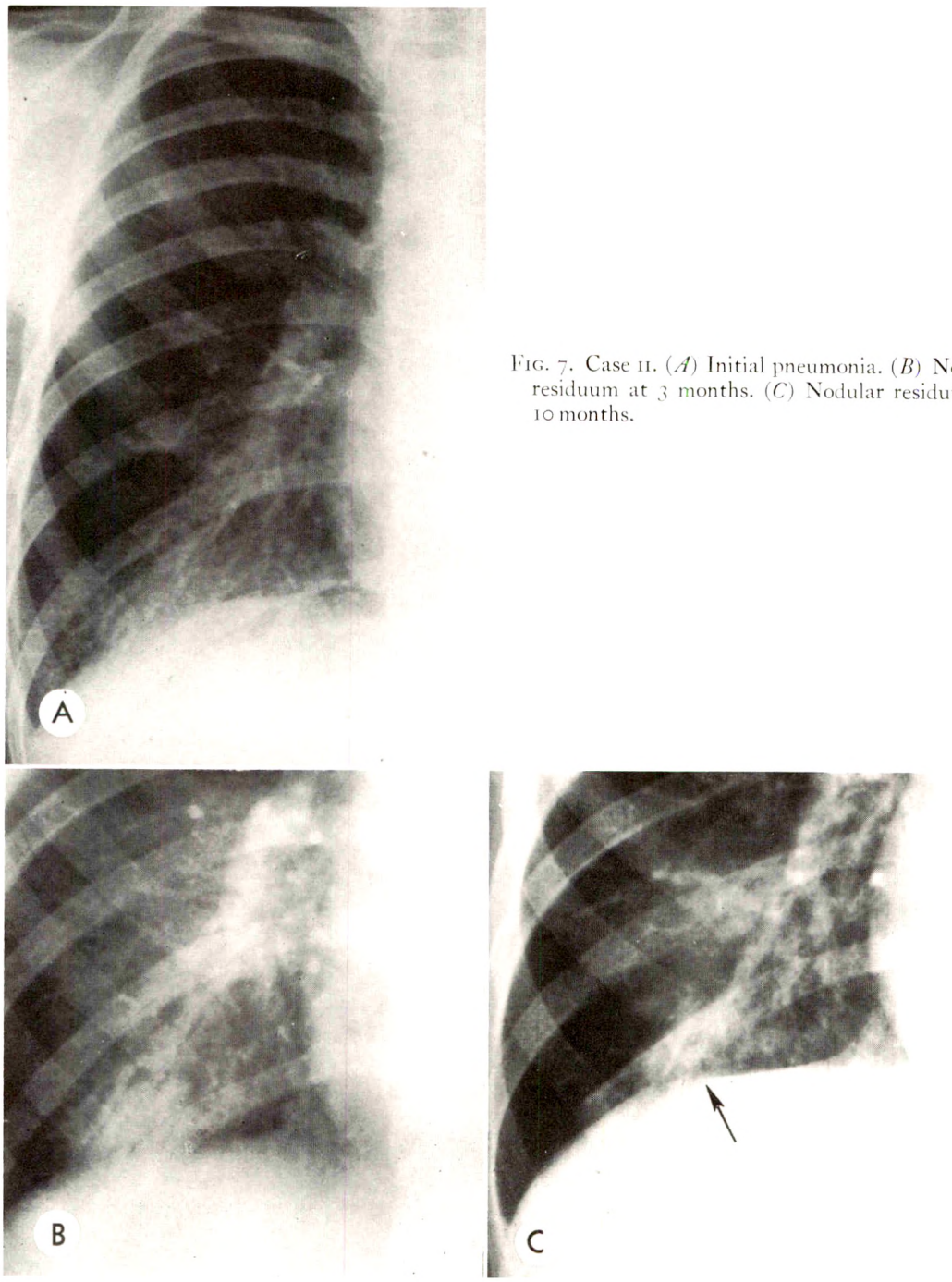


FIG. 7. Case 11. (*A*) Initial pneumonia. (*B*) Nodular residuum at 3 months. (*C*) Nodular residuum at 10 months.

raphy 1 year later showed no pulmonary residuum.

ROCHESTER SURVEY RESULTS

The 2 cases of nodular lesions in the lung in children who had atypical measles illness with pneumonia kindled interest in the possibility that they were in fact residual to the pneumonia. Survey of Rochester pediatricians yielded 8 other children who had atypical measles illness with pneumonia confirmed by roentgenography. In only 2

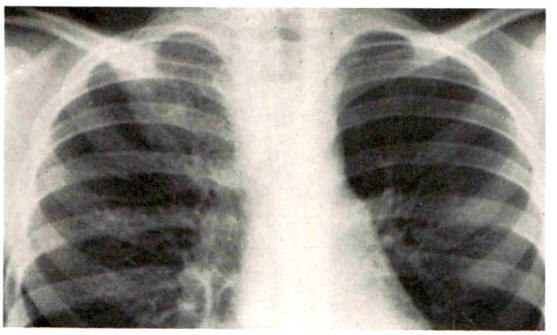


Fig. 8. Case III. Right upper lobe lobular pneumonia. Posteroanterior roentgenogram.

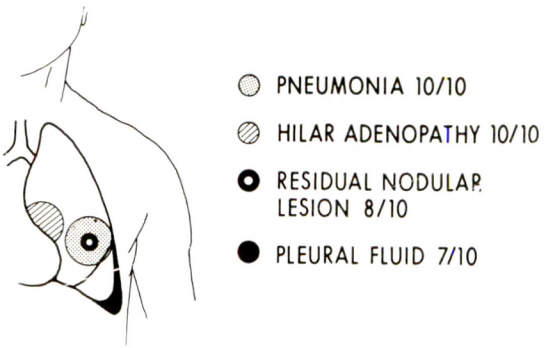


Fig. 9. Atypical measles illness. Schema of chest roentgenographic findings in 10 patients.

of these patients was there evidence for possible bacterial infection; in 1 a few colonies of *Staphylococcus aureus* were isolated by nasal culture, and in the other a few colonies of beta *Streptococcus hemolyticus* were isolated by throat culture. Of the total 10 children, all had 2 (monthly interval) 0.5 cc. subcutaneous injections of inactivated Edmondson B vaccine followed in the subsequent month by a 0.5 cc. subcutaneous injection of live attenuated Edmondson B measles vaccine. The patient ages ranged from $5\frac{1}{2}$ years to $10\frac{1}{2}$ years, and the interval between immunization and the atypical measles illness was 3 years to $4\frac{1}{2}$ years. Nine of 10 cases had the characteristic rash of atypical measles, but 1 child (Case III) had no rash. The diagnosis was confirmed in 9 out of 10 cases by serologic tests (Table 1), and in the other case, the clinical and the roentgenologic findings were characteristic of atypical measles. Eight of the pneumonias in this series were lower lobe in location, and 3 were upper lobe. All patients had hilar lymphadenopathy and 7 had pleural effusion (Fig. 9). In follow-up chest roentgenography, 8 patients had residual nodular lesions usually in the lung periphery (Fig. 9). Six of the nodular residua were lower lobe in location and 2 were upper lobe. These apparent alveolar residua have persisted for up to 30 months (Fig. 10).* The frequency of the nodular lesions in our cases argues against pre-existing processes and are strongly in favor of residua to pneumonia of atypical measles. Additional examples of the alveolar residua are shown in Figure 11, *A* and *B*. The frequency of the findings in our cases and a review of the literature[5,13,15] are presented in Table II.

* Follow-up chest roentgenograms on J. M. (Fig. 10 and Fig. 11*B*) at 30 months postpneumonia showed persistence of 1.2 cm. nodular residuum. It will be of interest in following these patients to determine if calcification of the residuum occurs.

DISCUSSION

The roentgenologic features of typical measles pneumonia are peribronchial inflammation, mild to moderate parenchymal infiltration (bronchopneumonic or lobular) and hilar lymphadenopathy.[9–11] These changes are merely corroborative evidence since the final diagnosis of measles rests with the clinical manifestations and serologic reactions. Lobar pneumonia in typical

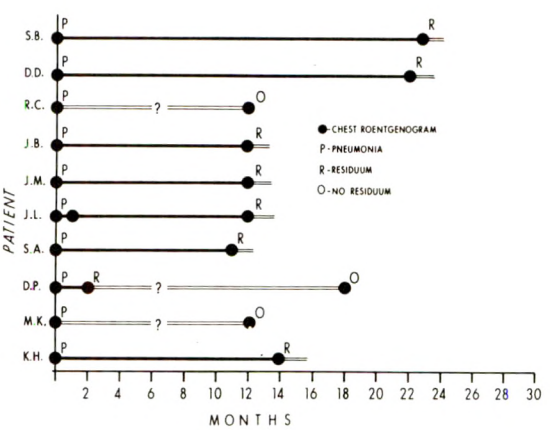


Fig. 10. Atypical measles illness. Roentgenographic residua to pneumonia.

TABLE I

ATYPICAL MEASLES ILLNESS WITH PNEUMONIA
SUMMARY OF ANTIBODY DATA

Patient	Age (yr.)	Days Post Rash Onset	Measles Antibody	
			HI*	CF†
S.B.	9½	2	<1:5	<1:8
		140	1:320	≥1:128
J.B.	10	1		<1:8
		19		>1:4,096
D.D.	6	4	1:40	1:16
		20	>1:640	1:2,048
		720	≥1:320	<1:8
R.C.	6	4	1:40	1:32
		20	>1:320	1:1,024
		720	1:160	<1:8
K.H.	7	3		<1:8
		7		>1:4,096
J.M.	5½	390	≥1:320	<1:8
J.L.	7	1		<1:8
		4		>1:4,096
S.A.	7	1	1:40	<1:8
		6	≥1:320	1:4,096
		380	1:160	<1:8
D.P.	10	5		1:256
		11		1:4,096
M.K.	9½	Days Post Illness Onset		
		4		<1:8
		17	≥1:320	>1:4,096
		380	≥1:320	<1:8

* HI = data from National Communicable Disease Center.
† CF = complement fixation.

measles is usually associated with superimposed bacterial infection.[3,4,16]

The pneumonia of atypical measles is a lobular or segmental infiltration with hilar lymphadenopathy and frequently pleural effusion. Pneumonia is a common finding in atypical measles illness in contrast to natural measles where it occurs less frequently.[5] The nodular pulmonary residuum is a finding that has not been observed following the pneumonia of typical measles.[9–11] The residua as observed in our cases have been fuzzy-bordered and have been confluent enough to suggest an alveolar location, occasionally abutting the pleural surface. The nodular shape of these 1.5 to 4 cm. lesions suggests a multisegmental distribution resulting from spread of the inflammatory process through interalveolar communications and lymphatics.[7]

The differential diagnosis for a *nodular pulmonary residuum* (solitary pulmonary nodule) in childhood includes the following: parenchymal cyst, arteriovenous malformation, hamartoma, granuloma, sequestration, hemangioma, organized pulmonary infarction or neoplastic metastasis. Distinction among the differential diagnostic possibilities and a nodular residuum to pneumonia may be possible by utilization of arteriography, bronchography and ultimately by surgical biopsy or resection and microscopy. None of the lesions described in the patients referred to in this report have been biopsied or resected.

The occurrence of "primary atypical pneumonia" due to measles in a patient previously immunized with inactivated measles virus vaccine has not been previously reported. We suspect that this form of altered reactivity to measles virus has occurred previously without being detected. Without a clue for an atypical measles illness (*e.g.*, a rash), the disease might not be suspected and specific diagnostic tests not performed. Patients with "primary atypical pneumonia" previously immunized with killed measles virus vaccine, and recently exposed to measles should stimulate consideration of atypical measles illness.

The mechanism involved in producing atypical measles illness has not been proven. Some evidence[2] strongly suggests an immune response to "wild" measles virus. Whether this response is an Arthus type reaction, as suggested by Buser[2] for the local reactions which occur at vaccine injection sites or delayed hypersensitivity or tissue injury mediated by antigen-antibody complexes has not been determined. Bellanti

TABLE II

ATYPICAL MEASLES ILLNESS WITH PNEUMONIA
FREQUENCY OF FINDINGS, LITERATURE AND THIS REPORT

Authors	Total Cases	Pneumonia by Roentgenogram	Rash	Limb Edema	Type of Measles Vaccine and Schedule
Rauh and Schmidt[15] March 1965	8	3	8	2	K*KK
Fulginiti *et al.*[5] December 1967	10	9	10	7	KKK(5 pts.) KKL†(5 pts.) KKL†
Nader *et al.*[13] April 1968	11	1	11	2	KKK†
Young, Smith and Glasgow	10	10	9	2	KKL

* Formalin inactivated Edmonson B.
† Live attenuated Edmonson B.

et al.[1] have evidence that many recipients of inactivated measles vaccine do not produce significant amounts of local secretory gamma A antibody, despite adequate serum antibody response. They suggest that as a result, exposure to natural measles results in viral replication in the respiratory tract, an accelerated response of serum antibody and formation of immune complexes in the lung with subsequent tissue injury. Several studies have suggested that delayed hypersensitivity to viral antigen is present in killed measles vaccine recipients. This hypersensitivity is not present in recipients of live vaccine alone.[6,8,12] Two-thirds of the patients of Fulginiti *et al.*[5,6] with delayed hypersensitivity had low or undetectable serum antibody. They suggest that children develop atypical measles when challenged with "wild" measles virus because of the discrepancy between serum antibody and delayed hypersensitivity.

It seems reasonable that lack of both local and systemic antibody would (accepting Fulginiti's hypothesis of causation due to delayed hypersensitivity[6]) result in atypical measles with rash on challenge with "wild" measles virus, whereas lack of local secretory antibody alone would allow viral replication in the respiratory tract with subsequent pneumonia without rash as in Case III.

Eight of 10 cases in this report have had tissue injury resulting in prolonged lung

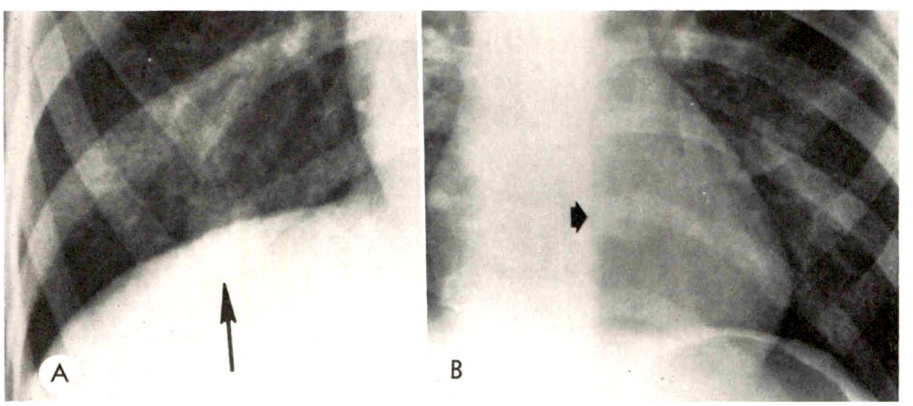


FIG. 11. (*A* and *B*) Examples of nodular residua in other patients.

residua. On this basis, we recommend follow-up chest roentgenograms in children with atypical measles illness and pneumonia. Moreover, to the list of childhood causes of nodular pulmonary lesions, the roentgenologist should add residuum to pneumonia of atypical measles.

SUMMARY

The pneumonia of atypical measles illness is a more severe pneumonia than that of regular measles and has been shown to be slow in resolution with a nodular residuum persisting as in most of our cases. Possible explanations and mechanisms for this phenomenon are mentioned.

Long term follow-up chest roentgenograms in children who develop atypical measles with pneumonia are recommended because of the frequency of nodular residua in our series.

The pneumonia of atypical measles illness is a new cause for nodular pulmonary lesions in children. An atypical measles illness with pneumonia may occur in the absence of skin rash.

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INTRAPULMONARY INTERSTITIAL EMPHYSEMA: A COMPLICATION OF HYALINE MEMBRANE DISEASE

By ROBERT E. CAMPBELL, M.D.*
PHILADELPHIA, PENNSYLVANIA

SIX years ago, the neonatologists at Pennsylvania Hospital began a comprehensive program for the clinical care of newborns with hyaline membrane disease (HMD). During the successive years, the roentgenologic examination of the chest has become increasingly more important in the early management of these neonates with severe respiratory distress. A recent review of the chest roentgenograms in this project unexpectedly revealed a significantly high association of intrapulmonary interstitial emphysema (IIE) with other extrapulmonary and intrapulmonary complications of HMD, such as pneumothorax, pneumomediastinum, pneumopericardium, extrathoracic air, and pseudocyst formation in the lungs. Since the association of IIE with these other abnormal collections of air in the chest did not appear coincidental, the chest roentgenograms were reviewed again to search for examples of IIE preceding the other complications of HMD. Two cases of IIE preceding the development of a pneumothorax were then discovered, and in a recent case, not included in the original series, the appearance of IIE lead to the correct prediction of a subsequent pneumothorax.

Although IIE in the newborn with respiratory distress is not a new entity, either roentgenographically[1,3,7,14] or pathologically,[6,9,15] its significance in the pathogenesis of other intrapulmonary and extrapulmonary complications has not been emphasized in the radiologic literature.

The purpose of this paper is to discuss the roentgenologic features and pathogenesis of IIE in the newborn with HMD and to postulate that other complications of HMD such as pneumothorax, pneumomediastinum, pneumopericardium, extrathoracic air, and pulmonary pseudocyst formation result directly from the progression of IIE. With the recognition of IIE, the radiologist can alert the clinician to the possible development of other, perhaps more serious, complications of HMD. Hopefully, with a better understanding of IIE and an appreciation of its sequelae, immediate proper management can be undertaken and the high death rate from HMD can be decreased.

MATERIAL AND METHOD

From January 1964 to June 1969, 84 newborns with respiratory distress had a clinical diagnosis of HMD confirmed by a chest roentgenogram at Pennsylvania Hospital. During this period, some of the newborns with clinically suspicious HMD did not have a chest roentgenogram, either because of early death or rapid recovery. The chest roentgenographic criteria utilized were: (1) bilateral, diffuse, fine reticulogranular radiodensities; and (2) an air-bronchogram[5] (Fig. 1). All of the roentgenograms of the chest were obtained with a mobile roentgen-ray machine which is permanently located in the intensive care nursery. These chest roentgenograms can be obtained rapidly under sterile conditions in an environment where skilled personnel with proper equipment are readily available if emergency situations arise at the time of the examination.

Of the 84 neonates with roentgenographically proved HMD, 17 developed an abnormal distribution of extrapulmonary and intrapulmonary air. IIE was identified

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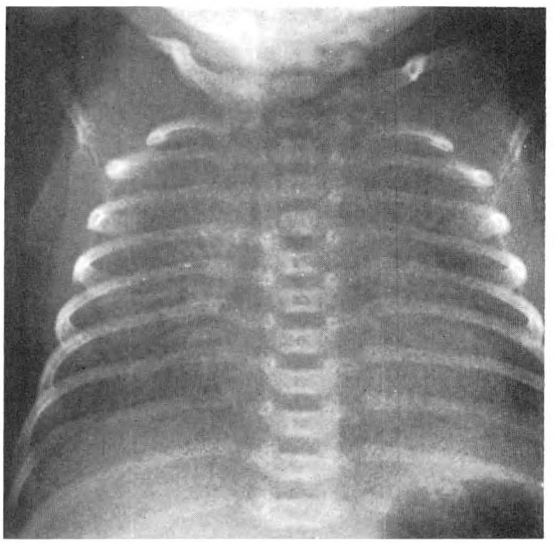


FIG. 1. A 1,700 gram female, age 3 hours. Hyaline membrane disease.

in 14 of these 17 patients; and in the remaining 3 patients its presence in the lungs was suggestive but not unequivocal. Pneumothorax, occasionally under tension, developed spontaneously in 16 of the 17 babies, unilaterally in 11 and bilaterally in 5. All of the other complications such as the 5 instances of pneumomediastinum, the 3 instances of pneumopericardium, the 2 instances of extrathoracic air, and the 5 instances of pseudocyst formation in the lungs occurred not only in association with IIE and pneumothorax but also in various combinations with each other. Most of the complications were discovered between the first 24 and 72 hours of life. Eight of the 17 neonates died.

PATHOGENESIS

The basic pathologic lung changes in HMD include: (1) distal alveolar resorption atelectasis; (2) alveolar duct dilatation; and (3) eosinophilic staining hyaline membranes lining terminal airways.[15] In 1939, Macklin[11] experimentally demonstrated alveolar rupture in the overdistended cat lung with dissection of air along the perivascular sheaths toward the hilum. In HMD, an air-block phenomenon leads to overdistention and rupture presumably of the alveolar ducts and nonatelectatic alveoli.[10] This rupture occurs spontaneously and is not related to artificial respiration. Pathologically, the interstitial air is located along the course of pulmonary vessels (Fig. 2*A*). Why this process develops in some, but not all, neonates with HMD is unknown.

It is likely that the progression of IIE is responsible for many of the complications of HMD. If IIE progresses centrally along the vascular sheaths, the air may dissect into specific anatomic compartments pro-

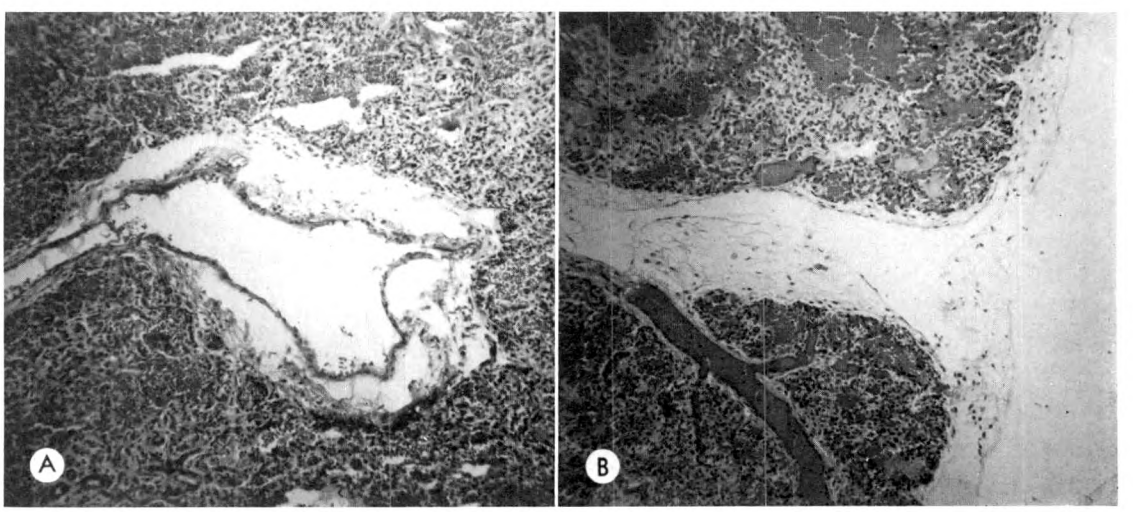


FIG. 2. A 1,400 gram male, died at age 5 days. Photomicrographs (100×). (*A*) Air ensheaths a pulmonary vein. (*B*) Air in interlobular septum and beneath visceral pleura.

ducing, singly or in combination, a pneumothorax, a pneumomediastinum, or a pneumopericardium. Further extension of air superiorly through the mediastinum leads to extrathoracic air collections.[3] If the IIE collects in interlobular septa or peripherally beneath the visceral pleura (Fig. 2*B*), pseudocysts may develop. Whether or not these pseudocysts rupture under tension is unknown. IIE was not a common roentgenographic finding in the 70 neonates with pneumomediastinum and pneumothorax reported by Rudhe and Ozonoff.[16] Only 4 of the 70 neonates had HMD. Nevertheless, in the Pennsylvania Hospital series, the discovery of IIE not only preceding but also occurring concomitantly with other complications lends strong support to the validity of the mechanism postulated by the author.

DISCUSSION

The roentgenologic appearance of IIE should not be confused with the air-bronchogram in HMD. In the latter, long, smooth, branching, linear radiolucencies decrease in caliber from the hilum and frequently disappear at the lung periphery. The air-bronchogram is usually seen best in the medial portion of the lower lung fields. IIE has 2 basic roentgenographic features: (1) linear, and (2) cyst-like radiolucencies. The 2 types often appear together (Fig. 3, *A* and *B*). The linear type measures from 3 to 8 mm. in length, varies in width but seldom exceeds 2 mm. and is coarse and nonbranching. The small cyst-like radiolucencies range in diameter from 1 to 4 mm. and although usually round, may appear oval or slightly lobulated. They may resemble bubbles of air in the lungs. The disorganized, haphazard distribution of IIE in localized areas is unlike the anatomically organized pattern of the air-bronchogram. IIE should be suspected when coarse radiolucencies appear in the lung periphery since the air-bronchogram usually disappears in this area. The radiolucencies of IIE appear more lucent on the chest roentgenograms than air within a bronchus. In a routine examination that consists of 4 exposures (anteroposterior supine, supine horizontal lateral, and both supine obliques), IIE is seen best in the anteroposterior supine projection. The radiolucencies of IIE may replace the diffuse fine reticulogranular radiodensities of HMD and become the dominant feature in the lungs. In the latter

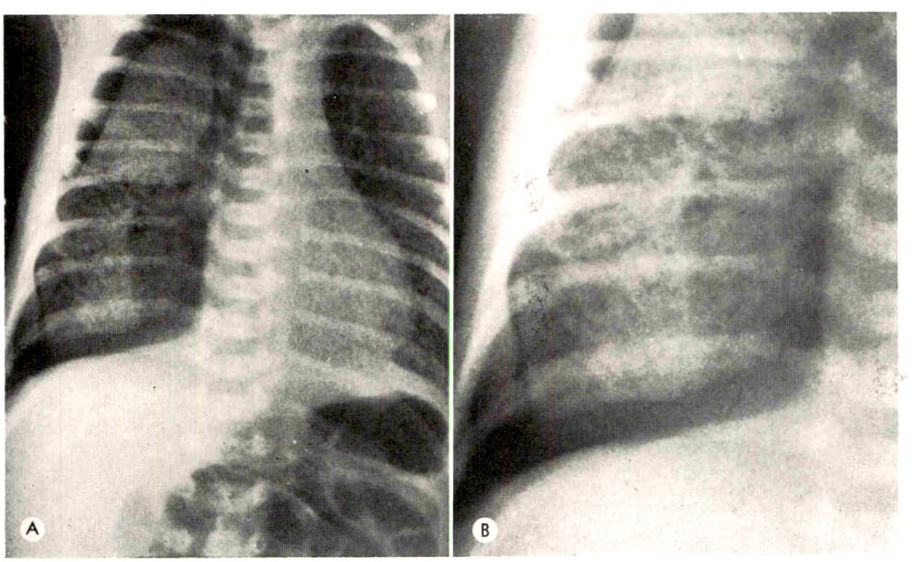


Fig. 3. A 1,900 gram male. (*A*) Age 43 hours. There is diffuse intrapulmonary interstitial emphysema and a pneumothorax on the right. (*B*) Enlargement of the right lower lung to show intrapulmonary interstitial emphysema.

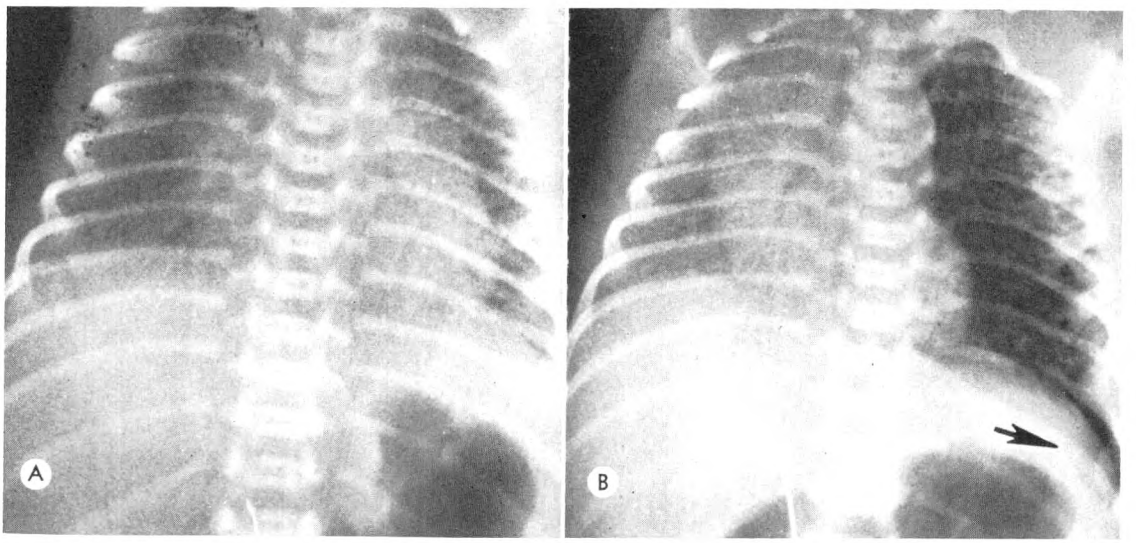


FIG. 4. A 1,410 gram female. (*A*) Age 15 hours. Intrapulmonary interstitial emphysema is visible at the periphery of the left lung. (*B*) Five hours later. There is a pneumothorax (arrow).

instance, misinterpretation of IIE as normally aerated lung surrounded by exudate as in aspiration pneumonia or pulmonary edema can occur.

IIE may be localized to a lobe or may diffusely involve a whole lung or both lungs. When bilateral, a delay of several hours or a few days may occur between its initial appearance in one lung and the subsequent appearance in the contralateral lung. In 1 neonate, the IIE was discovered at 10 hours of age. In the remaining neonates, it was identified after the first 24 hours, but usually within 72 hours, of life. The significance of IIE was not appreciated when most of these cases were examined, and the author suspects that if earlier roentgenograms are taken, it will be discovered within the first 24 to 48 hours of life. In most cases, the discovery of IIE was preceded by a mild decline in the babies' clinical condition.

Thirteen of the newborns with IIE developed a pneumothorax, and in 11 patients

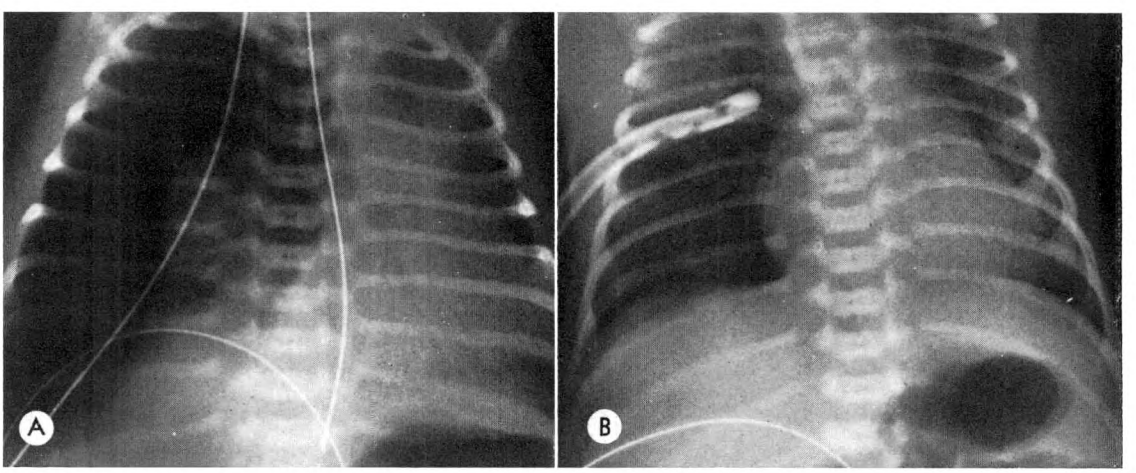


FIG. 5. A 1,910 gram female. (*A*) Age 50 hours. Intrapulmonary interstitial emphysema is not seen in the collapsed right lung under tension. (*B*) One hour later. Intrapulmonary interstitial emphysema is noted in the re-expanded right lung.

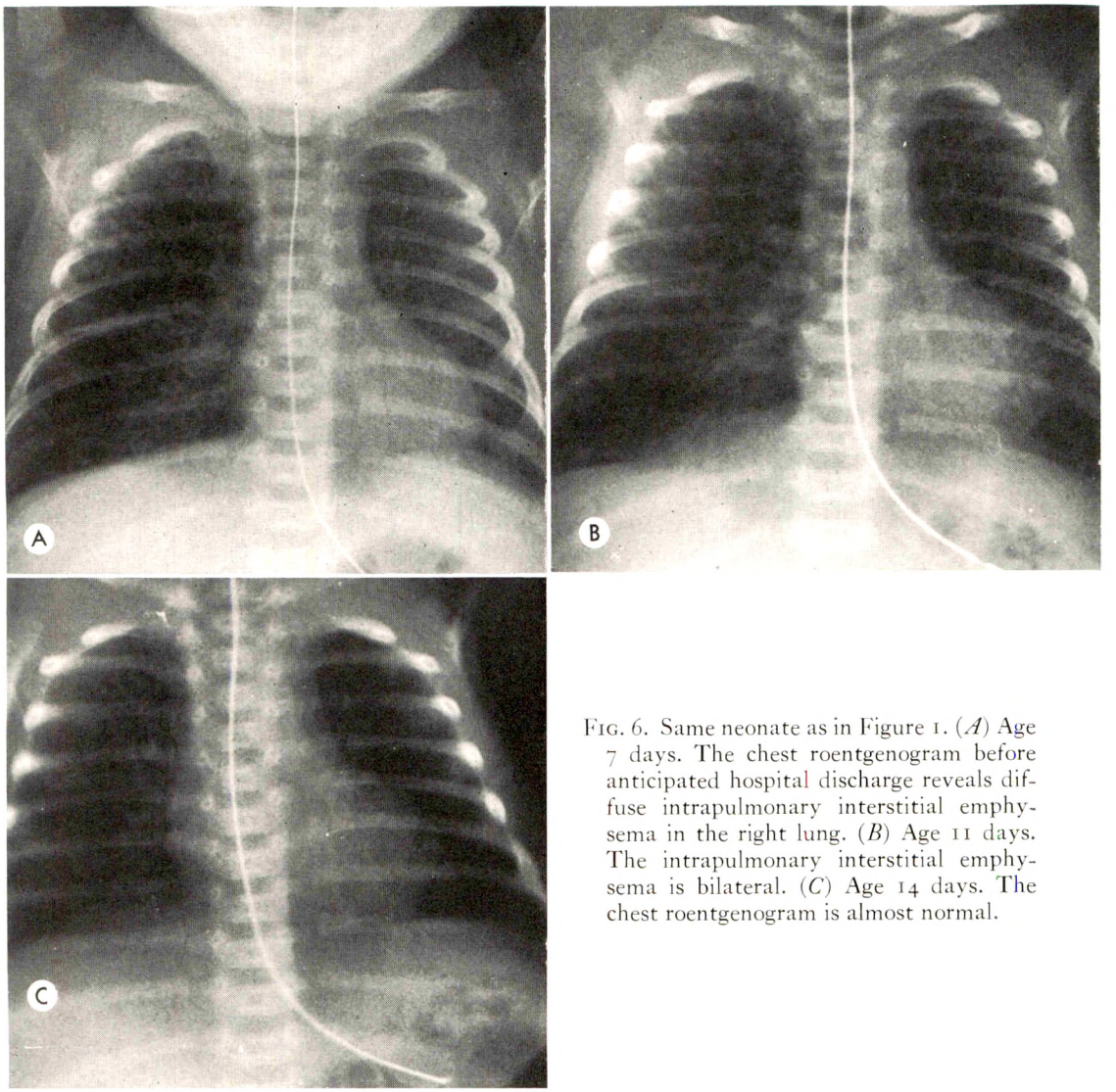


Fig. 6. Same neonate as in Figure 1. (*A*) Age 7 days. The chest roentgenogram before anticipated hospital discharge reveals diffuse intrapulmonary interstitial emphysema in the right lung. (*B*) Age 11 days. The intrapulmonary interstitial emphysema is bilateral. (*C*) Age 14 days. The chest roentgenogram is almost normal.

the 2 abnormalities were found concomitantly. However, in 2 cases, the IIE preceded the pneumothorax by several hours (Fig. 4, *A* and *B*). The observation of IIE without pneumothorax should alert the clinician to observe the neonate closely for the development of a pneumothorax. A dramatic decline in a baby's clinical condition with increasing respiratory rate, decreasing heart rate, need for higher oxygen concentration to control cyanosis, and occasional apneic spells, often heralds the onset of a pneumothorax. Since the physical diagnosis of a pneumothorax in newborns is occasionally impossible,[17] the lack of positive physical findings should not dissuade the clinician from ordering an emergency chest roentgenogram to verify a clinical suspicion of a pneumothorax. It is possible that the absence of observable IIE in some patients with pneumothorax is related to collapse of the lung. A chest roentgenogram prior to the pneumothorax may have revealed IIE since it was identified in some patients following re-expansion of the collapsed lung (Fig. 5, *A* and *B*).

In an unusual case (Fig. 6, *A–C*) at 7 days of age, while clinically improving, one

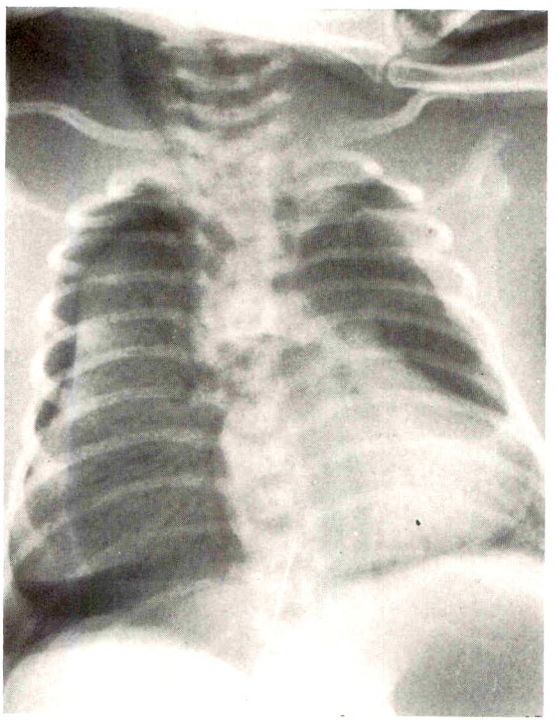


FIG. 7. A 1,460 gram male, age 24 hours. There is a pneumomediastinum, extrathoracic air in the neck, and a pneumothorax on the right. Note the haphazard distribution of intrapulmonary interstitial emphysema in the right lung. The thymus, outlined by air, resembles a "spinnaker sail."[13]

baby developed diffuse unilateral IIE, predominantly of the cystic type. Four days later, a similar appearance developed contralaterally without a change in the baby's clinical condition. At 2 weeks of age, both lungs were almost roentgenographically normal. A pneumothorax did not develop. Although the roentgen appearance in this case is somewhat similar to the condition described by Wilson and Mikity[18] and Baghdassarian *et al.*,[2] the clinical course of this patient was not similar to those patients described with pulmonary dysmaturity. The roentgen appearance is more likely secondary to diffuse IIE which rapidly cleared rather than to pulmonary septal thickening and alveolar distention.

In all of the cases of pneumomediastinum (Fig. 7), pneumopericardium (Fig. 8), and extrathoracic air (Fig. 7), IIE was noted in the lungs. Previous instrumentation did not precede any of these complications.

The pulmonary pseudocysts appeared to be localized peripherally beneath the visceral pleura or along fissure lines. These pseudocysts occurred singly or in groups usually localized to one area within the lung. Their diameters varied from 0.5 to 3 cm. (Fig. 9, *A–C*). A few appeared to have a thin peripheral wall which may represent a combination of visceral pleura and normal but collapsed lung. None required surgical removal and they regressed spontaneously within 10 to 14 days. They were not associated with previous pneumonia or pleural effusion. All these pseudocysts occurred before 72 hours of age. The pneumatoceles of staphylococcal pneumonias do not appear so early in the neonatal period.[8]

SUMMARY

1. Intrapulmonary interstitial emphysema is a common and important complication of hyaline membrane disease.

2. It is suggested that the progression of intrapulmonary interstitial emphysema

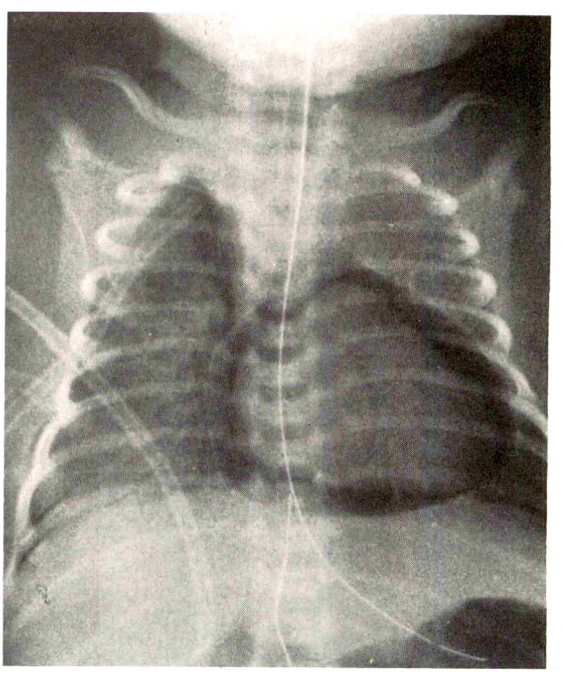


FIG. 8. A 1,680 gram female, age 55 hours. There is a pneumopericardium, pneumothorax on the right, and intrapulmonary interstitial emphysema in the right lung.

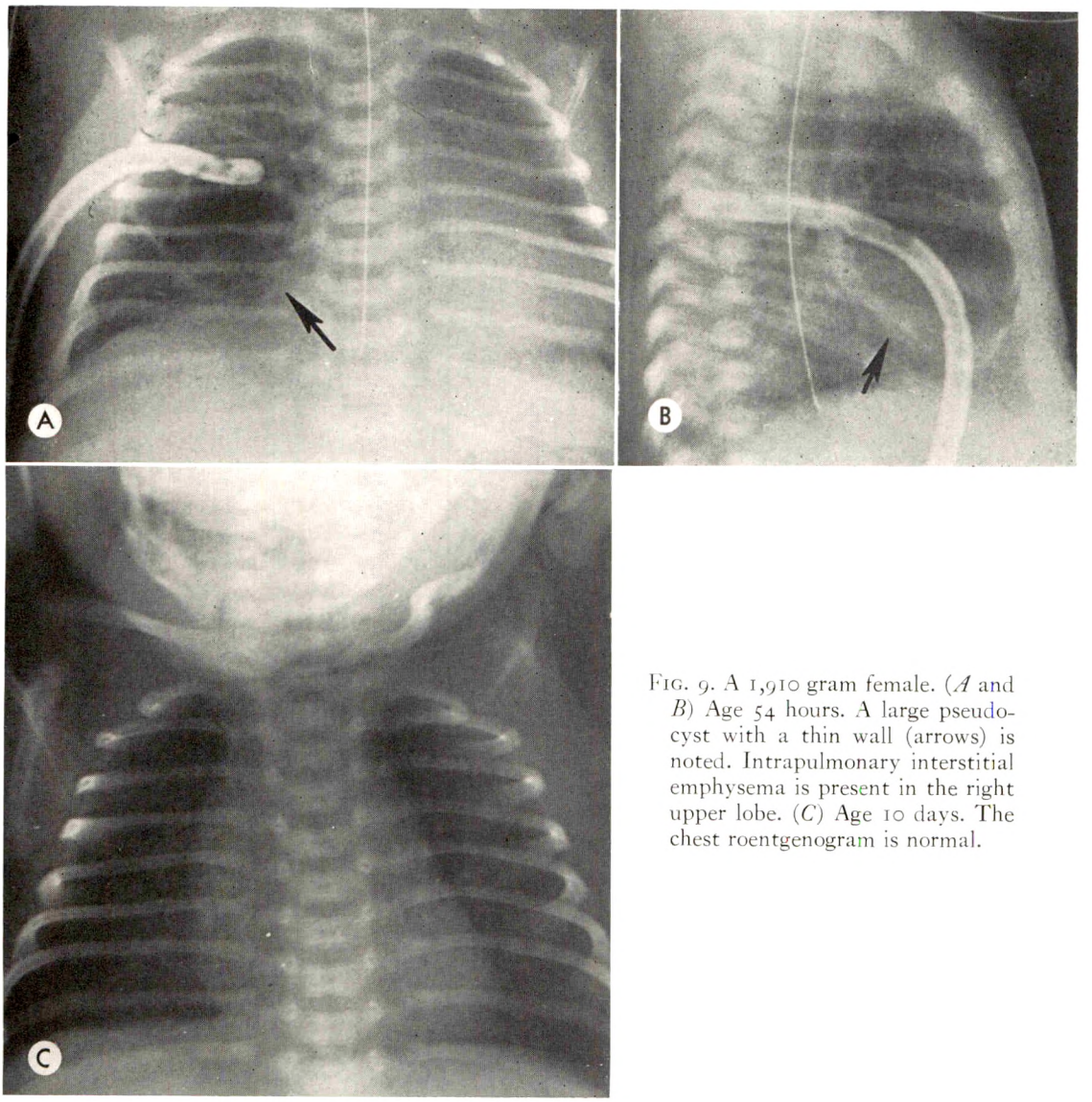


Fig. 9. A 1,910 gram female. (*A* and *B*) Age 54 hours. A large pseudocyst with a thin wall (arrows) is noted. Intrapulmonary interstitial emphysema is present in the right upper lobe. (*C*) Age 10 days. The chest roentgenogram is normal.

may lead to the development of other early complications of hyaline membrane disease, such as pneumomediastinum, pneumopericardium, extrathoracic air, and pseudocyst formation in the lungs.

3. The observation of intrapulmonary interstitial emphysema should alert the radiologist and the clinician of the possible subsequent development of other complications, particularly a pneumothorax.

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ROENTGENOGRAPHIC FINDINGS IN LIPOID PROTEINOSIS: A CASE REPORT*

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LIPOID proteinosis was originally described as a nodular dermatosis which also involved the mucous membranes of the oral cavity and larynx.[11,12] Histopathologically, the nodular lesions consist of a glycoprotein in association with free or loosely bound lipid.[6,7,9,13]

Hoarseness, secondary to nodular infiltration and thickening of the vocal cords, is a common presenting symptom. More recently, lipoid proteinosis is being recognized as a generalized disorder which can involve many organs. Involvement of the central nervous system, gastrointestinal tract, urinary tract, lung, lymph nodes, and striated muscle has been reported.[1–4,8,10] There has never been, to our knowledge, a description of the roentgenographic changes in the respiratory tract.

The purpose of this paper is to present the roentgenographic findings in the larynx and lower respiratory tract in one patient, as well as to review the roentgenographic findings in general.

REPORT OF A CASE

G.S., No. 26–61–67. This 13 year old Caucasian male has had multiple admissions at Children's Memorial Hospital, University of Oklahoma Medical Center, as well as other medical centers, since his first year of life. He was adopted in early infancy but is known to be the product of consanguineous parents. His medical history has been characterized by recurrent episodes of upper and lower respiratory tract infections including sinusitis and pneumonia, chronic conjunctivitis, and repeated and progressive bouts of hoarseness with stridor. The latter symptoms have necessitated repeated surgical removal of nodules from his vocal cords. The boy's mother stated that he did not have any skin lesions until he was 6 years of age, but that his voice first became hoarse when he was approximately 4 years old. He is mentally retarded, to a moderate degree, and has attended special education classes since entering school.

The diagnosis of lipoid proteinosis was first made in 1966 on the basis of the typical skin lesions, vocal cord lesions, and compatible biopsies from these areas. The skin lesions have become larger and more obvious during the past 2 years, and fissuring and hyperkeratosis of the skin around his mouth and lips have become especially prominent (Fig. 1). Repeated skin biopsies of typical nodules have shown hyperkeratosis, focal areas of necrosis, and nodular hyalinization with PAS positive material (Fig. 2).

Physical examination during his last admission in September, 1969, revealed a cooperative, asthenic boy who was mentally retarded. His skin was pale and contained many hyperkeratotic and translucent nodules ranging in size from



FIG. 1. Photograph taken in September, 1969 demonstrates typical skin lesions of lipoid proteinosis. The nodular infiltrates and pock-like scars are noteworthy.

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FIG. 2. Punch skin biopsy on August 25, 1967 shows hyperkeratosis (arrow) and PAS positive hyalinization of the nodule in the dermis. (PAS×approx. 50, E = epidermis, D = dermis.)

1 to 5 mm. in diameter. There was a tendency for confluence in some areas around the upper limbs and face. Similar nodules were present in lesser numbers on the hands, fingers, and extensor surfaces of the extremities. His lips were dry, keratotic, and cracked. An arched palate was present, and his teeth were carious, badly worn, and some appeared peg-like.

His voice was hoarse, and indirect laryngoscopy revealed irregular thickening of both true cords, and multiple nodular and plaque-like lesions of the false cords, oropharynx, and hypopharynx. A plaque-like excrescence on the right vocal cord was present. Occasional rales and rhonchi were scattered over both lung fields.

Chest roentgenographic findings on July 17, 1959, when he was 2 years old, revealed a right upper lobe pneumonia, presumably caused by aspiration. There were a few scattered reticular and nodular densities in the left upper and right lower lobes (Fig. 3). A bronchogram did not disclose any evidence of bronchiectasis (Fig. 4). In November, 1966, there were linear densities in both upper lobes with minimal overexpansion of the left upper lobe. Reticular and nodular densities were scattered in the mid and lower lung fields. Pleural thickening was present along the right lateral chest wall (Fig. 5). Roentgenograms of the paranasal sinuses revealed opacification of the right maxillary sinus (Fig. 6). On September 30, 1969, the pleural thickening had diminished considerably, and the changes in the upper lobes were static and resembled pulmonary fibrosis. There was no hilar or mediastinal lymphadenopathy, and the cardiac silhouette and pulmonary vascular patterns were normal. No parenchymal or mediastinal calcifications were present. Reticular and nodular densities in the mid and lower lung fields were unchanged.

Laminagraphy of the larynx on October 22, 1969, revealed thickening of the true and false cords, and partial obliteration of the vallecula, particularly on the right (Fig. 7; and 8). In phonation, there was narrowing of the subglottic area. With quiet respiration, the cords

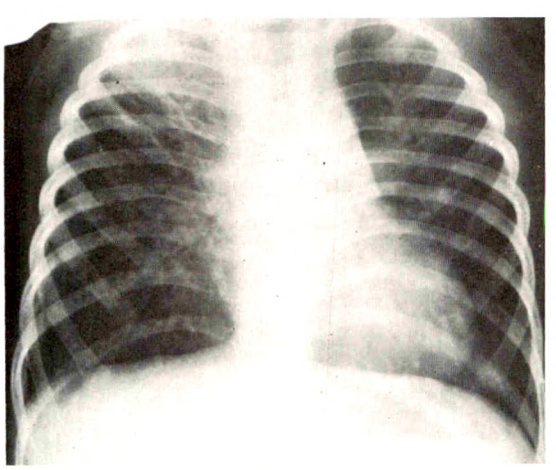


FIG. 3. Anteroposterior chest roentgenogram on July 17, 1959 demonstrates a right upper lobe pneumonia. There are a few reticular and nodular densities in the left upper and right lower lobes.

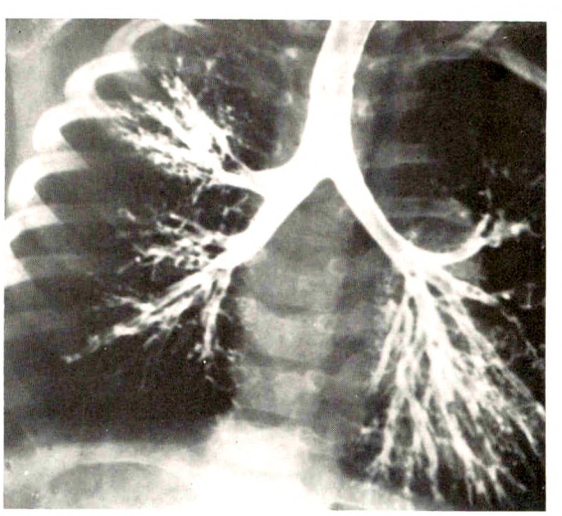


FIG. 4. Bronchogram on August 10, 1959. There is no evidence of bronchiectasis. There is residual loss of volume of the right upper lobe.

did not flatten normally, and the laryngeal airway remained narrowed. A skull series was normal.

DISCUSSION

Lipoid proteinosis is a rare hereditary familial metabolic disorder which follows a mendelian autosomal recessive pattern of transmission. There are many cases of consanguinity reported in the parents of these patients, and the relatives are usually of central European ancestry.[2,8] Normal longevity is possible, and instances have been reported where patients were over 65 years of age.[7] To date, there have been no distinctive laboratory findings and diagnosis is dependent primarily upon the physical findings and biopsy of the affected tissues.

The roentgenographic findings, although not pathognomonic, are certainly suspicious in certain combinations. The first roentgenographic abnormalities to be noted were bean-shaped intracranial calcifications located in the suprasellar area lateral to the mid line.[8] They were bilateral, and were seen in all lipoid proteinosis patients who presented with epilepsy.[3] Patients complaining of déja vu sensations only did not necessarily show intracranial calcifications.[5] The calcifications have subsequently been

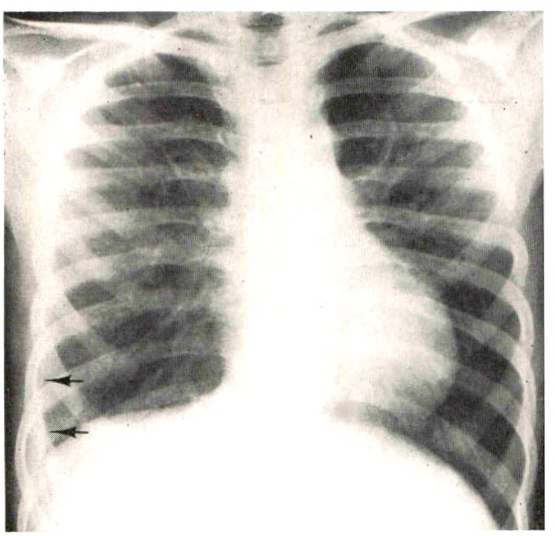


Fig. 5. Posteroanterior chest roentgenogram on November 16, 1966. There are a few linear densities in the upper lobes with minimal overexpansion of the left upper lobe. Reticular and nodular densities are present in the mid and lower lung fields. There is pleural thickening along the right lateral chest wall (arrows).

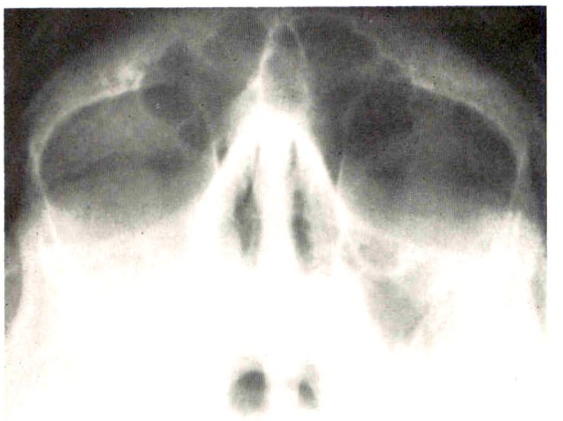


Fig. 6. Waters' projection of the paranasal sinuses on April 2, 1968 demonstrates right maxillary sinusitis.

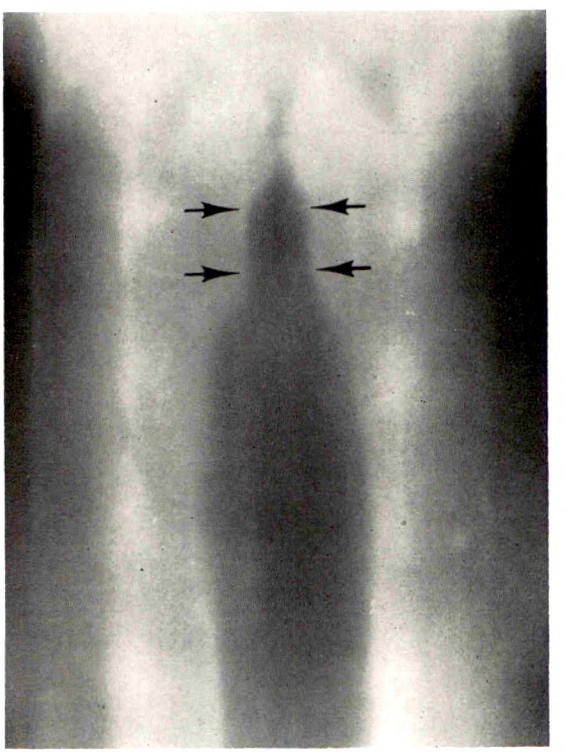


Fig. 7. Anteroposterior laminagram of the larynx with phonation on October 22, 1969. There is nodular thickening of the true and false cords and partial obliteration of the vallecula on the right. Subglottic narrowing is present (arrows).

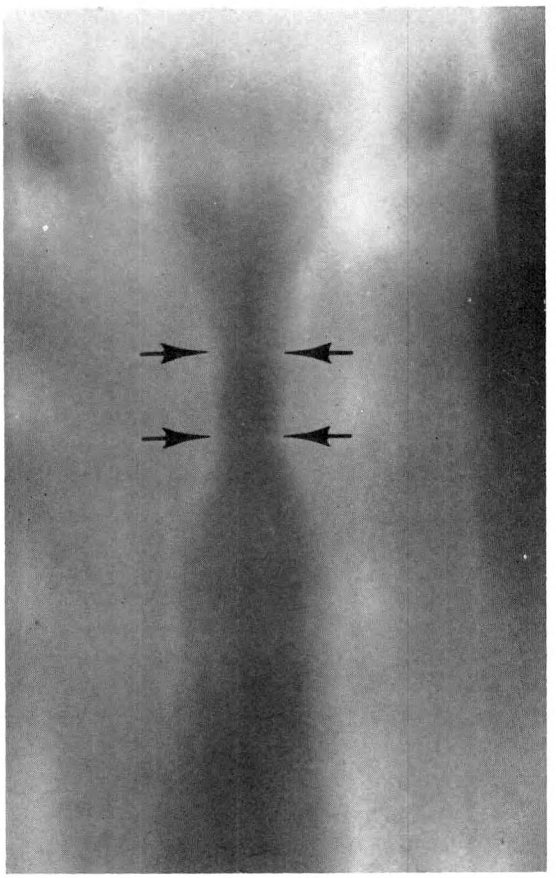


FIG. 8. Anteroposterior laminagram of the larynx with quiet respiration on October 22, 1969. There is narrowing of the laryngeal airway.

identified microscopically, and have been shown to occur around the capillaries of the hippocampal gyri of the temporal lobes.[1]

Roentgenographic evidence of vocal cord thickening, either diffuse or nodular, is easily demonstrated by conventional roentgenograms of the larynx, or with special studies such as laminagraphy or laryngography. The nodularity can extend into the vestibule and into the base of the pyriform sinuses. The cords are often stiff and fail to close properly. At laryngoscopy, yellowish-white plaques may be seen on the epiglottis, aryepiglottic folds, and interarytenoid region. Because of the nodular infiltrates, hoarseness is often the presenting symptom, being present from birth, or becoming evident by puberty. In fact, some young infants are unable to cry at birth because of early laryngeal involvement. This same nodular appearance has been described in the esophagus and stomach.[2]

Histopathologically, capillaries in the lungs of some afflicted individuals have shown deposition of lipid staining material within their walls.[1] Roentgenographically, the changes resemble interstitial fibrosis, and are nonspecific. In our patient, the first abnormality in the lungs was a right upper lobe pneumonia associated with a small pleural effusion. After the pneumonia cleared, residual changes resembling pulmonary fibrosis and pleural thickening were seen. It is possible that many of the pulmonary changes are secondary to repeated bouts of aspiration pneumonia due to improper approximation of the thick and stiff true and false vocal cords during swallowing. Supporting this theory is the mother's observation that, in our patient, the frequency of pneumonia markedly decreased after vocal cord stripping, but increased again as hoarseness (and new lesions) returned. Interference with the normal ciliary activity of the lower respiratory tract may also play a role in the problem of repeated bouts of pneumonia.

SUMMARY

A case of lipoid proteinosis, demonstrating vocal cord and pulmonary involvement, is reported.

The roentgenographic findings in this condition are reviewed, and it is suggested that, in certain combinations, they can be strongly suggestive of the diagnosis of this disorder.

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LEFT ATRIAL FUNCTION STUDIED BY CINEANGIOCARDIOGRAPHIC VOLUME DETERMINATIONS*

I. STUDIES WITH CASTS OF NORMAL HUMAN ATRIA

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IN RECENT years several methods for volume determinations of the left ventricle and the left atrium from angiocardiograms have been described. A great number of authors have performed studies with casts from the left ventricle in order to assess the accuracy of the various methods.[2–6,8,9] To our knowledge similar studies for the left atrium have been carried out only by Sauter and co-workers.[11] We therefore thought it worthwhile to repeat such studies in order to test different mathematical methods and to assess the accuracy under the conditions of our own set-up.

MATERIAL AND METHOD

The studies were carried out in co-operation with the Anatomical Institute of the University of Hamburg. In 10 nonfixed human heart-lung specimens from normal intact individuals ranging in age from 2 months to 70 years, both hila, the pulmonary artery, aorta, and both venae cavae were dissected and plastic tubes placed into these vessels. The specimens were flushed clear with water and freely suspended on a tripod. The cavities were then filled with plastic casting material,* using the superior vena cava for the right heart and one of the pulmonary veins or the auricular appendage for the left heart. In most cases the pericardium had been intact, or, if not, it was carefully sutured. In this way an overdistention was avoided. After hardening of the plastic material, all tissue was removed by using 30–50 per cent potassium hydroxide. Further details are published elsewhere.[7,12] The left atria were then separated from the left ventricles; pulmonary veins and the auricular appendage were cut off. "True" volume was assessed by water displacement, taking the average of 3 determinations. Cineroentgenograms were obtained from each model in 2 planes, using both the 5 inch and 9 inch image intensifier. The atria were placed in a position similar to the normal position of the left atrium within the thoracic cage. A slide gauge with a constant opening of the branches (5 cm.) was placed close to the casts approximately at the respective center.

The roentgenographic silhouettes of the atria were then traced on paper using a magnification that showed the branches of the slide gauge at the original distance. Thus a correction of the roentgenographic magnification was unnecessary.

The following measurements were carried out on the tracings (Fig. 1, *A* and *B*):

1. Length of the transverse diameter in the frontal plane (D_1)
2. Length of the horizontal and vertical diameters in the sagittal plane (D_2 and D_3)
3. The areas in both planes were measured using a planimeter.†

Three mathematical models were tested:

1. Following the suggestion of Arvidsson,[1] the left atrium was considered to be a spheroid and the formula

$$V_{LA} = \frac{\pi}{6} \cdot D_1 \cdot D_2 \cdot D_3$$ was applied,

* Technovit, Kulzer & Co.

† Aristo, No. 1100L.

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† Deceased, June 25, 1970.

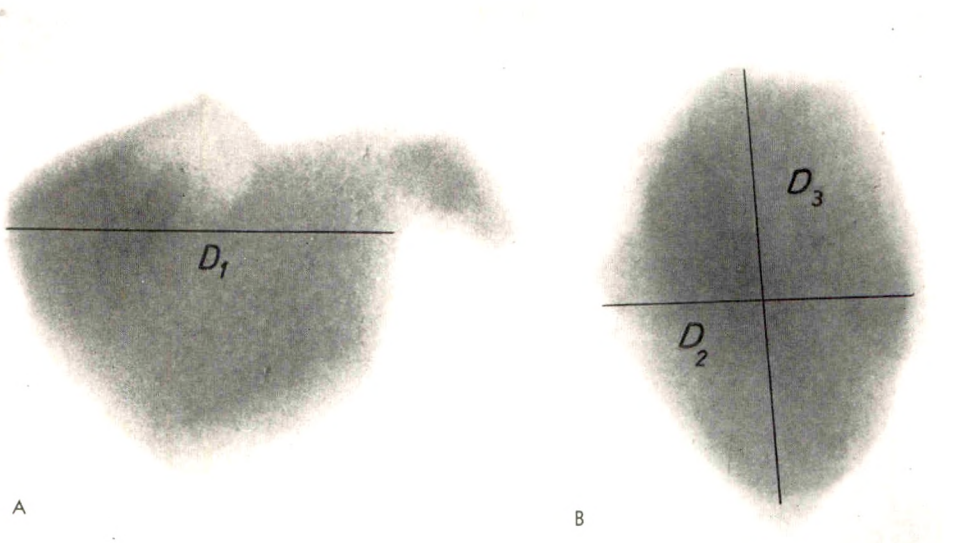


Fig. 1. Cineroentgenograms from a cast of a human atrium. (*A*) Sagittal projection. (*B*) Lateral projection. The 3 measured axes are shown.

where V_{LA} = volume of the left atrium; D_1 = transverse diameter in the frontal plane; D_2 = horizontal diameter in the sagittal plane; and D_3 = vertical diameter in the sagittal plane.

2. Studies of the left ventricle performed by Dodge and associates[4,5] had shown that deriving the short axes from the planimetered area of the ventricular silhouette instead of directly measuring them yielded better results. Therefore this procedure ("area-length-

Table I

"True" volumes (determined by water displacement), calculated volumes, and corrected volumes of ten casts of normal human atria. Per cent error before and after correction. Three different methods were tested

No.	V_t	Direct Measurement of Axes				Area—Length—Method				Area—Product—Method			
		V_{calc}	Per Cent Error	V_{corr}	Per Cent Error	V_{calc}	Per Cent Error	V_{corr}	Per Cent Error	V_{calc}	Per Cent Error	V_{corr}	Per Cent Error
9 Inch Intensifier													
1	65.8	77.80	18.23	68.99	4.84	72.24	9.7	63.84	−2.97	58.48	−11.1	64.59	−1.8
2	66.0	67.69	2.56	59.63	−9.65	64.16	−2.7	56.37	−14.59	50.76	−23.0	56.70	−14.0
3	60.6	56.43	−6.88	40.21	−33.64	67.42	11.2	59.38	−2.01	47.70	−21.2	53.57	−11.6
4	139.8	154.76	10.7	140.19	0.27	133.91	−4.1	120.93	−13.49	135.77	−2.8	143.53	2.6
5	105.2	103.84	−1.29	93.08	−11.52	109.94	4.5	98.72	−6.15	104.85	−0.3	111.95	6.4
6	80.0	93.27	16.58	83.30	4.12	103.70	29.6	92.95	16.18	87.75	9.6	94.48	18.1
7	59.3	78.73	32.76	69.85	17.79	78.21	31.8	69.37	16.98	64.85	9.3	71.09	19.8
8	85.0	116.37	36.9	104.67	23.14	114.04	34.1	102.52	20.61	76.81	−9.6	83.31	−1.9
9	100.0	106.68	6.68	95.71	−4.29	109.77	9.77	98.57	−1.43	95.59	−4.4	102.49	2.4
10	3.5	3.87	10.57	0.58	−38.42	4.31	23.1	0.99	−71.71	1.22	−65.1	6.10	74.2
5 Inch Intensifier													
1	65.8	73.82	12.18	65.30	−0.75	60.43	−8.1	52.92	−19.57	44.68	−32.0	58.93	−10.4
2	66.0	54.55	−17.34	47.47	−28.07	52.83	−19.9	45.88	−30.48	34.86	−47.1	48.58	−26.3
3	60.6	55.35	−8.66	48.21	−20.44	71.50	17.9	63.16	4.22	47.19	−22.1	61.57	1.6
4	139.8	152.41	9.02	138.02	−1.27	139.95	0.1	126.49	−9.52	138.80	−0.7	158.10	13.0
5	105.2	115.19	9.49	103.58	−1.53	127.73	16.6	110.56	5.09	108.87	3.4	126.56	20.3
6	80.0	97.23	21.5	86.96	8.7	109.58	36.9	98.39	22.98	87.24	9.0	103.77	29.7
7	59.3	82.92	39.83	73.72	24.31	78.49	32.3	69.62	17.40	61.80	4.2	76.97	29.7
8	85.0	124.55	46.52	112.24	32.04	124.81	46.8	112.48	32.32	78.74	−7.3	94.82	11.5
9	100.0	108.0	8.0	96.93	−3.07	105.85	5.8	94.94	−5.06	83.8	−16.2	100.15	0.1
10	3.5	4.64	32.57	1.30	−62.0	4.73	35.1	1.38	−60.57	1.23	−64.8	13.14	275.4

V_t = "true" volume; V_{calc} = calculated volume; V_{corr} = corrected volume.

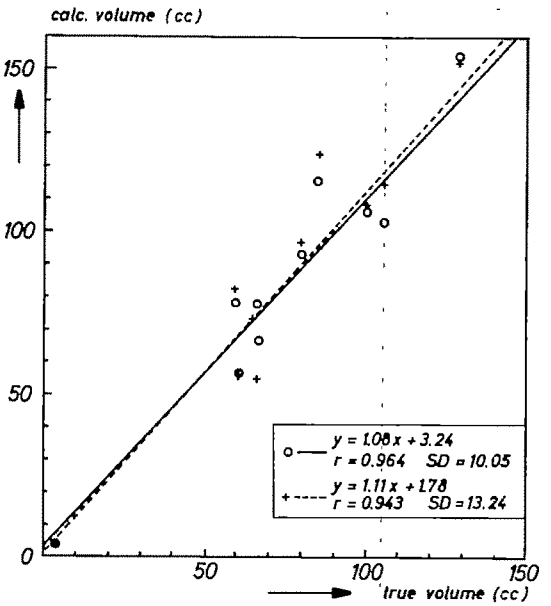


FIG. 2. Comparison of calculated volume and true volume. Direct measurement of axes. Clear circles and solid line = 9 inch image intensifier; crosses and dotted line = 5 inch image intensifier.

method") was also adapted for the left atrium.[11] We used the following formula:

$$V_{LA} = \frac{8}{3\pi} \cdot \frac{Aap \cdot Alat}{D_2},$$

where V_{LA} = volume of the left atrium; Aap = area of the left atrium in the frontal plane; $Alat$ = area of the left atrium in the sagittal plane; and D_2 = vertical diameter of the sagittal plane.

3. Under the assumption that the volume of a relatively regular body bears a constant relation to its areas projected at right angles,[3,10] we determined the relation of the "true" volume and the product of the 2 areas. Thus a factor was obtained and the volume was calculated with the formula

$$V_{LA} = A_{ap} \cdot A_{lat} \cdot K,$$

where V_{LA} = volume of the left atrium; A_{ap} = area of the left atrium in the frontal plane; A_{lat} = area of the left atrium in the sagittal plane; and K = constant factor (0.12 for the 9 inch and 0.11 for the 5 inch image intensifier).

RESULTS

"True" volumes, calculated volumes, corrected volumes, and per cent error before and after correction are shown in Table 1. Regression equations, correlation coefficients, and ±1 standard deviation are shown in Figures 2–4. Correlation coefficients varied between 0.952 and 0.979, and standard deviation between 7.0 and 10.7 cc. for the 9 inch image intensifier. For the 5 inch image intensifier, the respective values varied between 0.920 and 0.949 and 11.6 and 15.3 cc. A statistical correction of the calculated volumes was attempted using the equation

$$V_{corr} = \frac{V_{calc} - h}{b},$$

where V_{corr} = corrected volume; V_{calc} = calculated volume; h = intercept at the y-axis; and b = regression coefficient.

Individual errors of calculated volumes and corrected volumes are plotted as per

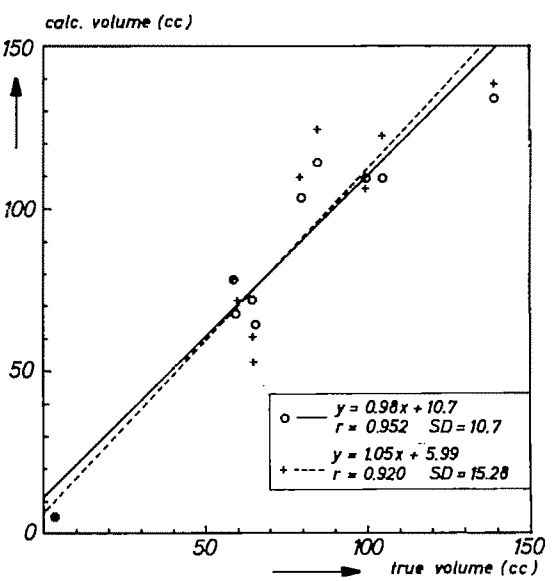


FIG. 3. Comparison of calculated volume and true volume. Area–length–method. Symbols are as in Figure 2.

Table II

MEAN PER CENT ERROR OF THE 3 METHODS TESTED BEFORE AND AFTER STATISTICAL CORRECTION. ONLY THE RESULTS OF THE 9 INCH IMAGE INTENSIFIER ARE GIVEN

Method	DMA		ALM		APM	
Correction	before	after	before	after	before	after
$\bar{x}$	12.7	−9.2	14.7	−7.7	−11.9	9.8
±1 SD	14.0	30.5	14.1	26.6	26.7	25.1

DMA = direct measurement of axes; ALM = area-length-method; APM = area-product-method; SD = standard deviation.

cent of the true volume in Table II. Mean per cent error before and after statistical correction is shown in Figure 5.

DISCUSSION

All 3 methods tested gave good results which are in close agreement with those of Sauter and associates.[11] The regression equation obtained by these investigators was $y = 0.895 + 9.51$, their standard error was ±6.8 cc. The best method proved to be the "area-product-method"; the differences, however, were not significant. Correlation was slightly better with the 9 inch image intensifier, as could be expected.

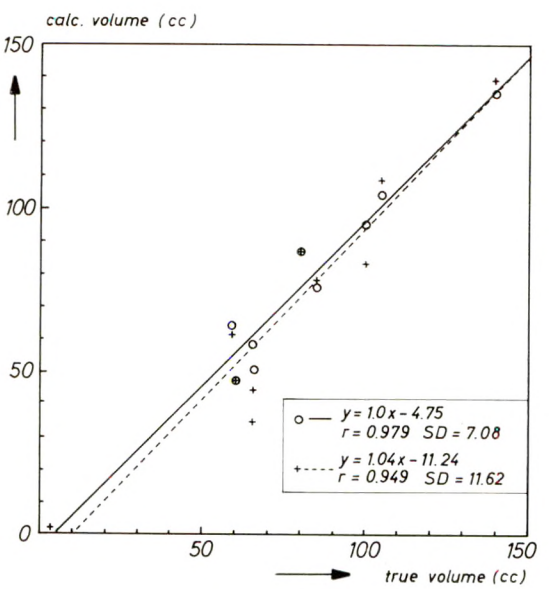


Fig. 4. Comparison of calculated volume and true volume. Area—product—method. Symbols are as in Figure 2.

Table II shows that the first 2 methods (direct measurement of axes and "area-length-method") on the average yielded too large volumes, whereas the area-product-method yielded too small a volume. Statistical correction resulted in a smaller mean per cent error; however, the standard deviation in all instances exceeded that before correction. This is in contrast to the results which other investigators and we ourselves obtained in studies with casts of the left ventricle. This is probably due to the large error in the smallest heart of the series—that of a 2 month old infant.

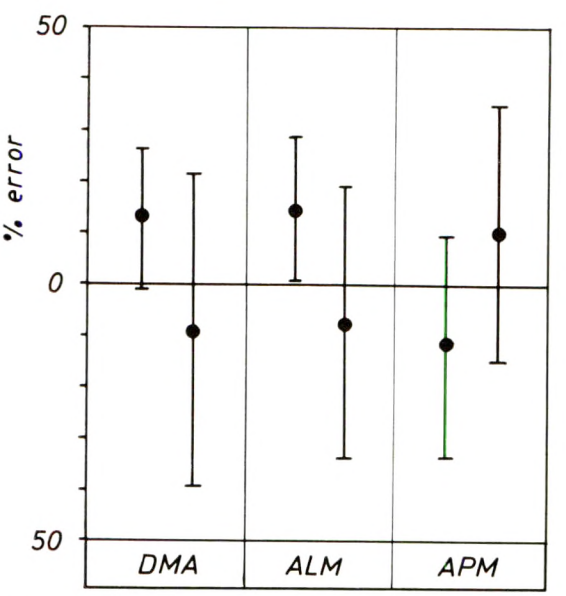


Fig. 5. Mean per cent error before and after statistical correction. Brackets indicate ±1 standard deviation. For details see text. DMA = direct measurement of axes; ALM = area—length—method; APM = area—product—method.

SUMMARY

Cineangiocardiographic volume determinations were carried out in 10 casts from normal human atria using 3 different mathematical models. Correlation with "true" volumes measured by water displacement proved to be good in all 3 methods tested; the differences were not significant. Statistical correction did not improve the results.

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LEFT ATRIAL FUNCTION STUDIED BY CINEANGIOCARDIOGRAPHIC VOLUME DETERMINATIONS*

II. CLINICAL APPLICATION

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SINCE preliminary studies with casts of normal human atria have shown good correlation between "true" volume and cineangiocardiographically determined volumes, as previously described by us, we were interested in investigating left atrial function by continuous cineangiocardiographic volume determinations. Such studies have been performed by several authors,[3,7,8,10,11,14–17] but mainly in adults, and using serial angiography with rates of up to 12 frames per second.

MATERIAL AND METHOD

Left atrial volume determinations were made in 13 cineangiocardiograms obtained from 12 patients, 3 boys and 9 girls, ranging in age from 1 month to 7 years. Seven of these had pulmonary valvular stenosis. It was thought that they did not present any significant alteration of their left heart function, so that they could serve as the control group. Four children had an obstructive lesion of the left ventricle (aortic valvular stenosis, coarctation of the aorta). In one of these children this was combined with mitral stenosis and mitral regurgitation. Another child had severe mitral stenosis with severe myocardiopathy.

All cineangiocardiograms were obtained during cardiac catheterization, the children being in an unanesthetized, resting, supine state. Premedication consisted of 1 mg. demerol and 2 mg. phenergan per kg. body weight; infants were sedated with chloral hydrate (0.6 gm.).

The roentgen equipment consisted of a biplane unit with closed circuit television, 9 inch and 5 inch image intensifiers,* and two 35 mm. "Arriflex" cinecameras running at 50–80 frames per second with pulsed radiation. Both planes were electrically synchronized with alternating pulsation. Electrocardiogram tracings were recorded during the angiocardiography with a paper speed of 50 mm./sec. Contrast medium (urografin, conray) was injected with an injector† within 0.8 to 1.5 seconds. The average amount was 1 ml. per kg. body weight.

DATA ACQUISITION

Each film was marked with a lead number 5 cm. in length. This lead number was held exactly against the grid in front of the image intensifier. In projecting the films for tracings, that magnification was chosen which showed the lead number in its original length. Tracings of the left atria and ventricles were obtained frame by frame in both planes over a period which would cover at least one complete cardiac cycle, maximum of four. On the average this equaled 60 frames in each plane, or $2\frac{1}{2}$ cycles.

The frame which showed the very first trace of contrast medium at the tip of the catheter was marked No. 1 and the subsequent frames were counted accordingly.

As previous investigations have shown (see Part I) that the 3 tested methods for volume calculations of the left atrium did not vary significantly, we chose the method with direct measurement of the axes, as it was somewhat less tedious. Three axes were drawn according to Figure 1, *A* and *B* and their length was measured. Volumes

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* From the Radiological Department (Head: Prof. M. A. Lassrich) and the Cardiological Department (Head: Prof. E. W. Keck) of the Pediatric University Hospital of Hamburg (Director: Prof. K. H. Schäfer), Hamburg, Germany.

† Deceased, June 25, 1970.

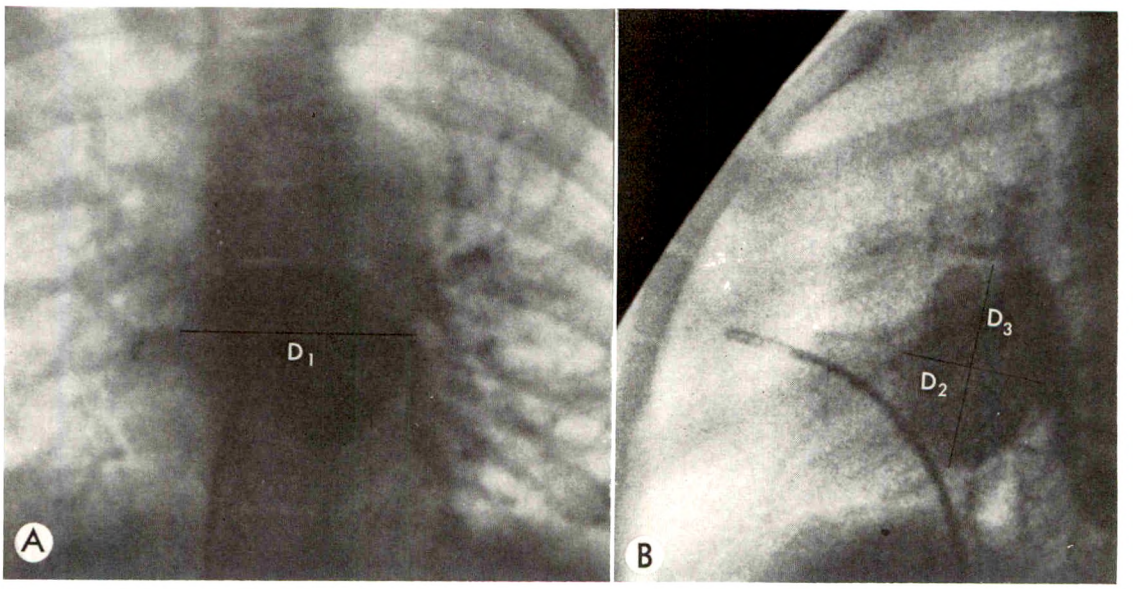


FIG. 1. Cineangiocardiograms of the left atrium. (*A*) Sagittal projection. (*B*) Lateral projection. The 3 measured axes are shown.

were then calculated using the following formula

$$V_{LA} = \frac{\pi}{6} \cdot D_1 \cdot D_2 \cdot D_3,$$

where V_{LA} = volume of the left atrium; D_1 = frontal diameter; and D_2 and D_3 = horizontal and vertical diameters in the lateral plane.

Roentgenographic distortion was corrected using the formula

$$y = \frac{FO}{FG},$$

where y = correcting factor; FO = focus-object distance; and FG = focus-grid distance.

Left ventricular volumes were calculated using the area-length-method of Dodge and associates.[5,6]

After calculation of the volumes, volume changes were plotted as curves. Volume curves and electrocardiogram tracings were mounted together photographically (Fig. 2). (An automatic film and an electrocardiogram marking device were not available at the time of our investigations.)

In order to obtain an analysis of the mode of atrial filling and emptying, systole and diastole in the 7 cases with pulmonary stenosis were arbitrarily divided into 3 equal portions, and the volume changes during each of these periods were determined.

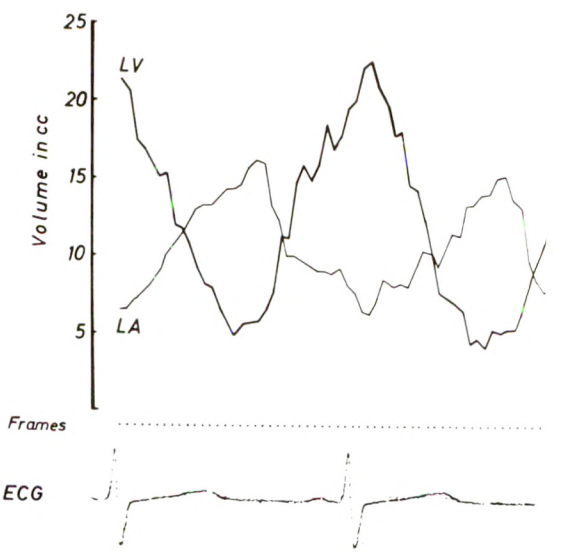


FIG. 2. Left ventricular (heavy line, LV) and left atrial (thin line, LA) volume curves from a 1.8 year old boy with pulmonary valvular stenosis. The values were obtained by cineangiocardiographic volume determination. Film frequency, 50 frames/sec. Three distinct phases of atrial emptying can be observed.

Table I

VOLUME DATA FROM 7 PATIENTS (8 FILMS) WITH PULMONARY VALVULAR STENOSIS AND 5 PATIENTS WITH VARIOUS CARDIAC MALFORMATIONS

Diagnosis	Patient	Sex	Age (yr.)	BSA (m^2)	LA_{max} (cc./m^2)	LA_{min} (cc./m^2)	LAVC
PS	T.H.	M	1; 8	0.47	33.0	13.4	0.59
PS	J.P.	M	3; 1	0.56	38.7	11.9	0.69
PS	M.M.	F	3; 8	0.61	28.5	7.7	0.59
PS	P.R.	F	2; 11	0.63	25.2	8.6	0.66
PS	K.N.	F	3; 9	0.70	25.1	9.9	0.61
					25.0	5.0	0.80
PS	A.V.	F	4; 10	0.71	23.0	9.9	0.51
PS	R.O.	F	7; 3	0.91	22.8	9.0	0.60
$\bar{x}$					27.7	9.4	0.63
SD±					5.6	2.6	0.09
Coarct.	S.K.	F	0; 1	0.22	51.8	31.6	0.39
Coarct.	M.K.	M	0; 2	0.22	26.4	13.1	0.51
Coarct.CMP	P.K.	M	0; 5	0.32	39.4	14.8	0.62
Coarct.MR.MS	D.H.	F	6; 1	0.83	132.0	80.0	0.30
MS.CMP	M.S.	F	4; 2	0.55	53.5	45.2	0.15

PS = pulmonary stenosis; Coarct. = coarctation of the aorta; CMP = cardiomyopathy; MR = mitral regurgitation; MS = mitral stenosis; LA_{max} = left atrial maximal volume; LA_{min} = left atrial minimal volume; LAVC = left atrial volume change = $(LA_{max} - LA_{min})/LA_{max}$; BSA = body surface area.

RESULTS

Maximal atrial volume, minimal atrial volume, and per cent volume change are charted in Table I. Average maximal atrial volume of the cases with pulmonary valvular stenosis was 27.7 cc./m^2 body surface area (BSA) with a standard deviation of 5.6 cc./m^2 BSA. Average minimal atrial volume was 9.4 cc./m^2 BSA (SD 2.6 cc./m^2 BSA), and the average volume difference was 18.8 cc./m^2 BSA (SD 4.9 cc./m^2 BSA). Per cent volume change was 63 per cent (SD 9 per cent). Atrial volume change on the average amounted to 40 per cent of the end-diastolic volume of the left ventricle; left atrial maximal volume was 51 per cent of the left ventricular end-diastolic volume.

Atrial filling in the cases with pulmonary stenosis occurred at approximately the same rate throughout the 3 periods. Atrial emptying, however, occurred at different rates (Fig. 3). During the first third of the atrial systole, emptying occurred at a relatively high rate. During this period approximately 50 per cent of the total atrial "stroke volume" was emptied into the left ventricle. During the second third, emptying slowed down (retarded inflow[9]). During the third period, emptying occurred at an increased rate as a result of atrial contraction. This pattern of atrial emptying was frequency dependent: in 3 patients with a mean heart rate of 91 (86–99) a marked plateau was observed in the second period. With increasing heart rates (5 patients, mean 125 beats/min., range 110–146) emptying rates during the 3 periods were almost equal.

Table II shows left ventricular residual volume, atrial volume difference and the amount of "conducted" blood volume expressed as per cent of left ventricular end-diastolic volume of the 5 pathologic cases studied. In all cases left ventricular end-systolic volume (residual volume) expressed as per cent of end-diastolic volume was increased. Atrial volume difference expressed as per cent of left ventricular end-diastolic volume was increased in 2 patients with coarctation of the aorta and markedly in-

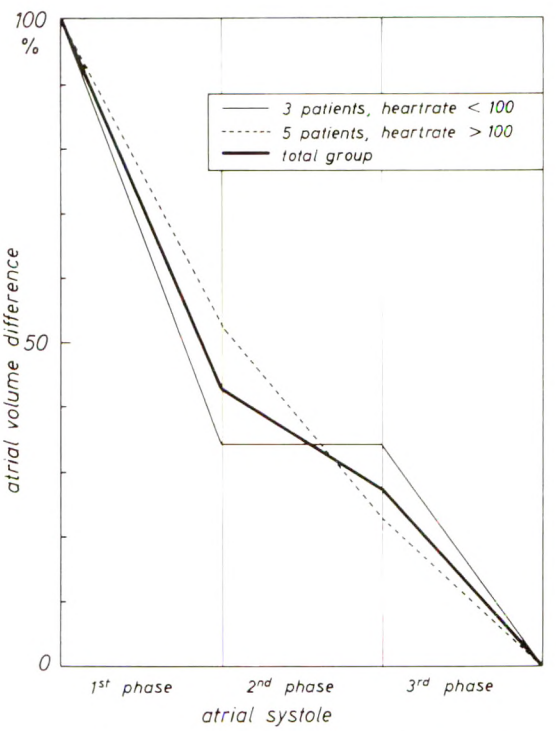


FIG. 3. Emptying pattern of the left atrium in 7 cases (8 films) with pulmonary valvular stenosis. Atrial systole was arbitrarily divided into 3 equal parts. The average atrial volume decrease of the 7 cases during the respective periods—expressed as per cent of total atrial volume difference—is shown. For details see text.

creased in the patient with combined coarctation of the aorta and mitral lesion. It was decreased in the 2 patients with cardiomyopathy.

DISCUSSION

Determination of left atrial volumes in adult patients have been carried out by several authors.[1,3,8,10,11,14,15–17] Most of the published data were not corrected for body surface area, so that comparison with these values is limited to certain authors. In "normal" children, to our knowledge, such determinations were performed only by Graham and associates.[7]

Our values are in close agreement with those of Graham *et al.*,[7] especially if the fact is taken into account that our oldest patient was only 7.3 years old. There is also close agreement with the data of Murray and co-workers[11] (Table III).

In analyzing left ventricular and atrial volume curves, one important fact becomes evident—atrial volume difference is markedly smaller than left ventricular stroke volume. This implies that the filling of the ventricle is not solely accomplished by left atrial volume difference. Atrial volume change on the average amounted to 40 per cent of left ventricular end-diastolic volume in our cases, and in the cases of Graham *et al.*,[7] to 35.8 per cent. (This value was calculated from the authors' data. For better comparison, only patients under the age of 8 years were considered.) This discrepancy between atrial and ventricular "stroke volume" had already been noted by Björk and Lodin,[3] who explained it as the result of an inaccurate volume determination because of the difficulty in distinguishing the left atrium from the first part of the pulmonary veins. This explanation, however, is not sufficient. The fact that atrial fibrillation does not necessarily impede ventricular filling shows that the main action of the left atrium cannot be the filling of the left ventricle by active contraction. Boehme,[4] Nilsson and Kramer,[12] Asmussen and Nielsen,[2] Grant and associates,[8] and Murray

TABLE II

RESIDUAL LEFT VENTRICULAR VOLUME, LEFT ATRIAL VOLUME DIFFERENCE AND CONDUCTED BLOOD VOLUME EXPRESSED AS PER CENT OF LEFT VENTRICULAR END-DIASTOLIC VOLUME

Diagnosis	Residual Volume (per cent of LVEDV)	Left Atrial Volume Difference (per cent of LVEDV)	Conducted Volume* (per cent of LVEDV)
Normal	35	40	25
Coarct.	42.2	54.2	3.6
Coarct.	34.4	50.4	15.2
Coarct. +CMP	57.2	23.4	19.4
Coarct. +MR+MS	31.0	75.6	− 6.6
MS+CMP	77.8	9.5	12.7

* Calculated as the difference between 100 per cent and residual, left ventricular volume plus left atrial volume difference.

Coarct.=coarctation of the aorta; CMP=cardiomyopathy; MR=mitral regurgitation; MS=mitral stenosis; LVEDV=left ventricular end-diastolic volume.

Table III

COMPARISON OF OUR LEFT ATRIAL VOLUME DATA WITH THOSE OF OTHER INVESTIGATORS

Authors	No. of Patients	LA_{max} (cc./m²)	LA_{min} (cc./m²)	LAVD (cc./m²)	LAVC	LAVD / LVEDV	LA_{max} / LVEDV
Graham *et al.*[7]	19	33.6 (SD 10.1)	12.7 (SD 4.2)		0.61 (SD 0.13)	0.36*	0.57
Murray *et al.*[11]	22 (adults)	35.0 (SD 8.7)	17.8 (SD 5.8)	18.0 (SD 7.4)			
Present investigation	8	27.7 (SD 5.6)	9.4 (SD 2.6)	18.8 (SD 4.9)	0.63 (SD 0.09)	0.40	0.51

* Calculated from the author's data, only the patients under the age of 8 years were considered. Abbreviations as in Table 1; LAVD = left atrial volume difference; LVEDV = left ventricular end-diastolic volume; SD = ± standard deviation.

et al.[11] were able to show that the left atrium functions as an elastic reservoir for the left ventricle. During ventricular systole the left atrium is dilated by the continuing blood flow from the pulmonary veins. After opening of the mitral valve during ventricular diastole, blood flows from the atrium into the left ventricle. However, atrial volume decrease is less than left ventricular volume increase, therefore some portion of blood must be conveyed into the left ventriclewithout affecting atrial volumedifference. For this portion of blood (approximately 25 per cent of left ventricular end-diastolic volume), the left atrium serves only as a conductive pipe.

As investigations of Grant and co-workers[8] have shown, the left atrium stores blood and energy as it is distended during ventricular systole. This energy is used to convey the blood from the left atrium into the left ventricle during the first part of atrial systole, as investigations of Gribbe *et al.*[9] in dogs, and our own investigations have shown. A period then follows in which atrial volume decreases at a smaller rate. This was called the phase of "retarded inflow" by Gribbe and associates.[9] However, as ventricular filling does not slow down correspondingly, flow cannot be retarded. It is probably during this phase that blood is conducted from the atrium into the left ventricle with little or no active contribution of the left atrium. Only at the end of ventricular diastole does atrial volume decrease, again markedly, as a result of active atrial contraction. The average volume change during this third phase of atrial emptying in our 7 cases of pulmonary valvular stenosis was 25 per cent of the total atrial volume change.

Murray and co-workers[11] distinguish only 2 phases during atrial systole—conduction phase and contraction phase—whereas Gribbe and associates[9] and we ourselves observed 3 distinguishable phases with varying rates of blood flow. This is probably due to the fact that Murray *et al.*[11] used serial angiocardiographies with maximally 12 exposures per second, whereas Gribbe and co-workers and the present study used cineangiocardiography with 48 and 50 frames/sec., respectively.

As should be expected from theoretic considerations,[11] some of the 5 pathologic cases studied showed marked alterations of their filling and emptying pattern. In those cases where there was an increased resistance to left ventricular ejection, left atrial volume difference (expressed as per cent of end-diastolic left ventricular volume) was greater than in the "normal" cases studied (Table II). Residual left ventricular volume (as per cent of LVEDV) was normal in 2 of these cases and only slightly elevated in 1. This implies that in these cases the "conducted" volume must be smaller than in the control group. In other words, in

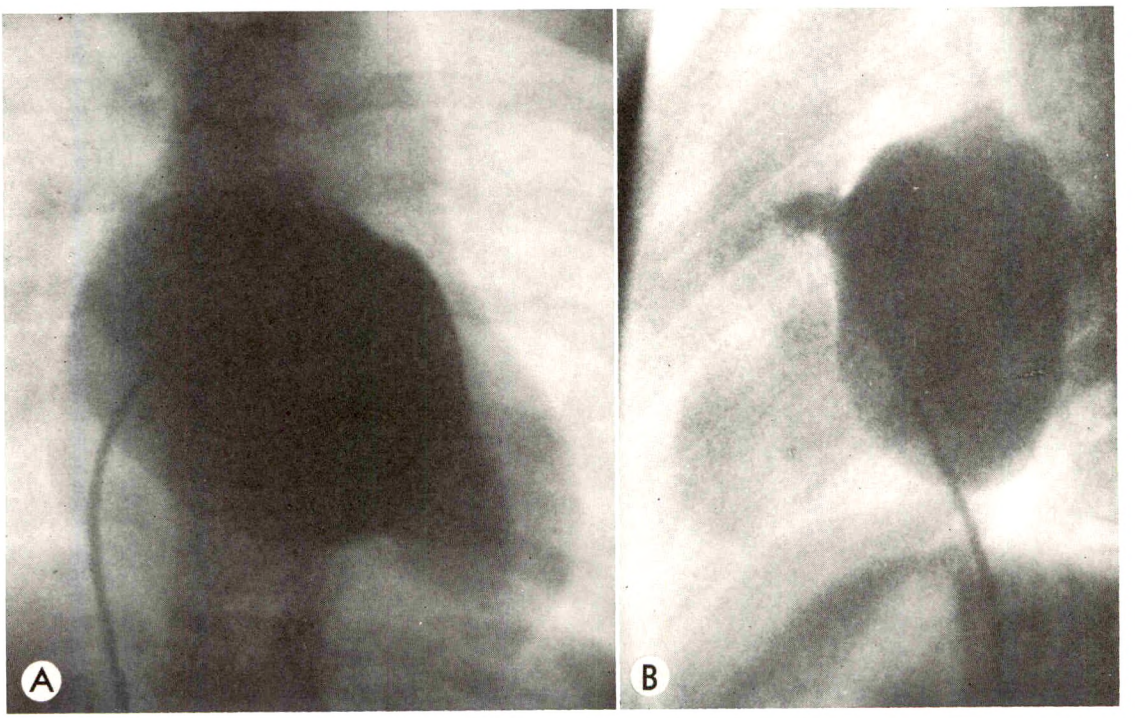


FIG. 4. Cineangiocardiograms of a 6.1 year old girl with mitral stenosis and regurgitation and coarctation of the aorta. Injection into the grossly enlarged left atrium, which showed only slight excursions. (*A*) Sagittal projection. (*B*) Lateral projection.

these cases with left ventricular outflow obstruction, atrial volume difference is of greater significance for left ventricular filling than in the normal. The highest atrial volume difference was found in the case with coarctation of the aorta combined with mitral regurgitation and mitral stenosis. It is noteworthy that the large atrial contribution to left ventricular filling was accomplished by only slight atrial excursions. As a result of gross atrial enlargement (Fig. 4, *A* and *B*), relatively small atrial excursions result in a large amount of ejected blood.

SUMMARY

Left atrial volume determinations were carried out on 13 cineangiocardiograms from 12 patients ranging in age from 1 month to 7 years. Seven of the patients had pulmonary valvular stenosis and were considered as a "normal" control group. Four patients had left ventricular outflow obstruction, in 1 this was combined with mitral stenosis and regurgitation. One child had severe mitral stenosis with severe myocardiopathy.

Left atrial and left ventricular volume curves were obtained by calculating the volumes frame by frame (50 frames/sec.). Arvidsson's method[1] (direct measurement of axes) was used to calculate left atrial volumes. Left ventricular volumes were calculated using the "area-length-method" of Dodge *et al.*[5,6]

Average maximal atrial volume was 27.7 cc./m^2 BSA. Average minimal atrial volume was 9.4 cc./m^2 BSA and the average atrial volume difference was 18.8 cc./m^2 BSA. Per cent volume change was 63 per cent. Atrial volume difference on the average amounted to 40 per cent of the end-diastolic volume of the left ventricle; left atrial maximal volume was 51 per cent of the left ventricular end-diastolic volume.

Atrial volume difference was therefore markedly smaller than left ventricular stroke volume. This implies that the filling of the left ventricle is not solely accomplished by left atrial contraction. During

ventricular systole, the left atrium is distended by the continuing blood flow from the pulmonary veins. After opening of the mitral valve, blood flows from the left atrium into the left ventricle; however, left atrial volume decrease is less than left ventricular volume increase. Therefore, some portion of blood is conveyed into the left ventricle without affecting left atrial volume difference. For this portion the left atrium serves only as a conductive pipe.

Three distinct phases of atrial emptying can be distinguished at heart rates below 100 beats/min. During the first phase approximately 50–65 per cent of the total atrial volume decrease takes place. Then a phase follows in which atrial volume decreases at a slower rate. It is during this phase that blood is conducted from the atrium into the left ventricle with little or no active contribution of the left atrium. Only at the end of ventricular diastole does atrial volume decrease again markedly as a result of active atrial contraction (at the end of the P-wave). These results are in close agreement with the investigations of Gribbe and associates[9] in dogs and with the investigations of Grant *et al.*[8] and Murray and co-workers[11] (who used serial angiocardiography) in adults.

In 5 patients with left atrial, left ventricular, and aortic lesions, marked alterations of their filling and emptying patterns were noted. In the cases with increased resistance to left ventricular ejection, atrial "stroke-volume" proved to be of greater significance for left ventricular filling than in the normal. In the 2 cases with cardiomyopathy, left atrial volume difference was decreased.

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CARDIOANGIOGRAPHIC EVALUATION OF THE RELATIONSHIP BETWEEN ATRIOVENTRICULAR AND SEMILUNAR VALVES: ITS DIAGNOSTIC IMPORTANCE IN CONGENITAL HEART DISEASE*

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ANATOMOPATHOLOGIC investigations in recent years have shown that the relationship between the atrioventricular and semilunar valves follows a definite pattern and plays an important role in the identification of different types of congenital heart anomalies. However, the angioroentgenographic evaluation of this relationship has not yet been fully defined.

In this paper the anatomic relationship between the atrioventricular and semilunar valves is considered in the light of the embryologic development of the conotruncal region of the heart. The relevant cardioangiographic features in the normal heart and in certain congenital heart diseases with conotruncal anomalies are then described, and their diagnostic value discussed.

This study is based on the re-evaluation of 35 cases of corrected transposition of the great vessels, 25 cases of complete transposition of the great vessels, 9 cases of persistent truncus arteriosus, 6 cases of origin of both great vessels from the right ventricle and 55 cases of tetralogy of Fallot, for a total of 130 cases.

Our knowledge concerning the relationship between the great vessels to assist in defining the type of congenital heart disease has progressed during the years. Earlier definitions were based upon alterations in the relationship of the great vessels relative to each other and to the ventricles from which they originated.[1] Another approach was introduced by Lev,[5] who stated that this anatomic classification is not sufficiently inclusive and added another criterion: the relationship of the arterial trunks to the ventricular septal defect and its pathophysiologic consequences. Recently, based on a new concept of pathogenesis, the point of references has again been changed to the relationship between the semilunar valves and the atrioventricular valves.[4] According to this latter concept, in the normally developed right ventricle the infundibulum (embryologically the muscular conus) is located below the pulmonary valve and separates the ventrally, cranially and relatively left-sided pulmonary valve from the tricuspid valve. In the normal left ventricle, no conal myocardium is present below the relatively right-sided, low and posteriorly located aortic valve. Therefore, a fibrous continuity exists between the posterior leaflet of the aortic valve and the anterior leaflet of the mitral valve.

EMBRYOLOGY

A consideration of the embryologic development of the outflow tracts of the ventricles and the great vessels is necessary for a better understanding of the anatomic findings.

It is appropriate to start with the primitive straight tube heart with its 4 distinct components: the truncus arteriosus, the bulbus cordis, the ventricular and the atrial segments. It is from the truncus arteriosus that the great vessels develop. The bulbus

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cordis is destined to become the outflow tract of the right ventricle and the fore-runner of the muscular conus (infundibulum), while the primitive ventricle is the fore-runner of the left ventricle and the sinus part of the right ventricle. Following the bending of the cardiac tube to the right in levocardia (the converse is dextrocardia), the muscular conus will be situated on the upper left side of the cardiac loop. Due to this left and cranial localization of the muscular conus, an inseparable relationship develops between the latter and that part of the vascular trunk from which the pulmonary artery will develop. Consequently, the growing and anteriorly protruding muscular conus carries with it the concomitantly developing pulmonary artery, pushing it cranially and ventrally. The final result is one in which the pulmonary valve is localized normally; that is, in a ventral, cranial and left-sided position, continuous with the muscular conus. The normal development of the muscular conus also contributes to the formation of the tricuspid valve. Two leaflets of each atrioventricular valve originate embryologically from the respective anterior and posterior endocardial cushions, but the third, the septal leaflet of the tricuspid valve, develops from the normally developing muscular conus. Consequently, the tricuspid valve is related to the ventricle in which the conus is located and separated from the pulmonary valve. Since there is no muscular conus present below the aortic segment of the truncus, the aortic valve remains caudally, dorsally, relatively right-sided and continuous with the mitral valve.

In the condition in which there is abnormal development of the conotruncus, the muscular conus is located below the pulmonary valve, but is closely related to the aortic valve. Consequently, the aortic valve is higher and anterior, not continuous with the mitral valve and separated from the tricuspid valve by the muscular conus. On the other hand, because of the absence of the muscular conus below the pulmonary valve, the latter is caudal and dorsal and, in general, continuous with the mitral valve. In these situations, where the muscular conus is closely related to the aortic valve, separating it from the atrioventricular valve, transposition of the great vessels is always present.[8]

CARDIOANGIOGRAPHY

In general, the semilunar valves can be visualized by cardioangiography better than the atrioventricular valves. The optimal demonstration of the atrioventricular valves is achieved by selective injection of the contrast material into the appropriate ventricle. The tricuspid valve is best visualized by this method during ventricular systole in both the lateral and frontal views by intraatrial bulging of the leaflets in the form of a crescent limited by the tricuspid ring (Fig. 1*A*). In ventricular diastole, the valvular ring can be seen only while the floating leaflets are hidden in the contrast filled ventricle (Fig. 1*B*). The noncontinuity of the tricuspid valve and the pulmonary valve in the normal heart due to the interposition of the crista supraventricularis is optimally visualized in the lateral view (Fig. 1*B*). On the other hand, the continuity of the anterior leaflet of the mitral valve with the aortic valve, forming the dorsal border of the outflow tract of the left ventricle is well visualized in diastole in the lateral view; it has a straight or slightly convex defined border, sometimes crescentic or oval shaped; the oval shape results from the accumulation of contrast material behind the leaflets, while nonopacified blood passes through the mitral orifice (Fig. 2*A*). Occasionally, in the frontal view, the line of attachment of the posterior mitral leaflet can be seen in diastole due to the accumulation of contrast material below its edges (Fig. 2*B*).

Because selective right heart cardioangiography is usually preferred for the diagnosis of congenital heart disease, the evaluation of the relationship between the mitral and the aortic valves has been neglected. However, if attention is given to this detail it can be recognized in most cases

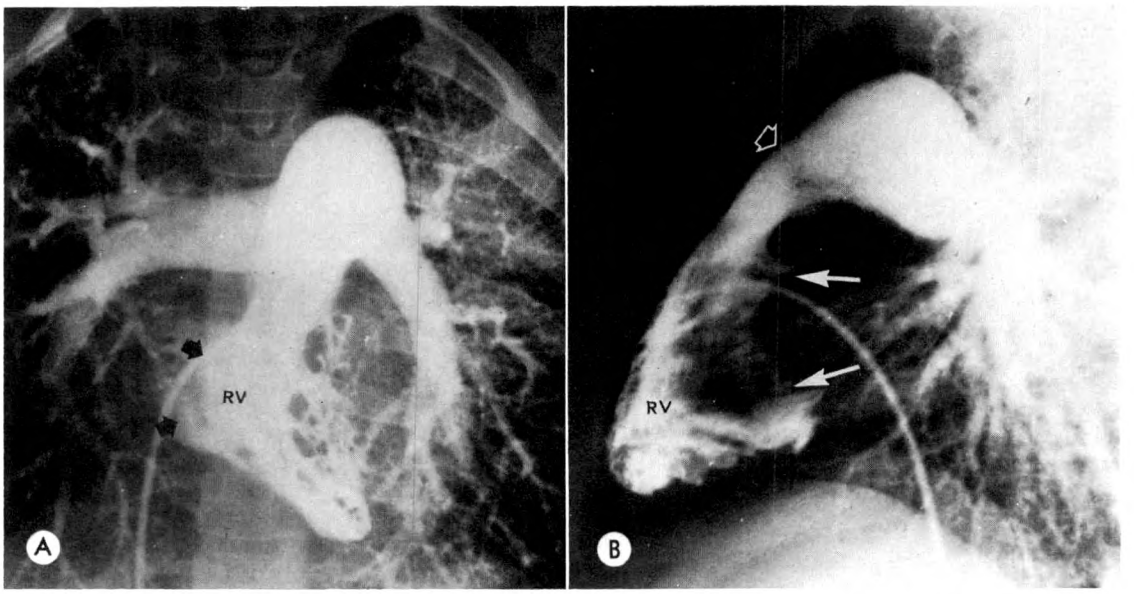


FIG. 1. Selective right ventriculography. (*A*) Frontal and (*B*) lateral views in a case of mild pulmonic stenosis showing the normal relationship of the tricuspid and pulmonary valves. (*A*) The intraatrial bulging of the tricuspid valve leaflets (black arrows) is demonstrated in systole. (*B*) The tricuspid ring (between white arrows) in diastole. There is no continuity of the tricuspid and pulmonary valve (hollow arrow), separated by the outflow tract of the right ventricle. RV = Right Ventricle.

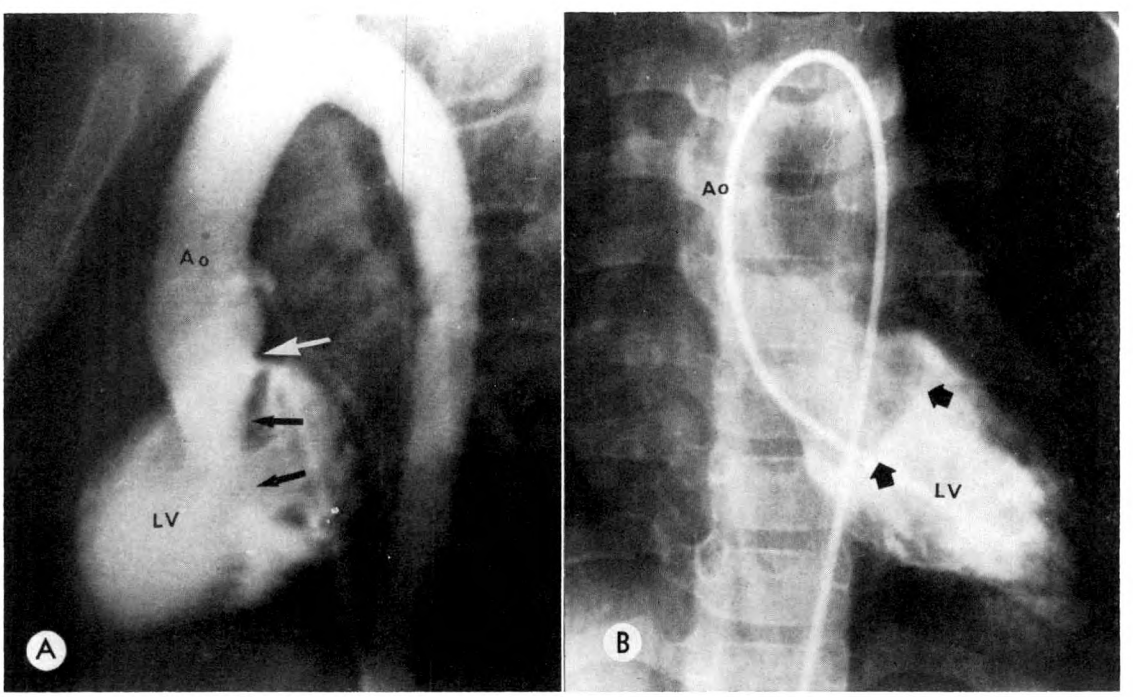


FIG. 2. Selective left ventriculography in diastole. (*A*) Lateral and (*B*) frontal views in a normal subject showing the normal relationship of the mitral and aortic valves. (*A*) The anterior leaflet of the mitral valve (black arrows), which forms the posterior border of the outflow tract of the left ventricle, is continuous with the aortic valve (white arrow). (*B*) The edge of the posterior leaflet of the mitral valve (black arrows) is not continuous with the aortic valve. LV = Left Ventricle; Ao = Aorta.

in the levophase of the examination, or in those instances when the left ventricle is visualized through a ventricular septal defect with right-to-left shunt.

In tetralogy of Fallot the relationship of the atrioventricular and semilunar valves is normal. There is continuity between the mitral and aortic valves, while the tricuspid valve is separated from the pulmonary valve by the interposed crista supraventricularis (Fig. 3, *A–D*). The diagnostic importance of the relationship of the atrioventricular valves and the semilunar valves is apparent, especially in the severe cases of tetralogy in which there is significant dextroposition and dilatation of the aorta, which may lead to the false assumption of some form of transposition. However, the continuity of the mitral valve with the low positioned aortic valve and the noncontinuity of the tricuspid and the high positioned pulmonary valve in tetralogy of Fallot rule out this false assumption (Fig. 4, *A* and *B*).

The above normal relationship is also true in persistent truncus arteriosus. Continuity of the mitral valve and the truncal valve rules out any malformation of the transposition complex group.

In complete transposition of the great vessels the muscular conus is located below the aortic valve, separating it from the tricuspid valve. There is no continuity between the aortic and mitral valve, the latter being continuous with the pulmonary valve (Fig. 5, *A–D*). Occasionally, in this condition, part of the muscular conus extends into the left ventricular outflow tract, causing subpulmonary stenosis. Although this additional fibromuscular tissue is interposed between the pulmonary valve and the mitral valve, there is still continuity between the mitral and the pulmonary valves on cardioangiography (Fig. 5*C*).

Corrected transposition of the great vessels is defined anatomically as transposition of the great vessels, with inversion of the ventricles together with their atrioventricular valves.[7] It follows, therefore, that the aorta originates from a left-sided anatomically right ventricle. The atrioventricular valve of this ventricle is tricuspid and is separated from the aortic valve by the interposed muscular conus (infundibulum). The pulmonary artery originates from a right-sided anatomically left ventricle. The atrioventricular valve of this ventricle is mitral and is in continuity with the pulmonary valve due to the absence of muscular conus (Fig. 6, *A* and *B*). In some cases, additional tissue may exist below the pulmonary valve, slightly separating the right atrioventricular valve from the pulmonary valve (Fig. 7, *A* and *B*).

In origin of both great vessels from the right ventricle, the muscular conus can be closely related to the aortic valve or to the pulmonary, or intermediate between the two.[3,6,8] In the majority of cases no continuity exists between the aortic valve and the mitral valve (Fig. 8, *A* and *C*). Occasionally, cases with this condition have been reported where there was continuity between the aortic and the mitral valves at necropsy.[2,6]

The evaluation of the relationship between the atrioventricular valves and the semilunar valves is particularly helpful in the differential diagnosis of transposition complexes. Thus (Table 1) in both complete and corrected transposition there is no continuity between the left atrioventricular valve (mitral in complete transposition, tricuspid in corrected transposition) and the aortic valve. However, in corrected transposition the right atrioventricular valve (mitral) is continuous with the pulmonary valve (Fig. 6*A*), while in complete transposition, the tricuspid valve is not continuous with the aortic valve (Fig. 5, *A–C*). Furthermore, in complete transposition of the great vessels and origin of both great vessels from the right ventricle there is no continuity between the mitral and aortic valves, while in complete transposition of the great vessels there is continuity between the mitral and the pulmonary valves (Fig. 5*C*), which is usually not the case with origin of both great vessels from the right ventricle (Fig. 8, *A–D*).

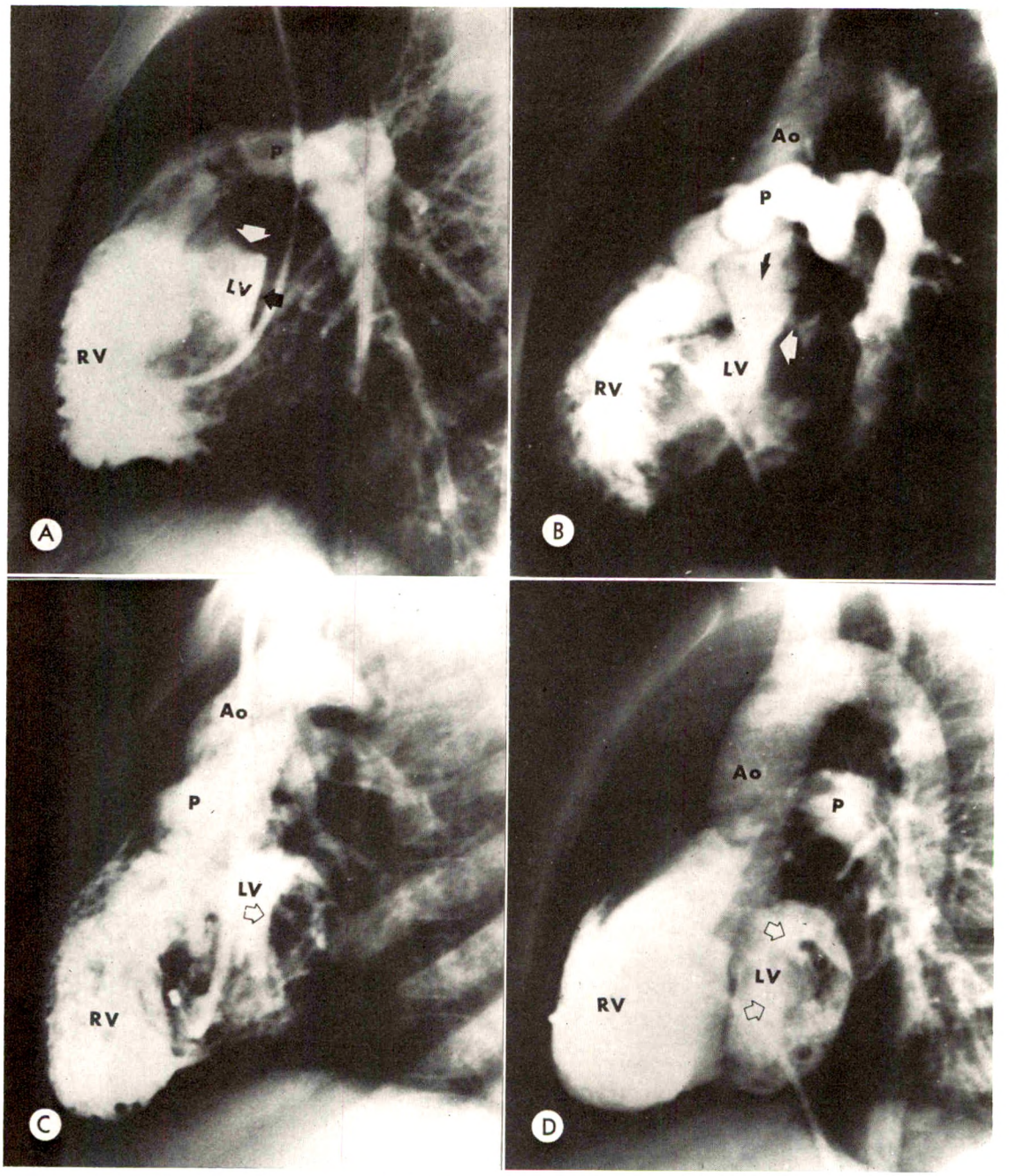


FIG. 3. Selective right ventriculography. (*A–D*) Lateral views, in 4 cases of tetralogy of Fallot. The different shapes assumed by the mitral valve and its continuity with the aortic valve are demonstrated. (*A*) Contrast material passed through the ventricular septal defect into the left ventricle, but the aorta is not yet visualized. The aortic valve leaflets (white arrow) are seen indirectly by their imprints into the left ventricular outflow tract. The anterior leaflet of the mitral valve (black arrow), appearing as a vertical straight line, borders the contrast material in the left ventricle. The continuity between the mitral and aortic valves is clearly seen. (*B*) The anterior leaflet of the mitral valve (white arrow), appearing as a slightly concave line, forms the posterior border of the outflow tract of the left ventricle and is continuous with the aortic valve (black arrow). (*C* and *D*) The mitral valve (hollow arrows) appears as a crescentic filling defect, surrounded by the contrast material in the left ventricle. RV = Right Ventricle; LV = Left Ventricle; P = Main Pulmonary Artery; Ao = Aorta.

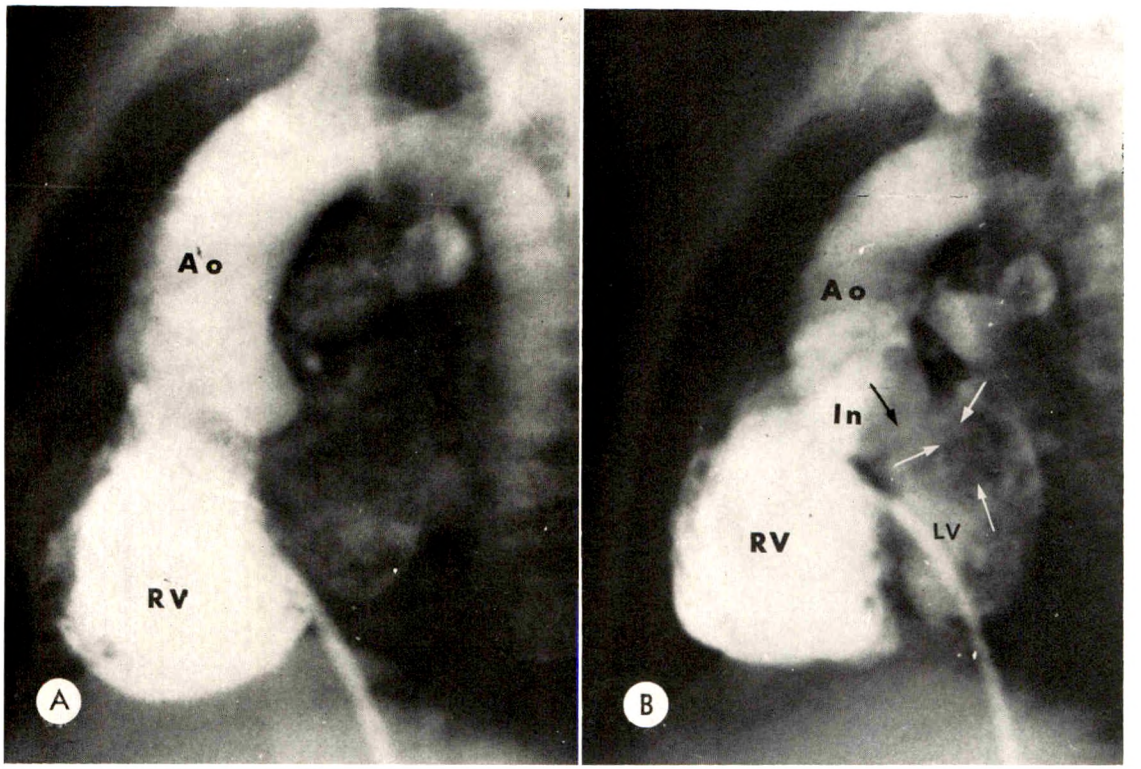


Fig. 4. Selective right ventriculography. Lateral views in (*A*) systole and (*B*) diastole, in a case of extreme tetralogy of Fallot with patent ductus arteriosus. (*A*) In systole, the extreme anterior position of the aorta gives the misleading impression that the aorta originates entirely from the right ventricle. (*B*) However, during diastole, the posteriorly tilted aortic valve (black arrow) is seen in continuity with the mitral valve (between white arrows) ruling out any kind of transposition. The presence of the infundibulum (In) excluded persistent truncus arteriosus. RV = Right Ventricle; LV = Left Ventricle; Ao = Aorta; In = Infundibulum.

Again, both in corrected transposition of great vessels and in origin of both great vessels from the right ventricle, the aorta arises from the anatomically right ventricle, and in both the left atrioventricular valve is noncontinuous with the aortic valve. However, in corrected transposition of the great vessels the right atrioventricular valve, being a mitral valve, is continuous with the pulmonic valve; this is in contradiction to the situation in origin of both great vessels from the right ventricle, where none of the atrioventricular valves are continuous with the semilunar valves (Fig. 9, *A–E*).

In the majority of cases of single ventricle there is transposition of the great vessels. In these cases, the muscular conus (infundibulum) is always below the aortic valve. When the position of the infundibulum and the aorta are to the right, there is no inversion of the infundibulum (complete transposition–Fig. 10, *A* and *B*). When the infundibulum and the aorta are to the left, there is inversion of the infundibulum (corrected transposition–Fig. 11, *A* and *B*). The pulmonary valve, due to its posterior position and the absence of the muscular conus below it, is continuous with the atrioventricular valves in the majority of cases (Fig. 10, *A* and *B*). In others, a ridge of muscle separates the pulmonary from the atrioventricular valves. Much less frequent are the cases with a single ventricle without transposition of the great vessels, in which there is usually a normal relationship between the atrioventricular valves and the semilunar valves (Fig. 12, *A–C*).

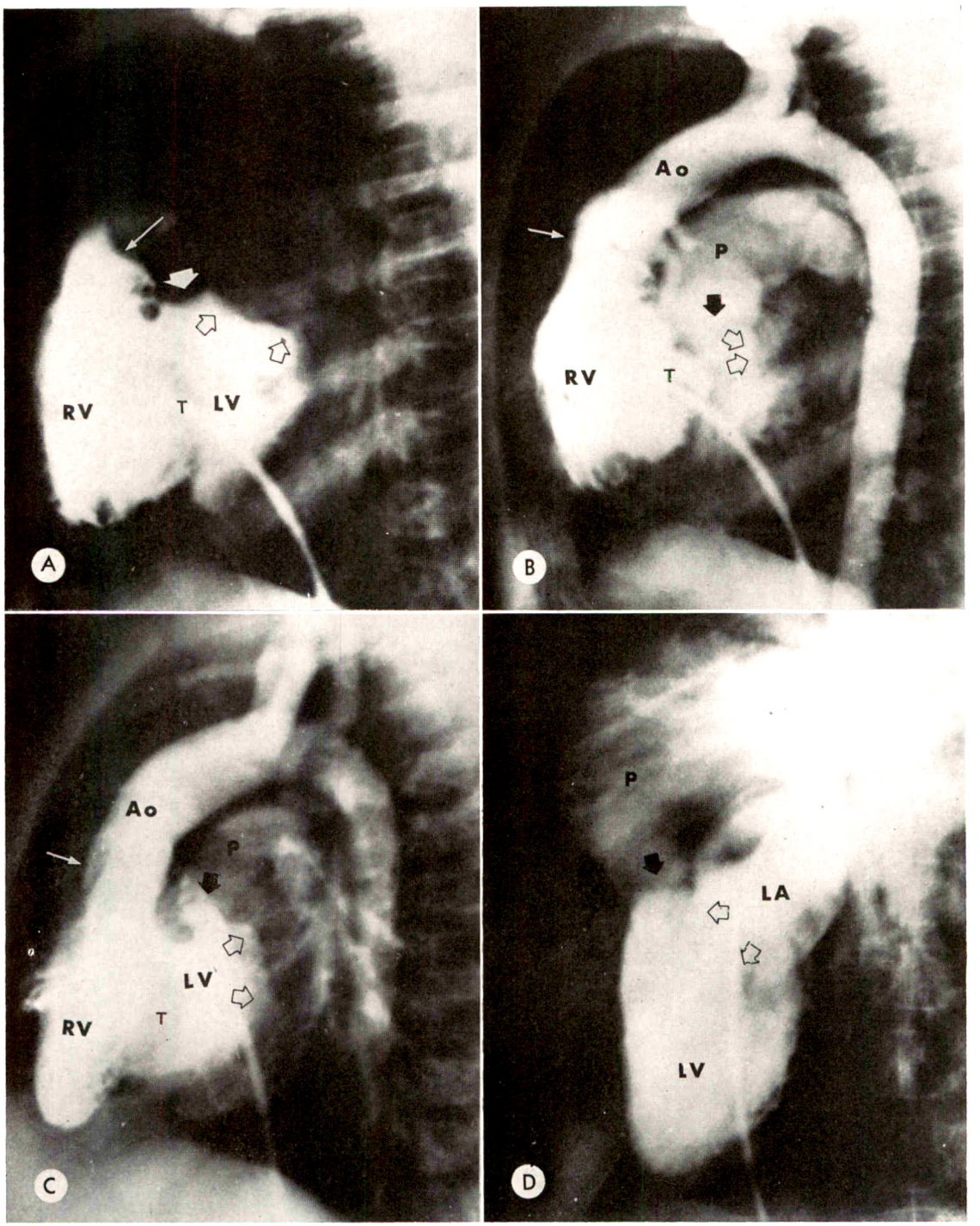


FIG. 5. Selective right ventriculography. (*A–C*) Lateral views, in 3 cases of complete transposition of the great vessels. The continuity between the pulmonary valve (black arrow) and the mitral valve (between hollow arrows), and the noncontinuity between the tricuspid valve and the aortic valve (white arrow) are demonstrated. (*D*) Selective angiocardiography into the left atrium illustrating another aspect of the continuity between the mitral valve (between hollow arrows) and pulmonary valve (black arrow). LV = Left Ventricle; RV = Right Ventricle; LA = Left Atrium; P = Main Pulmonary Artery; Ao = Aorta; T = Tricuspid Valve.

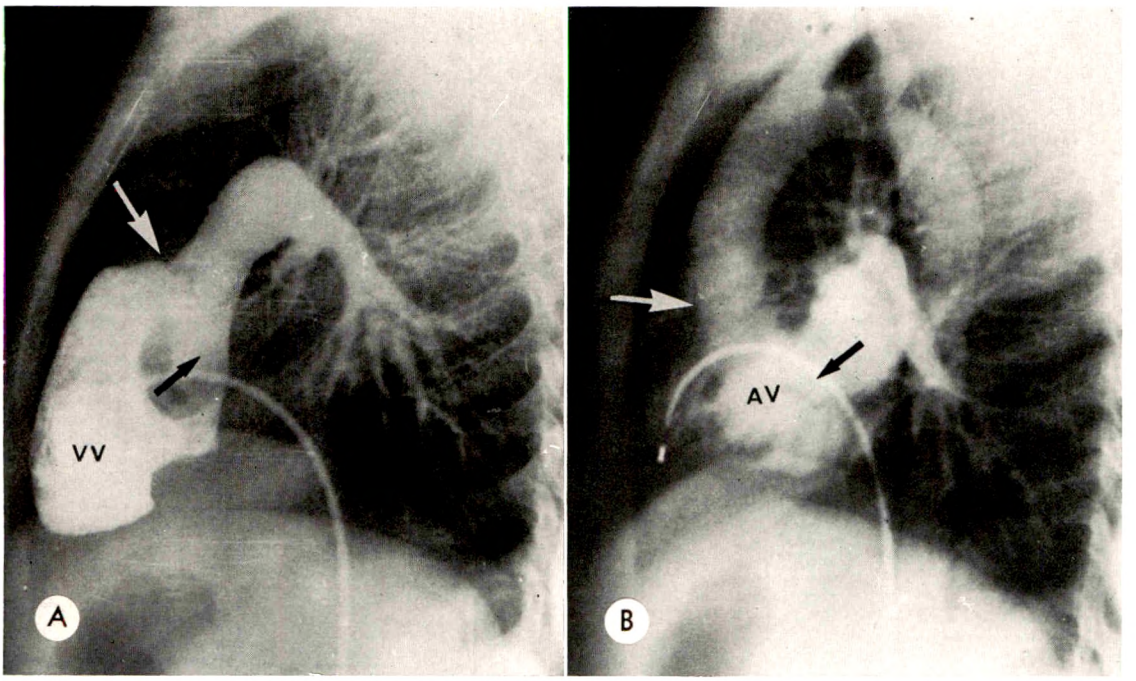


Fig. 6. (*A*) Venous and (*B*) arterial phases of selective right ventriculography, lateral view, in a case of corrected transposition of the great vessels. (*A*) The continuity of the right-sided atrioventricular valve (black arrow) and the pulmonary valve (white arrow) is shown. (*B*) The noncontinuity of the left atrioventricular valve (black arrow) and the aortic valve (white arrow) is demonstrated. VV = Venous Ventricle; AV = Arterial Ventricle.

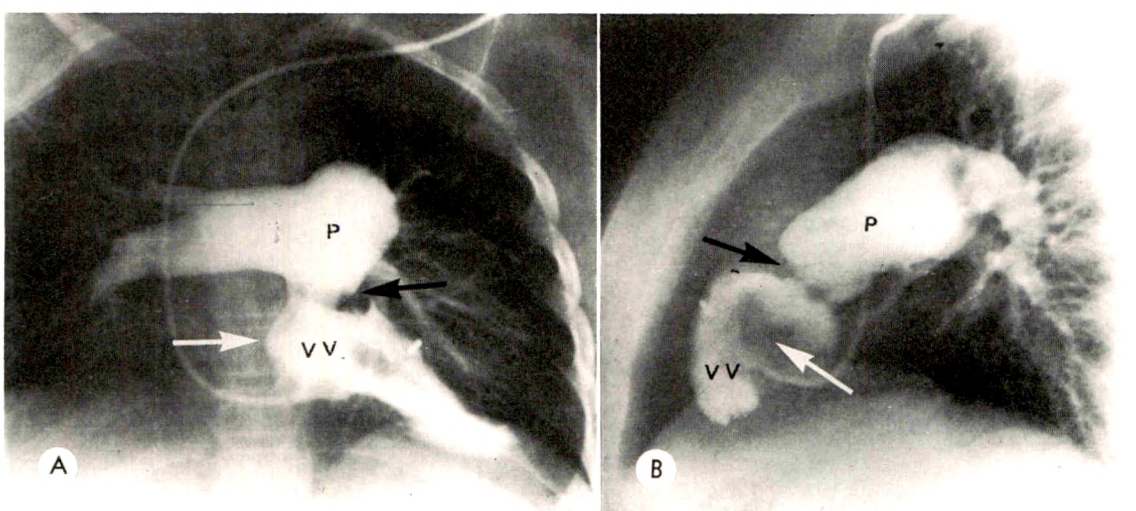


Fig. 7. Selective right ventriculography. (*A*) Frontal and (*B*) lateral views, in a case of corrected transposition of the great vessels with subpulmonary stenosis. The close relationship of the right-sided atrioventricular valve (white arrow) and the pulmonary valve (black arrow) is shown. Additional tissue, probably anomalous conal myocardium, below the pulmonary valve, causes subvalvular pulmonary stenosis, but the continuity between the atrioventricular and the pulmonary valves is still preserved. VV = Venous Ventricle; P = Main Pulmonary Artery.

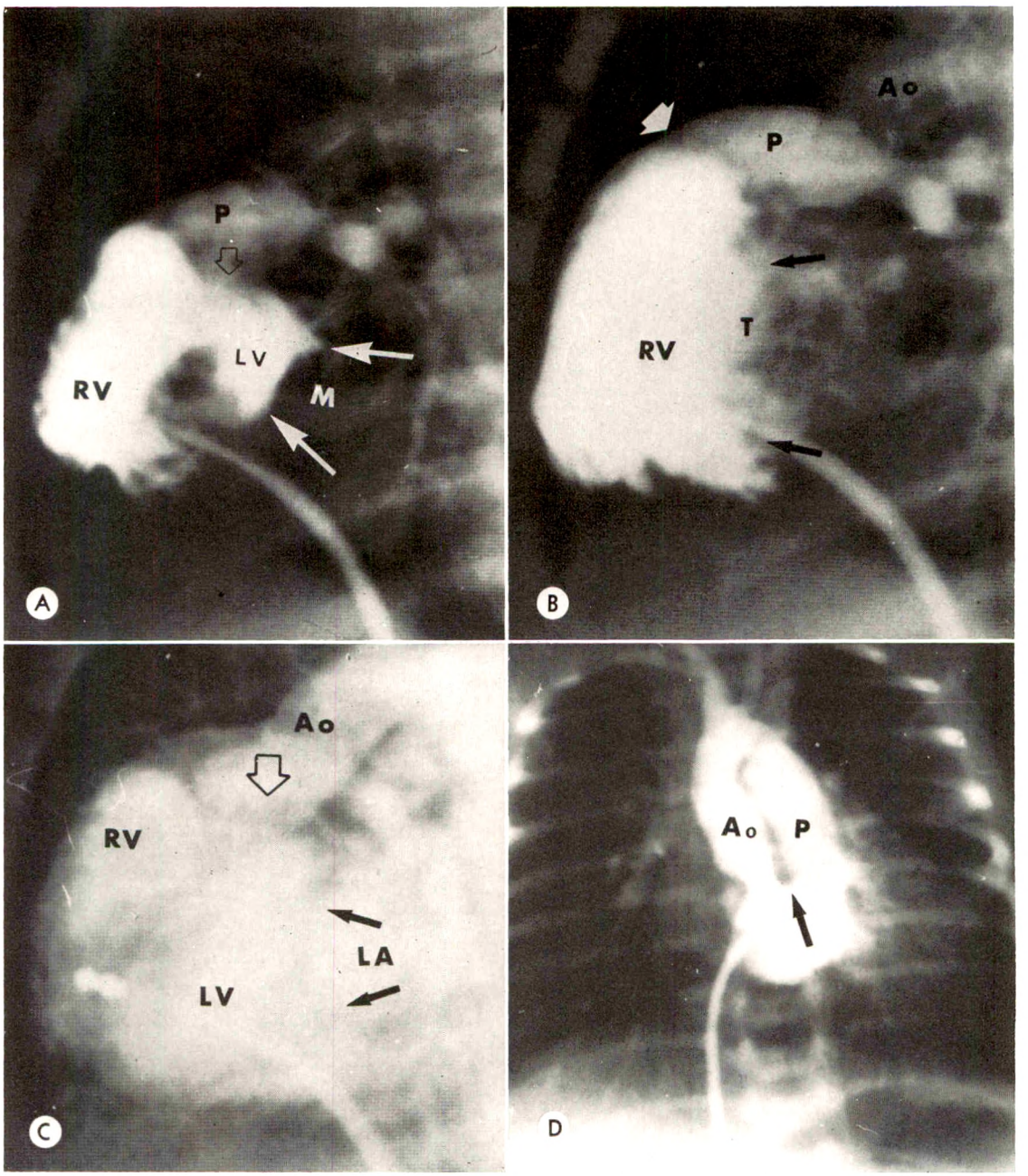


FIG. 8. Selective right ventriculography in a case of origin of both great vessels from the right ventricle. (*A* and *B*) Early venous phases, lateral view, systole and diastole. (*C* and *D*) Levophases, lateral and frontal view. The characteristic noncontinuity between the semilunar and the atrioventricular valves is visualized: in *A* and *C*, noncontinuity of the mitral valve (between arrows) with the aortic valve (hollow arrow); in (*B*) noncontinuity of the pulmonary valve (white arrow) with the tricuspid valve (between arrows). Furthermore, in the lateral view (*C*), the high and anteriorly positioned aortic valve (hollow arrow) and in the frontal view (*D*), the vertical position of the aorta (Ao) to the right of the pulmonary artery (P), separated at their origin by a part of the muscular conus (arrow), are diagnostic features of origin of both great vessels from the right ventricle. RV = Right Ventricle; LV = Left Ventricle; LA = Left Atrium; Ao = Aorta; P = Main Pulmonary Artery.

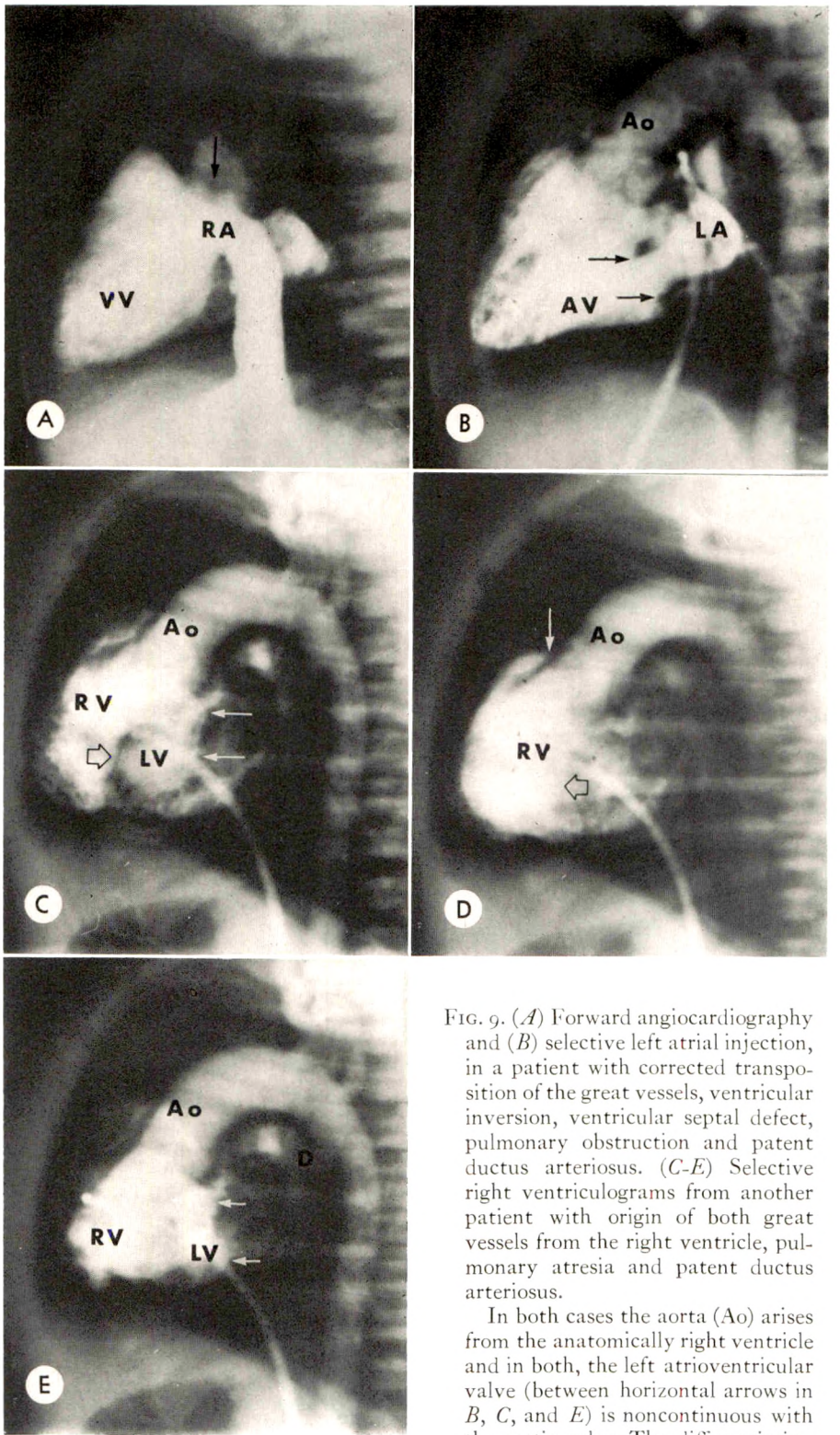


FIG. 9. (*A*) Forward angiocardiography and (*B*) selective left atrial injection, in a patient with corrected transposition of the great vessels, ventricular inversion, ventricular septal defect, pulmonary obstruction and patent ductus arteriosus. (*C-E*) Selective right ventriculograms from another patient with origin of both great vessels from the right ventricle, pulmonary atresia and patent ductus arteriosus.

In both cases the aorta (Ao) arises from the anatomically right ventricle and in both, the left atrioventricular valve (between horizontal arrows in *B*, *C*, and *E*) is noncontinuous with the aortic valve. The differentiating feature is that in the case with corrected transposition, the pulmonary valve (vertical arrow in *A*) is near the tricuspid valve (see right atrium RA), while in the case with origin of both great vessels from the right ventricle, the pulmonary valve (vertical arrow in *D*) is distant from the tricuspid valve (hollow arrow in *C* and *D*). VV = Venous Ventricle; RV = Right Ventricle; LA = Left Atrium; RA = Right Atrium; D = Patent Ductus Arteriosus.

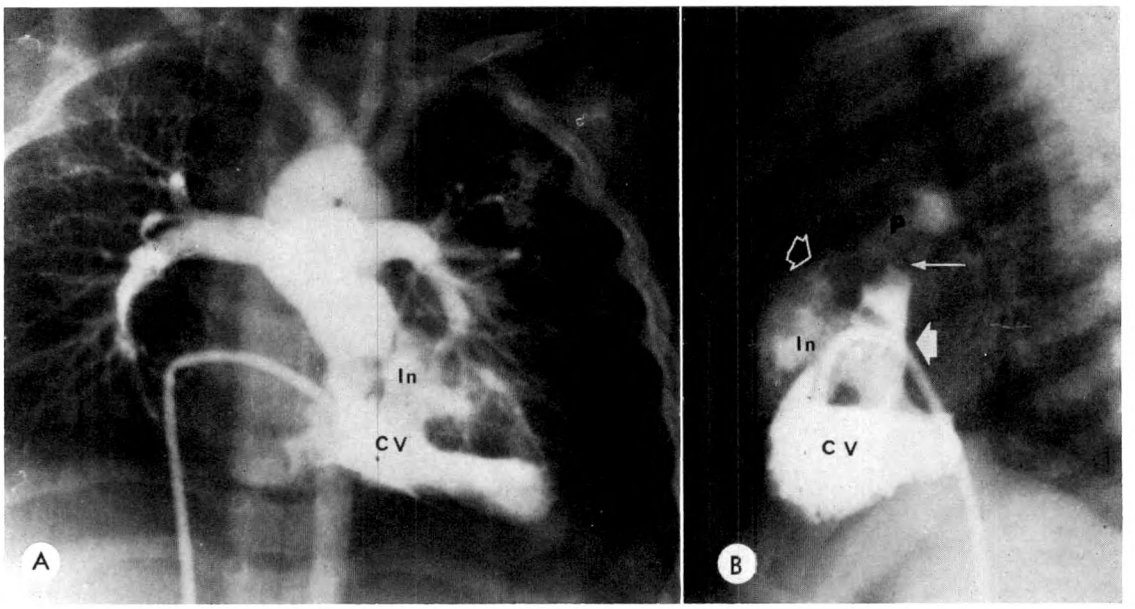


FIG. 10. Selective ventriculography. (*A*) Frontal and (*B*) lateral views, in a case of single ventricle with transposition of the great vessels, and noninversion of the infundibulum. The infundibulum (In) is situated on the left and anteriorly. There is continuity of the common atrioventricular valves (thick arrow) with the pulmonary valve (thin arrow). There is no continuity between the anteriorly positioned aortic valve (hollow arrow) and the common atrioventricular valves (thick arrow). CV = Common Ventricle; P = Main Pulmonary Artery.

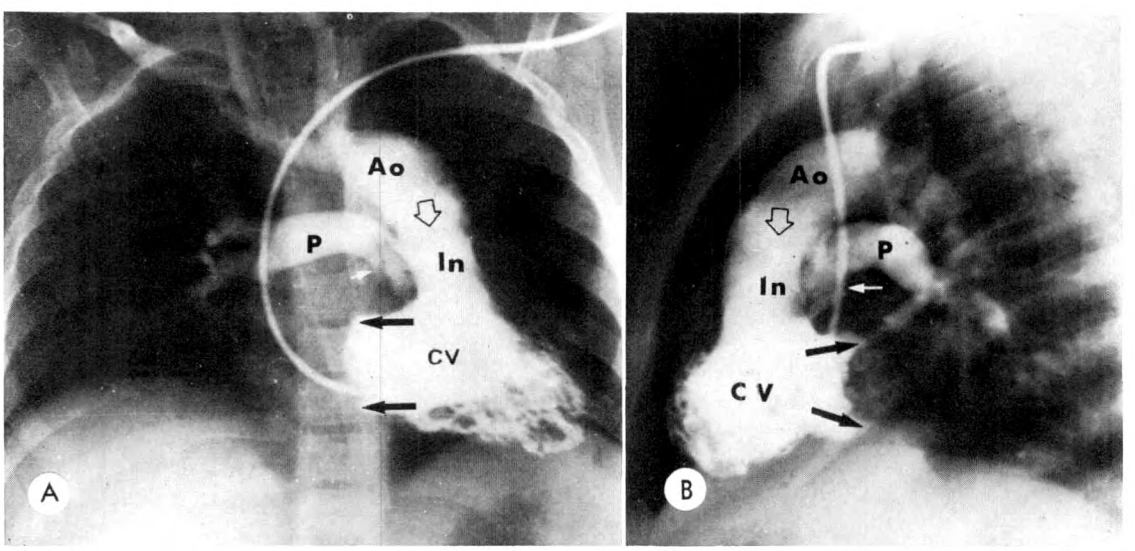


FIG. 11. Selective ventriculography. (*A*) Frontal and (*B*) lateral views, in a case of single ventricle with transposition of the great vessels, pulmonary stenosis and inversion of the infundibulum. The infundibulum (In) is situated on the left and anteriorly. There is noncontinuity of the common atrioventricular valves (between arrows) and the pulmonary valve (white arrow). The ventrally and high positioned aortic valve (hollow arrow) is separated from the common atrioventricular valves (between arrows) by the interposition of the infundibulum. In = Infundibulum; Ao = Aorta; P = Main Pulmonary Artery.

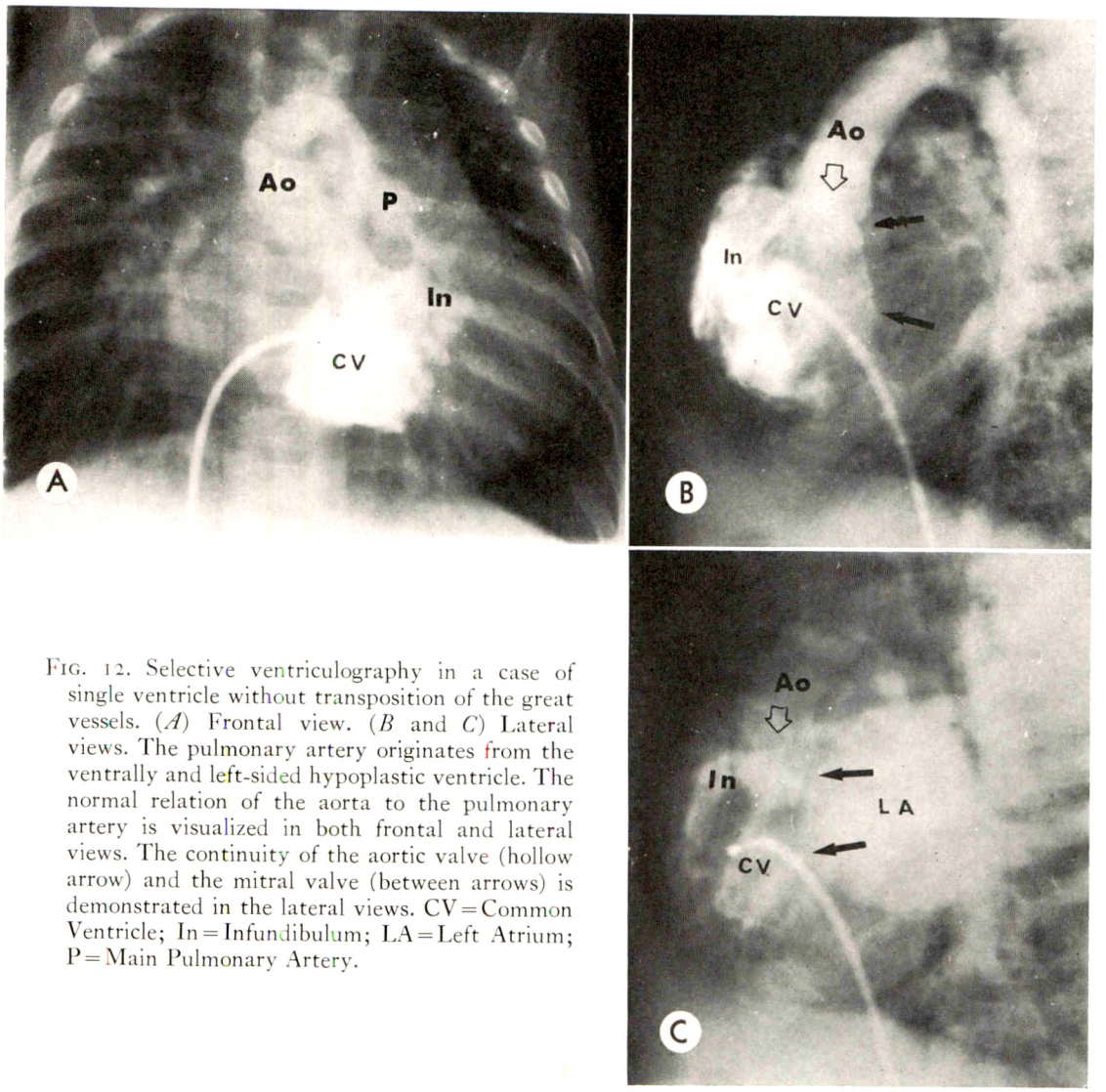


FIG. 12. Selective ventriculography in a case of single ventricle without transposition of the great vessels. (*A*) Frontal view. (*B* and *C*) Lateral views. The pulmonary artery originates from the ventrally and left-sided hypoplastic ventricle. The normal relation of the aorta to the pulmonary artery is visualized in both frontal and lateral views. The continuity of the aortic valve (hollow arrow) and the mitral valve (between arrows) is demonstrated in the lateral views. CV = Common Ventricle; In = Infundibulum; LA = Left Atrium; P = Main Pulmonary Artery.

Re-evaluation of the 130 cases of our material revealed that the usual diagnostic cardioangiographic features were inadequate for the exact anatomic diagnosis in some. However, the evaluation of atrioventricular-semilunar valve relationship (Table 1) was of great help for the exact identification of these cases. The difficulties arose chiefly in cases complicated with marked pulmonic stenosis. In extreme tetralogy of Fallot, as previously mentioned, a very dilated and dextroposed aorta may be projected in the lateral view anteriorly to a hypoplastic pulmonary artery, and falsely suggest complete transposition of the great vessels (Fig. 4, *A* and *B*). On the other hand, cases of origin of both great vessels from the right ventricle with pulmonary stenosis may be falsely diagnosed as tetralogy of Fallot (Fig. 13, *A* and *B*). In other instances (Fig. 14, *A–C*) the "tongue-like" appearance of the crista supraventricularis, which has been described as characteristic for origin of both great vessels from the right ventricle,[2] was confusing. In all these instances, a consider-

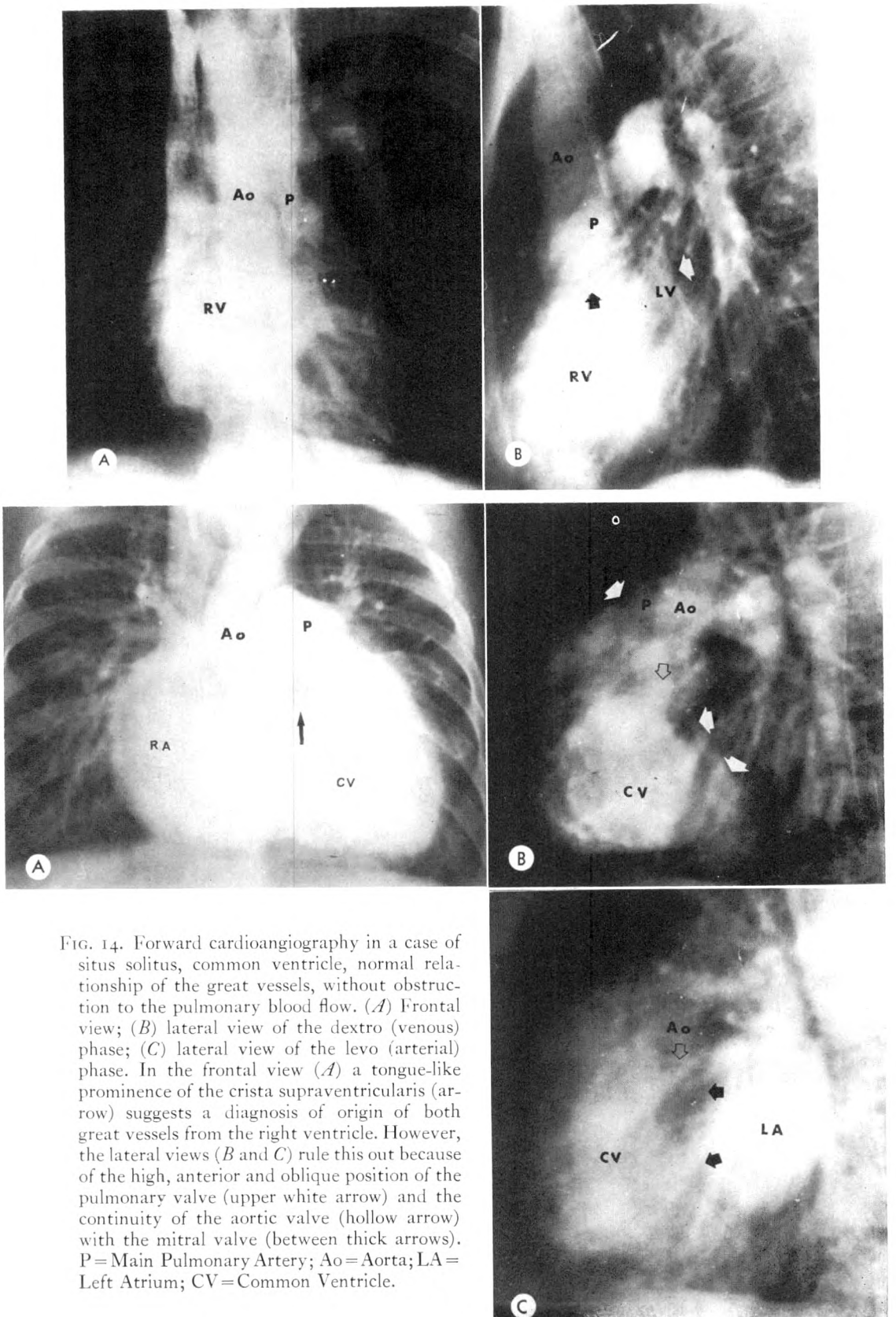


FIG. 14. Forward cardioangiography in a case of situs solitus, common ventricle, normal relationship of the great vessels, without obstruction to the pulmonary blood flow. (*A*) Frontal view; (*B*) lateral view of the dextro (venous) phase; (*C*) lateral view of the levo (arterial) phase. In the frontal view (*A*) a tongue-like prominence of the crista supraventricularis (arrow) suggests a diagnosis of origin of both great vessels from the right ventricle. However, the lateral views (*B* and *C*) rule this out because of the high, anterior and oblique position of the pulmonary valve (upper white arrow) and the continuity of the aortic valve (hollow arrow) with the mitral valve (between thick arrows). P = Main Pulmonary Artery; Ao = Aorta; LA = Left Atrium; CV = Common Ventricle.

Fig. 13. Forward cardioangiography. (*A*) Frontal and (*B*) lateral views in a case of origin of both great vessels from the right ventricle, with pulmonary stenosis. (*A*) In the frontal view, the position of the aorta to the right of the hypoplastic pulmonary artery, and the presence of right aortic arch are seen. (*B*) In the lateral view, the aorta is interposed. These features could confirm the diagnosis of tetralogy of Fallot; however, the noncontinuity of the mitral valve (white arrow) with the aortic valve (black arrow) excluded this diagnosis; it is diagnostic of origin of both great vessels from the right ventricle. The noncontinuity of the mitral valve with the pulmonary valve (seen superimposed on the aortic valve), excludes the diagnosis of complete transposition of the great vessels. RV = Right Ventricle; Ao = Aorta; P = Main Pulmonary Artery.

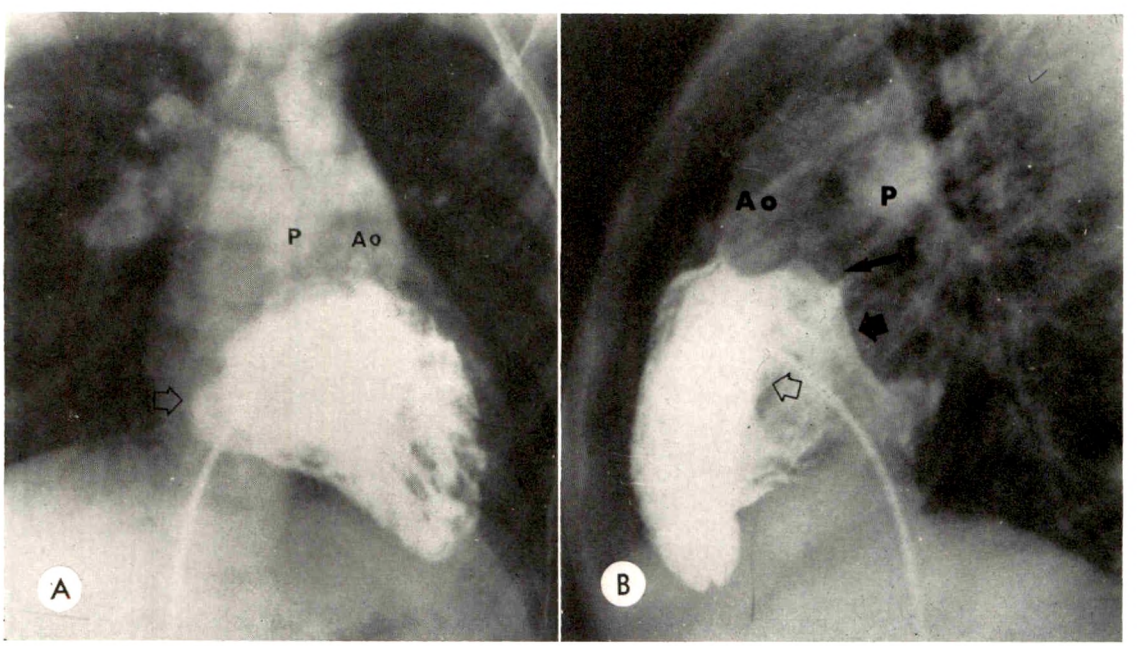


Fig. 15. Selective right ventriculography. (*A*) Frontal and (*B*) lateral views in a case of complete transposition of the great vessels. The left location of the ascending aorta and the medial and posterior position of the pulmonary artery, as seen on the frontal view, may suggest the diagnosis of corrected transposition of the great vessels. However, the continuity of the mitral valve (thick arrow) with the pulmonary valve (thin arrow), as seen on the lateral view, and the noncontinuity of the tricuspid valve (hollow arrow) with the pulmonary valve (thin arrow) excludes this diagnosis. Moreover, the anteriorly high positioned and anteriorly tilted aortic valve, distant from the mitral valve (thick arrow), as seen in the lateral view, and the continuity of the pulmonary valve (thin arrow) with the mitral valve (thick arrow) are diagnostic of complete transposition of the great vessels. CV = Common Ventricle; RA = Right Atrium; LA = Left Atrium; P = Main Pulmonary Artery; Ao = Aorta.

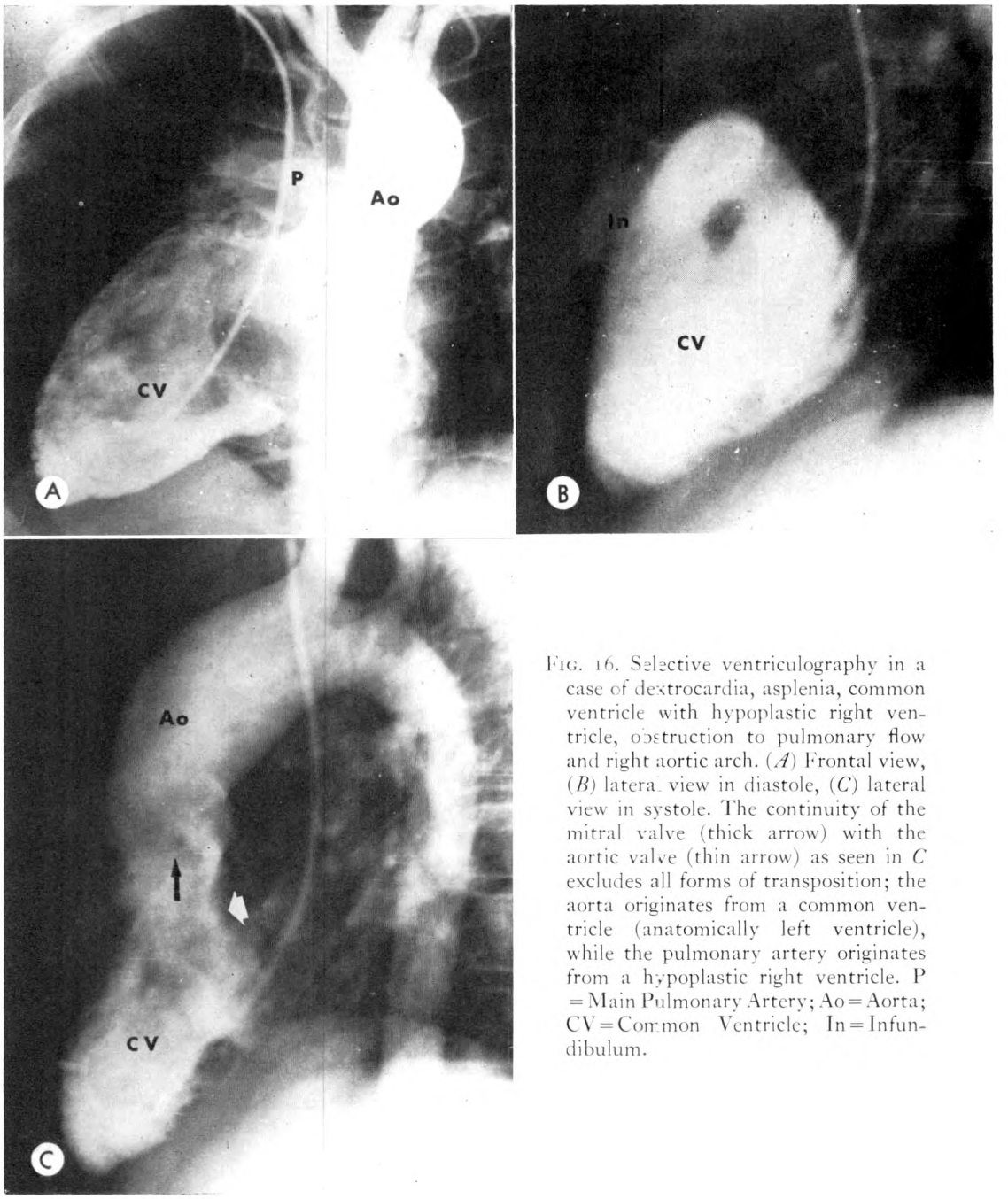


FIG. 16. Selective ventriculography in a case of dextrocardia, asplenia, common ventricle with hypoplastic right ventricle, obstruction to pulmonary flow and right aortic arch. (*A*) Frontal view, (*B*) lateral view in diastole, (*C*) lateral view in systole. The continuity of the mitral valve (thick arrow) with the aortic valve (thin arrow) as seen in *C* excludes all forms of transposition; the aorta originates from a common ventricle (anatomically left ventricle), while the pulmonary artery originates from a hypoplastic right ventricle. P = Main Pulmonary Artery; Ao = Aorta; CV = Common Ventricle; In = Infundibulum.

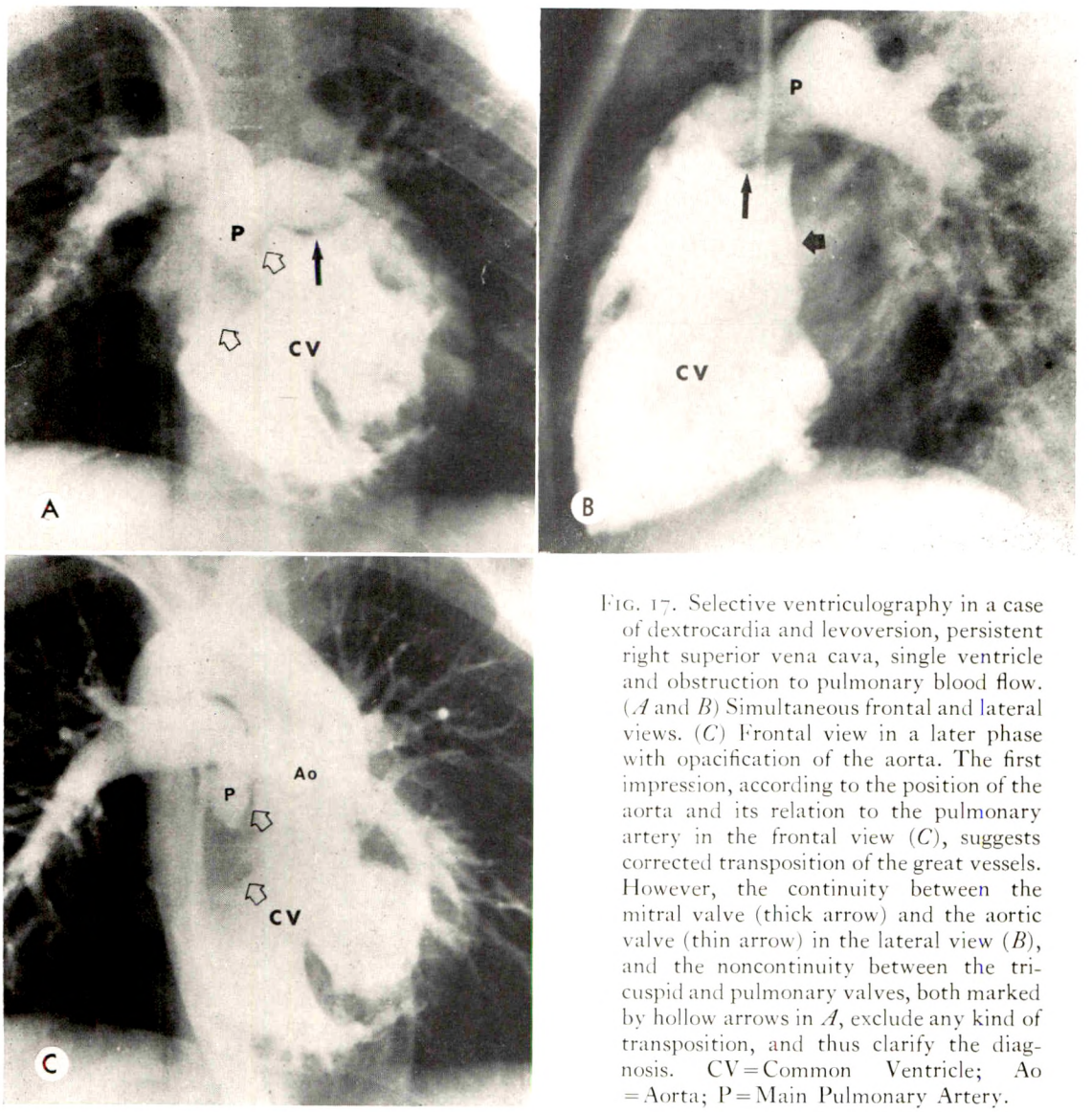


Fig. 17. Selective ventriculography in a case of dextrocardia and levoversion, persistent right superior vena cava, single ventricle and obstruction to pulmonary blood flow. (*A* and *B*) Simultaneous frontal and lateral views. (*C*) Frontal view in a later phase with opacification of the aorta. The first impression, according to the position of the aorta and its relation to the pulmonary artery in the frontal view (*C*), suggests corrected transposition of the great vessels. However, the continuity between the mitral valve (thick arrow) and the aortic valve (thin arrow) in the lateral view (*B*), and the noncontinuity between the tricuspid and pulmonary valves, both marked by hollow arrows in *A*, exclude any kind of transposition, and thus clarify the diagnosis. CV = Common Ventricle; Ao = Aorta; P = Main Pulmonary Artery.

ation of the atrioventricular and semilunar valve relationship may clarify the diagnosis.

Likewise, the left-to-right relationship of the great vessels (the aorta being to the left of the pulmonary artery) may also be confusing, and may lead in some cases to the false diagnosis of corrected transposition (Fig. 15, *A* and *B*). Here, once more, the recognition of the relationship of the atrioventricular to the semilunar valve may constitute the crucial point for the correct diagnosis. In cases of dextrocardia and dextroversion, the evaluation of the relationship of the atrioventricular valves to the semilunar valves is even more important diagnostically (Fig. 16, *A–C*; and 17, *A–C*), since the distorted relationship of the great vessels and heart chambers may be another source of difficulty in diagnosis.

SUMMARY

The anatomopathologic relationships between atrioventricular and semilunar valves are helpful in classifying congenital heart disease. In our experience this relationship

TABLE I

ATRIOVENTRICULAR-SEMILUNAR VALVE RELATIONSHIP IN CONGENITAL HEART DISEASES WITH ANOMALOUS DEVELOPMENT OF THE TRUNCO-CONAL MYOCARDIUM

	Right A-V Valve		Left A-V Valve	
	Pulmonary valve	Aortic valve	Pulmonary valve	Aortic valve
Normal Heart	NC	NC	NC	C
Tetralogy of Fallot	NC	NC	NC	C
Persistent Truncus Arteriosus	—	NC	—	C
Complete Transposition of the Great Vessels	NC	NC	C	NC
Corrected Transposition of the Great Vessels	C	NC	NC	NC
Origin of Both Great Vessels from the Right Ventricle	NC	NC	NC	NC

A-V = Atrio-Ventricular.
NC = No Continuity.
C = Continuity.

presents a specific angiocardiographic aspect in each anomaly involving the development of conal myocardium. This was studied in 35 patients with corrected transposition of the great vessels, 25 with complete transposition, 9 with persistent truncus arteriosus, 6 with origin of both great vessels from the right ventricle and 55 with tetralogy of Fallot for a total of 130 cases. In tetralogy of Fallot and in persistent truncus arteriosus the atrioventricular-semilunar valve relationship was found to be normal. In contradistinction, in corrected transposition there was continuity between the right atrioventricular and the pulmonary valves and noncontinuity on the left side. In complete transposition the left atrioventricular valve was continuous with the pulmonary valve. In origin of both great vessels from the right ventricle no continuity between the atrioventricular and semilunar valves was present.

This approach was particularly helpful in differentiating between borderline cases of transposition complexes with or without dextroversion or dextrocardia, and tetralogy of Fallot from cases with origin of both great vessels from the right ventricle with obstruction to pulmonary flow.

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HYPOPLASIA OF RIGHT VENTRICULAR MYOCARDIUM*

(UHL'S DISEASE)

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UHL, in 1952, described a girl who died at 7 months of age; at necropsy almost total absence of the myocardium of the right ventricle was noted.[11] We have found in the English language literature reports of 9 other patients with a similar abnormality.[1–6, 8–11] This report describes 2 patients with Uhl's disease: one, a girl who died at age 17 months, has been described by Sherman;[9] the other, a boy now 15 years of age, was first seen at the Children's Hospital of Pittsburgh at age 1 month. We believe that he is the first patient in whom intracardiac electrocardiography has helped to establish the diagnosis.

REPORT OF CASES

CASE 1. A girl at age 5 months began to show intermittent cyanosis. Three months later she was admitted to the Children's Hospital of Pittsburgh after she vomited, became cyanotic, and lost consciousness. Blood pressure was 90/60 mm. Hg, respirations were normal, and the liver edge extended 3 cm. below the right costal margin. No murmur was present; her heart sounds were faint. Hemoglobin was 19 gm./100 ml. An electrocardiogram showed right atrial enlargement and small QRS complexes (Fig. 1). In a chest roentgenogram the heart was large and the vascular markings in the lungs were normal (Fig. 2).

She was hospitalized a second time at age 15 months for treatment of congestive heart failure. Clubbing of the fingers was now present. Medical treatment was not effective and she died at age 17 months during an attempt to anastomose the superior vena cava to the right pulmonary artery.

At necropsy the right atrium and right ventricle were dilated. The thin wall of the right ventricle consisted almost entirely of fibrous and elastic tissues (Fig. 3). The myocardium of the left ventricle was normal.

CASE 11. A boy was admitted to the Children's Hospital of Pittsburgh at age 1 month with fever, tachypnea, and cyanosis. Heart sounds were normal. There were no signs of congestive heart failure. An electrocardiogram showed low voltage QRS complexes. In a roentgenogram of the chest the heart and lungs were normal (Fig. 4*A*).

Physical examination when he was 4 months of age showed normal findings; an electrocardiogram at that time showed normal QRS complexes, but right atrial enlargement and T wave abnormalities were present (Fig. 5*A*).

At age 13 years he began to tire easily and to have exertional dyspnea. Pulse rate was 112, blood pressure was 110/80 mm. Hg in the right arm and 160/120 in the right leg. Neck veins were not distended, the liver was not enlarged, peripheral edema and rales were absent. Although heart sounds were faint, the rhythm was normal. Roentgenographic examination of the chest showed enlargement of the heart; the vascular markings in the lungs were normal (Fig. 4, *B* and *C*). An electrocardiogram suggested biatrial enlargement. QRS complexes

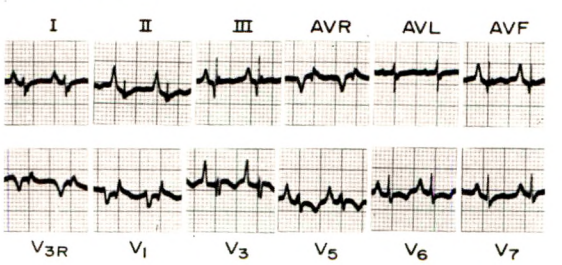


FIG. 1. Case 1. Electrocardiogram at age 8 months. Tall peaked P waves indicate right atrial enlargement. QRS voltage is low and T waves are abnormal.

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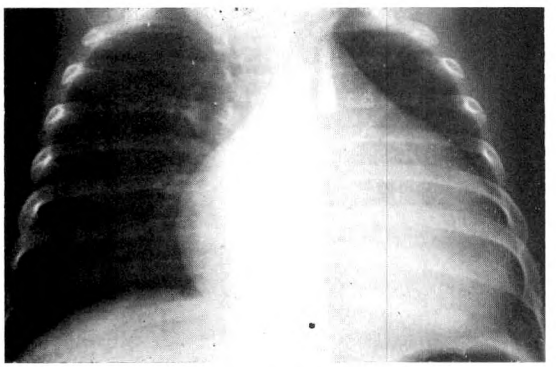


FIG. 2. Case 1. Chest roentgenogram at age 8 months. The heart is large and the vascular markings in the lungs are normal. Barium is in the esophagus and has dripped onto the front of the chest.

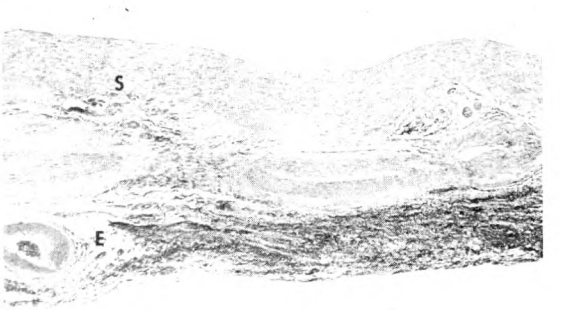


FIG. 3. Case 1. The right ventricle near the apex is devoid of myocardial fibers. The ventricular wall is composed of fibrous scar (S), and epicardium (E) with coronary arteries. Hemorrhage secondary to surgery accounts for the many cells in the epicardium. (Hematoxylin and eosin ×100.)

were small (Fig. 5*B*). Hemoglobin was 15 gm./100 ml.

The data from cardiac catheterization are summarized in Table 1. No shunt was present by oxygen determinations or indicator dilution curves. The elevated mean pressure in the right atrium was almost equal to the mean pressure in the pulmonary artery. Atrial contraction waves were evident in the right ventricular and pulmonary artery pressure tracings. No evidence of an atrialized portion of the right ventricle could be obtained by use of an electrode catheter (Fig. 6). Selective cineangiocardiograms showed the tricuspid valve in a normal position. The wall of the right ventricle was thin; the right ventricle and right atrium were dilated (Fig. 7).

Two years later the electrocardiogram and chest roentgenogram show similar findings. The patient's symptoms have not changed; he continues to tire quickly with moderate activity.

DISCUSSION

The disability in patients with Uhl's disease arises from deficiency of the right ventricular myocardium. Ineffective right ventricular contraction results in elevated right atrial pressure, right atrial enlargement, and signs of congestive heart failure. If the foramen ovale is patent, a right-to-left shunt causes cyanosis. Interposition of the large and flaccid right ventricle between the wall of the chest and the left ventricle, and absence of tricuspid and pulmonic components cause the first and second heart sounds to be poorly heard.

The findings in patients with Uhl's disease reported in the literature are summarized in Table II. Eight infants had symptoms before 5 months of age, and 5 died before 2 years of age.

The patient discussed by Sprague and Castleman[2] died with congestive heart failure at age 25 years; the patient of Reeve and Macdonald[8] died of subarachnoid hemorrhage at the age of 47 years; the patient of Gould and colleagues[5] died of leukemia at 66 years of age.

The abnormalities of the right ventricle which were found at necropsy varied from focal deficiency of right ventricular myocardium in adults to almost total absence in infants.

Although Uhl's disease is not common, the diagnosis should be considered in infants who have congestive heart failure with cyanosis, faint heart sounds, and cardiomegaly, especially if evidence of right atrial enlargement is coupled with low voltage QRS complexes on the electrocardiogram. However, other forms of myocardial disease, pericardial effusion, and Ebstein's disease are more common and may be impossible to distinguish from Uhl's disease clinically. Although in our second patient the shape of the heart in chest roentgenograms resembles that of Ebstein's disease, the faint first and second heart sounds and

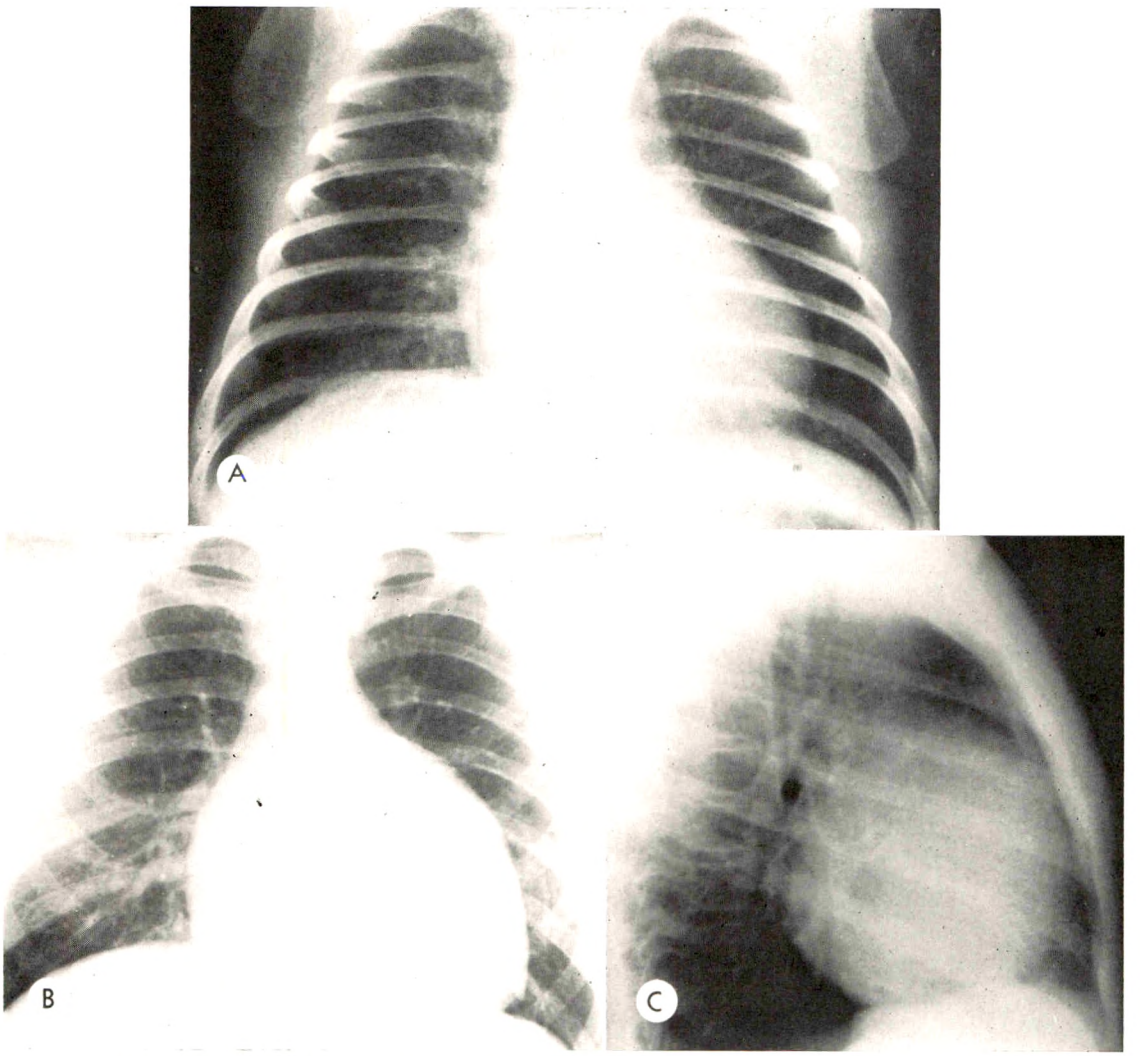


Fig. 4. Case II. (*A*) Chest roentgenogram at age 1 month. The heart and lungs are normal. (*B* and *C*) Chest roentgenograms in frontal and lateral projections at age 13 years. The heart image is enlarged; the vascular markings are normal.

the absence of diastolic sounds are not consistent with that diagnosis.

Since the clinical, electrocardiographic, and roentgenographic findings are not pathognomonic, it is necessary to base the diagnosis on the findings at catheterization and cineangiocardiography. Similar mean pressures in the pulmonary artery and right atrium and a prominent or dominant A wave in the right ventricle and pulmonary artery indicate ineffective contraction of the right ventricle. The angiocardiographic features described by Arcilla and Gasul[1,4] were present in our second patient: normal location of the tricuspid valve, dilatation of the thin-walled right ventricle, and absence of right ventricular trabeculation. Another point of differentiation between Uhl's disease and Ebstein's disease, which was found in our second patient, is absence of atrialization of part of the right ventricle as demonstrated by intracardiac electrocardiography and pressure recordings. However, atrialization of part of the right ventricle may not be demonstrable by this method in some patients with Ebstein's disease.[12]

The cause of Uhl's disease is not known.

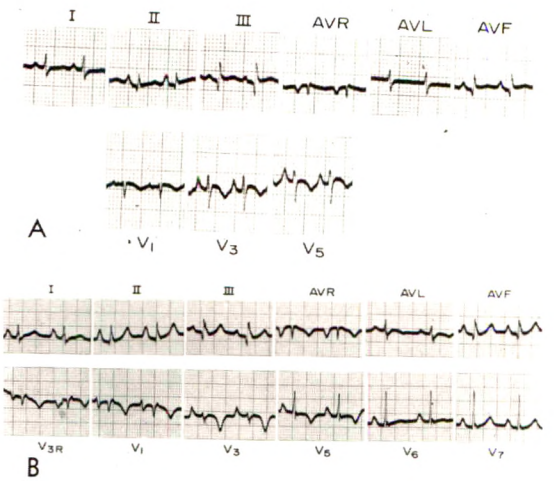


FIG. 5. Case II. (*A*) Electrocardiogram at age 4 months. The tall and peaked P waves indicate right atrial enlargement. The deep T wave inversion in lead V_5 is probably abnormal. (*B*) Electrocardiogram at age 13 years. The P waves are tall, peaked and widened and indicate biatrial enlargement. The QRS complexes are small.

Infantile and adult cases may result from different causes. The presence of focal disease is compatible with survival beyond infancy; extensive absence of right ventricular myocardium results in early death.

Treatment has been unsatisfactory although some patients have improved with digitalis. Two infants did not survive the attempt to anastomose the superior vena cava to the right pulmonary artery. However, this operation is known to be hazardous in infants; perhaps it would be beneficial in older patients whose symptoms justify surgical operation. Two infants with Uhl's disease, who were part of a larger group of patients treated with balloon atrial septostomy, did not survive the Waterston shunt done after the septostomy.[7]

TABLE I

MEASUREMENTS OF OXYGEN SATURATION AND PRESSURE WITH THE CATHETER TIP IN SUPERIOR VENA CAVA (SV), RIGHT ATRIUM (RA), RIGHT VENTRICLE (RV), PULMONARY ARTERY (PA), AND RIGHT BRACHIAL ARTERY (RBA)

	Per Cent Saturation (O_2)	Pressure (mm. Hg)
SVC	74	
RA	76	(13)
RV	76	20/9
PA	77	20/9(15)
RBA	97	120/55(75)

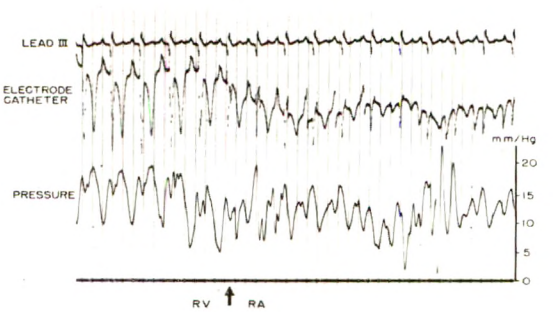


FIG. 6. Case II. Pressure and electrode catheter tracings. The pressures in the right ventricle and right atrium are of similar amplitude but a dominant A wave is present in the right atrium. The intracardiac electrocardiogram shows no evidence of an atrialized portion of the right ventricle.

SUMMARY

Two children with hypoplasia of the myocardium of the right ventricle are re-

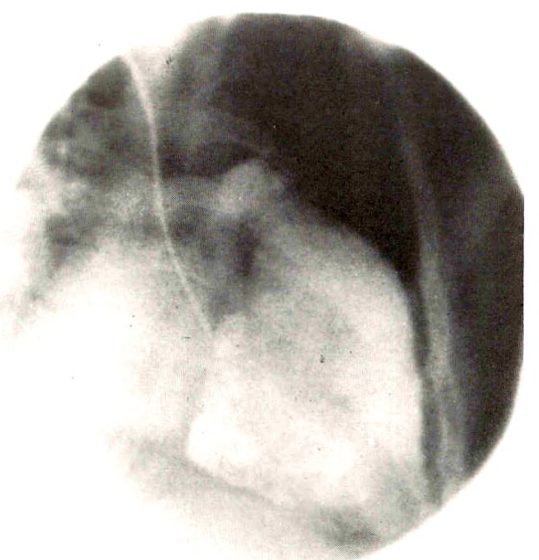


FIG. 7. Case II. Right ventricular cineangiocardiogram. The right ventricular cavity is large and untrabeculated. The wall is thin.

TABLE II

UHL'S DISEASE

Author	Date Reported	Age at Onset of Symptoms	Age at Death	Clinical Findings					Electrocardiogram		Chest Roentgenogram	Necropsy		Comments
				Cyanosis	Gallop	Heart Sounds	Murmur	CHF	RAE	QRS Voltage		Right Ventricle	Left Ventricle	
1. Uhl[11]	1952	4 mo.	7 mo.	+	+	Faint	0	+	+	Low	Cardiomegaly	Nearly absent	Normal	
2. Sprague and Castleman[2]	1952	23 yr.	25 yr.	—	+	—	Faint systolic	+	0	Low	Cardiomegaly	Very thin	Normal	
3. Arcilla and Gasul[1]	1961	5 mo.	1 yr.	0	+	Faint	Short soft systolic	+	+	Normal	Cardiomegaly	Nearly absent	Hypertrophy and dilatation; endocardial fibroelastosis	
4. Sherman[9] (Present Case 1)	1963	5 mo.	17 mo.	+	0	Faint	0	+	+	Low	Cardiomegaly	Nearly absent	Normal	Superior vena cava to pulmonary artery anastomosis
5. Reeve and Macdonald[8]	1964	Adol.	47 yr.	+	—	—	Faint systolic	? During pregnancy	+	Low	Cardiomegaly	Two areas of deficient myocardium	Focal fibrosis	Died of CVA; had episodes of paroxysmal tachycardia
6. Cumming *et al.*[3]	1965	3 mo.	7 mo.	+	+	Faint	—	+	+	Low	Cardiomegaly	Nearly absent	Normal	Superior vena cava to pulmonary artery anastomosis
7. Perrin and Mehrizi[6] (Case 1)	1965	4 mo.	10 mo.	+	+	Faint	Faint systolic	+	+	Normal	Cardiomegaly	Anterior wall thin; normal conus	Focal myocytolysis	Two siblings died of heart disease, type unknown
8. Perrin and Mehrizi[6] (Case 2)	1965	Birth	6 yr.	+	—	—	Continuous	+	0	Normal	Cardiomegaly	Nearly absent	Slight endocardial fibroelastosis	Aortico-pulmonary anastomosis, later congestive heart failure
9. Gasul *et al.*[4]	1966	5½ mo.	Living; age 2 yr.	—	+	Faint	—	+	+	—	Cardiomegaly	Living		
10. Gould *et al.*[5]	1967	None	66 yr.	0	0	Normal	0	0	0	Normal	Normal	Two areas of deficient myocardium	Hypertrophy	Died of leukemia
11. Present report (Case 11)		1 mo.	Living; age 15 yr.	Transient in infancy	0	Normal early, now faint	0	0	+	Low	Cardiomegaly	Living		

ported. One, a girl, died at the age of 17 months, and the necropsy findings are described. In the other, a boy now 15 years of age, who was first seen at the age of 1 month, the diagnosis was made by means of cardiac catheterization, cineangiocardiography, and intracardiac electrocardiography.

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VENTRICULAR SEPTAL DEFECT IN TETRALOGY OF FALLOT*

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OBSTRUCTION of the pulmonary circulation and ventricular septal defect are considered to be the 2 essential elements determining the clinical and hemodynamic patterns in tetralogy of Fallot.[4,9] Total corrections are now being performed in all cases of the tetralogy except for cases with severe peripheral pulmonary stenosis. Consequently, it is very important to evaluate, before operation, these 2 elements not only for the selection of the mode of right ventriculotomy but also for the determination of the prognosis after operation.

The purpose of this paper is to study angiographically the ventricular septal defect in tetralogy of Fallot.

MATERIAL AND METHOD

Sixty patients with tetralogy of Fallot were examined by angiocardiography. All patients underwent total correction in the Department of Surgery in Osaka University Hospital. The series comprised 40 males and 20 females. Their ages ranged from 5 months to 22 years. Angiocardiography was performed by injecting sodium iothalamate into the right ventricle with an NIH type catheter, which was, as a rule, inserted through the saphenous vein. Simultaneous anteroposterior and lateral views were obtained at the speed of 6 films per second for the first 2 seconds, 4 films per second for the following 1 second and 2 films per second for the last 2 seconds. Simultaneous electrocardiography was recorded. In 12 cases, thoracic aortography and/or left heart angiography were performed.

ANATOMY

The intracardiac landmarks, which define the location of the ventricular septal defect, are the crista supraventricularis and the pulmonary valve from the right ventricular aspect. In the usual type of tetralogy of Fallot, the ventricular septal defect is situated in the anterior portion of the muscular ventricular septum, anterior to the pars membranacea. On the right ventricular aspect, the defect is localized in the lower part of the conus beneath the crista supraventricularis. It involves the conal septum to varying degrees, from the tricuspid valve to the region posterior to the crista supraventricularis. The parietal band of the crista supraventricularis, which is displaced anteriorly in the tetralogy, separates the ventricular septal defect from the right ventricular infundibulum. The aorta straddles the ventricular septal defect and arises from both ventricles. On the left ventricular aspect, the defect lies inferior to the right aortic cusp.

Infrequently, the defect may involve the subpulmonary portion of the conal septum, anterosuperiorly to the crista supraventricularis. In this type of tetralogy, the defect lies immediately beneath the pulmonary valve and is remote from the tricuspid valve. The defect is, on the left ventricular aspect, localized inferior to the commissure between the right and left aortic cusps and remote from the anterior leaflet of the mitral valve. There is close relationship between the pulmonary and aortic valves through the ventricular septal defect in this type of tetralogy, because there is no interposed subpulmonary conal septal musculature to divide them. The crista supraventricularis is not seen between the 2 ventricular outflow tracts.

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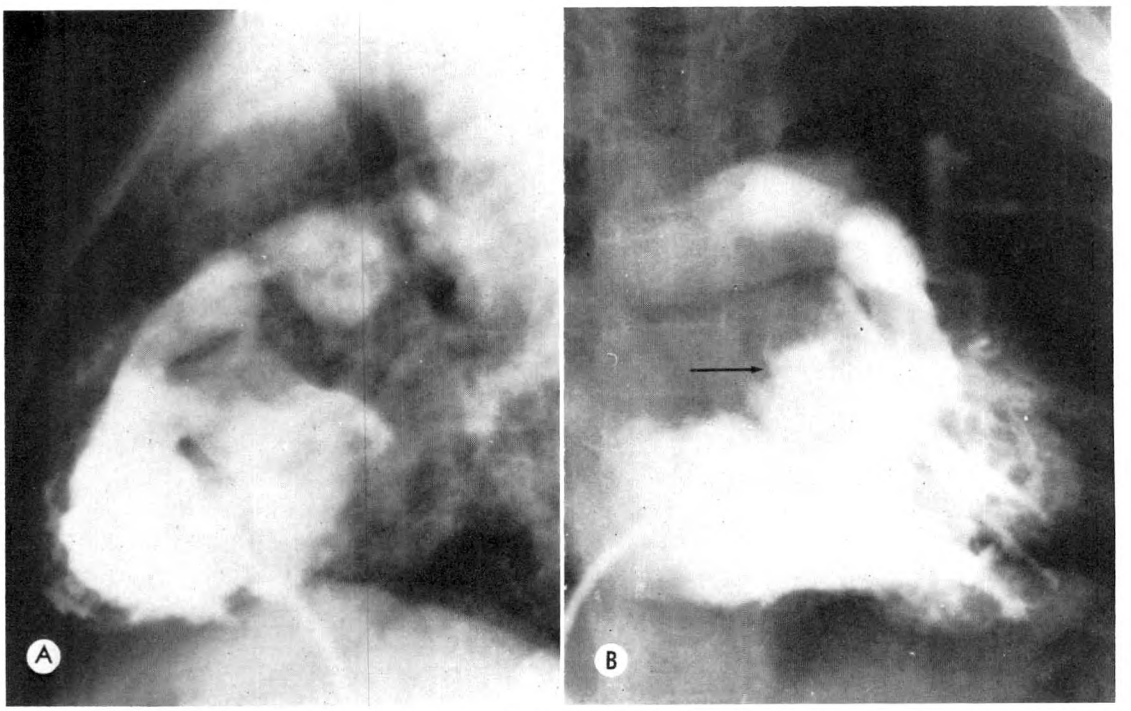


FIG. 1. Tetralogy of Fallot with infracristal ventricular septal defect in a boy aged 5 years. Right ventriculograms. (*A*) Lateral view. Mild infundibular stenosis and severe pulmonary valvular stenosis are noted. Ventricular septal defect is demonstrated beneath the crista supraventricularis. The right aortic cusp is shown above the defect as a sharply defined radiolucency. (*B*) Anteroposterior view. The ventricular septal defect is noted as an increased density beneath the right aortic cusp (arrow).

RESULTS

The ventricular septal defect is demonstrated during the early ventricular ejection phase and also during the ventricular relaxation phase. In the latter phase, the contrast medium ejected via the defect fills the left ventricular sinus instead of passing directly into the aorta, which occurs during the former phase.

Forty-six patients (77 per cent) demonstrated the infracristal ventricular septal defect and the remaining 14 patients (23 per cent) the subpulmonary (supracristal) ventricular septal defect.

INFRACRISTAL VENTRICULAR SEPTAL DEFECT

In the lateral view, the ventricular septal defect was shown to be in the inferior portion of the right ventricular infundibulum, being rather remote from the pulmonary valve, and was cranially demarcated by the crista supraventricularis (Fig. 1*A*; and 2*A*). In the lateral view, the crista separating the aortic root and outflow tract of the right ventricle was demonstrated in 37 of 46 cases, while, in the anteroposterior view, this was demonstrated only in 25 cases.

Both ventricular outflow tracts were superimposed in 7 cases; this obscured the presence of the crista in the lateral view.

In the remaining 2 cases, the contrast medium was so diluted that the crista was not visualized.

On the left ventricular aspect, the location of the ventricular septal defect could be found in relation to the right aortic cusp. The defect was noted below the level of the right aortic cusp. No defect was demonstrated above the level of the right aortic cusp. In the anteroposterior view, the ventricular septal defect was noted as a small area of increased density just beneath the right aortic cusp during the ventricular relaxation phase in 17 cases, in which the

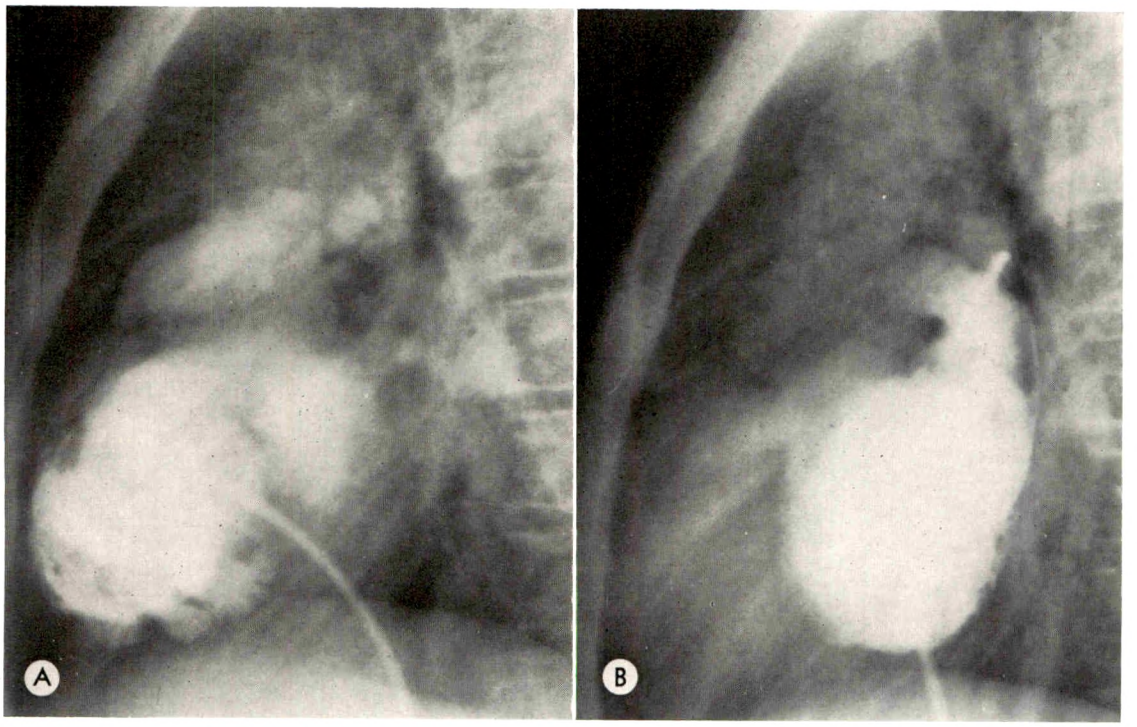


Fig. 2. Tetralogy of Fallot with infracristal ventricular septal defect in a boy aged 11 years. (*A*) Lateral view of right ventriculogram. The ventricular septal defect is noted beneath the crista supraventricularis and right aortic cusp. Marked infundibular stenosis is demonstrated. (*B*) Lateral view of left heart angiogram. The ventricular septal defect is shown just beneath the right aortic cusp. Contrast medium fills the right ventricular sinus.

defect was also shown in the lateral view (Fig. 1*B*). During the early ventricular ejection phase, the contrast medium crossed the defect and was demonstrated from beneath the right aortic cusp passing into the aorta.

The posterior wall of the upper portion (*i.e.*, subpulmonary region) of the right ventricular infundibulum was clearly outlined in 36 cases. This indicated the absence of the defect in the subpulmonary region.

Left heart angiography was performed in 10 cases. The ventricular septal defect was shown to be beneath the right aortic cusp. The contrast medium crossed the defect beneath the right aortic cusp from the left ventricle, and filled the right ventricular sinus and the outflow tract (Fig. 2*B*).

Subpulmonary Ventricular Septal Defect

The crista supraventricularis was not demonstrated in either the anteroposterior or the lateral view. The right ventricular infundibulum was largely superimposed by the aortic root and the left ventricular outflow tract in the lateral view. Both structures were very close in the anteroposterior view. The ventricular septal defect was shown to be located in the upper part of the right ventricular infundibulum and to be close to the pulmonary valve (Fig. 3*A*). The posterior wall of the subpulmonary region of the right ventricular infundibulum was consequently obscured. The upper outline of the ventricular septal defect was not clearly seen in the lateral view, while the lower outline was occasionally well defined. The defect was demonstrated on other occasions beneath the commissure between the right and left aortic cusps as an increased density during the ventricular relaxation phase in the anteroposterior view (Fig. 3*B*). During the ejection phase,

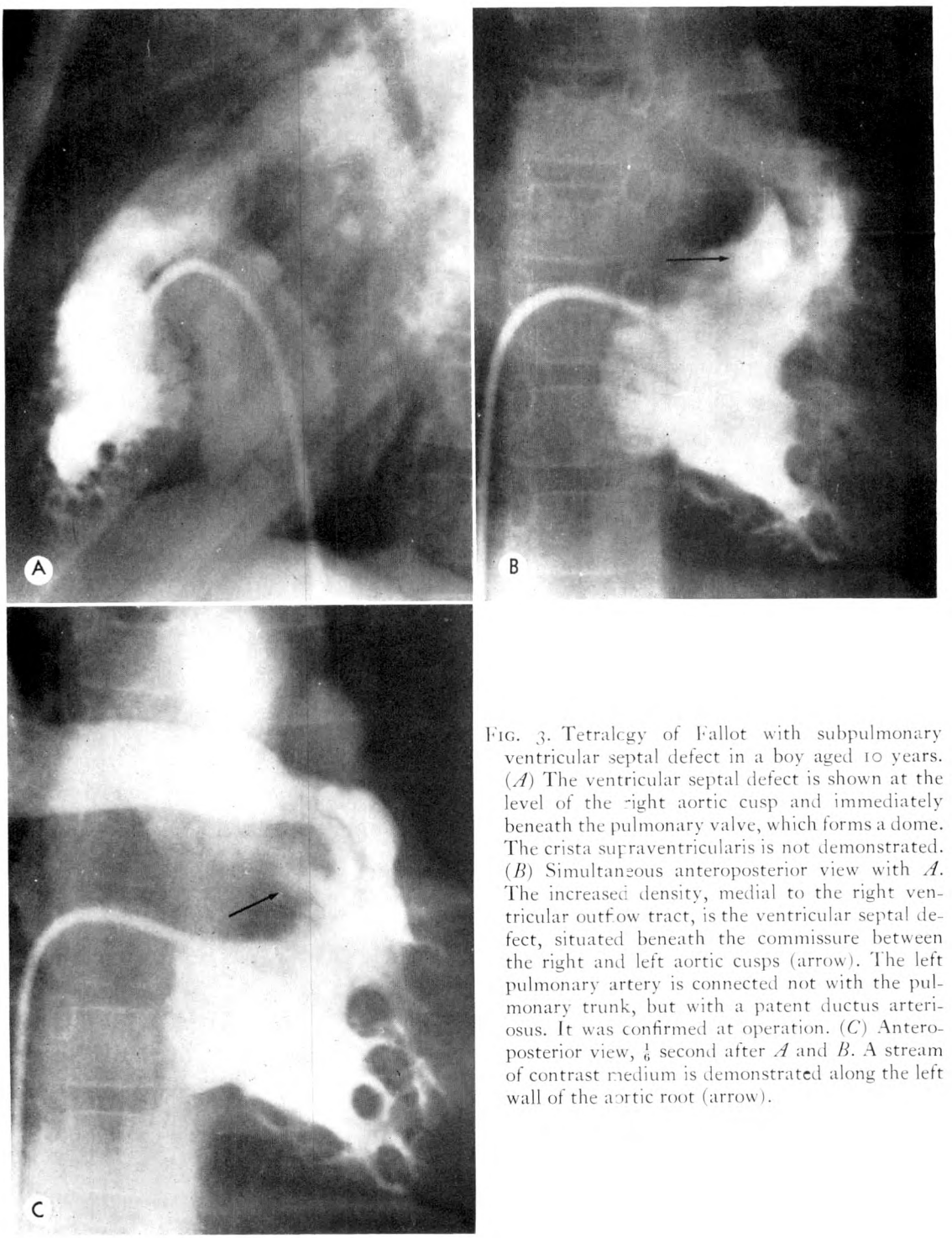


FIG. 3. Tetralogy of Fallot with subpulmonary ventricular septal defect in a boy aged 10 years. (*A*) The ventricular septal defect is shown at the level of the right aortic cusp and immediately beneath the pulmonary valve, which forms a dome. The crista supraventricularis is not demonstrated. (*B*) Simultaneous anteroposterior view with *A*. The increased density, medial to the right ventricular outflow tract, is the ventricular septal defect, situated beneath the commissure between the right and left aortic cusps (arrow). The left pulmonary artery is connected not with the pulmonary trunk, but with a patent ductus arteriosus. It was confirmed at operation. (*C*) Anteroposterior view, $\frac{1}{6}$ second after *A* and *B*. A stream of contrast medium is demonstrated along the left wall of the aortic root (arrow).

the contrast medium was seen as a stream along the left wall of the aortic root (Fig. 3*C*).

Two cases underwent left heart angiography in addition to right ventriculography. In 1 case, the right ventricular infundibulum was narrowed by the right aortic cusp, which protruded through the subpulmonary defect (Fig. 4*A*). The crista was not demonstrated in both projections and the defect was shown in the subpulmonary region at the level of the right aortic cusp. In left ventriculography, the herniated right aortic cusp partially filled the defect. The contrast medium was ejected into the right ventricular outflow tract via the unfilled defect (Fig. 4*B*). The thoracic aortogram demonstrated regurgitation of the contrast medium into the left ventricle (Fig. 4*C*). In the other case, the right ventricular sinus was visualized in the left ventriculogram and the defect was demonstrated at the level of the aortic cusp.

DISCUSSION

The ventricular septal defects related to the ventricular outflow tract are classified into 2 groups: The defects involving the region posterior to the crista supraventricularis, and those anterior to the crista. The incidence of the latter defects is about 5–8 per cent of all isolated ventricular septal defects.[2,6,11] Baron *et al.*[1] reported the angiographic findings in the diagnosis of the subpulmonary ventricular septal defect. They stressed the value of left ventriculography to differentiate the 2 types of defects in relation to the aortic cusps and the direction of the flow of the contrast medium. In respect to the anatomy of the ventricular septum, tetralogy of Fallot is included in the spectrum of the isolated ventricular septal defects.[14] The exact incidence of the subpulmonary ventricular septal defect in tetralogy of Fallot is still unknown. In patients with the tetralogy, right ventriculography is the method of choice to determine the anatomy of the right ventricular outflow tract as well as the ventricular septal defect. It is necessary, therefore, to find out the features serving to differentiate the 2 types of the defect in the right ventriculograms.

In tetralogy of Fallot, the crista supraventricularis is well developed and its parietal band is deviated anteriorly. Therefore, the crista is better visualized than in the isolated ventricular septal defect. Furthermore, the right ventricular hypertrophy in the tetralogy tends to rotate the heart clockwise around the longitudinal axis. This clockwise rotation moves the ventricular septum parallel to the frontal plane. Consequently, the ventricular septum is definitely demonstrated as a radiolucent band separating the 2 ventricles in the lateral view and the defect can be well shown in most of the cases. In addition the right-to-left shunt in the tetralogy also visualizes the left ventricular outflow structure. In cases of the infracristal ventricular septal defect without superimposition of the ventricular outflow tracts, the crista can be found as a tongue-like shadow separating the ventricular outflow tracts either in the anteroposterior view or in the lateral view. If the crista is definitely demonstrated above the ventricular septal defect, the diagnosis of the infracristal defect can be established without difficulty. On the contrary, the crista is underdeveloped and not found in cases of the subpulmonary defect. When the crista is not demonstrated, difficulty arises in the differentiation of the 2 types of the defect. The infracristal defect is usually demonstrated beneath the right aortic cusp in the lateral view. Close relationship of the defect with the pulmonary valve in such cases is indicative of the subpulmonary defect. Obscuring of the posterior wall of the subpulmonary region results from high position of the defect. The contrast medium from the right ventricle visualizes the aortic cusps, which is helpful to define the anatomic features of the defect, the crista supraventricularis and the pulmonary valve. Right-to-left shunt occurs in the early ventricular ejection phase and the isovolumic relaxation phase.[8] The early ejection phase is more suitable than

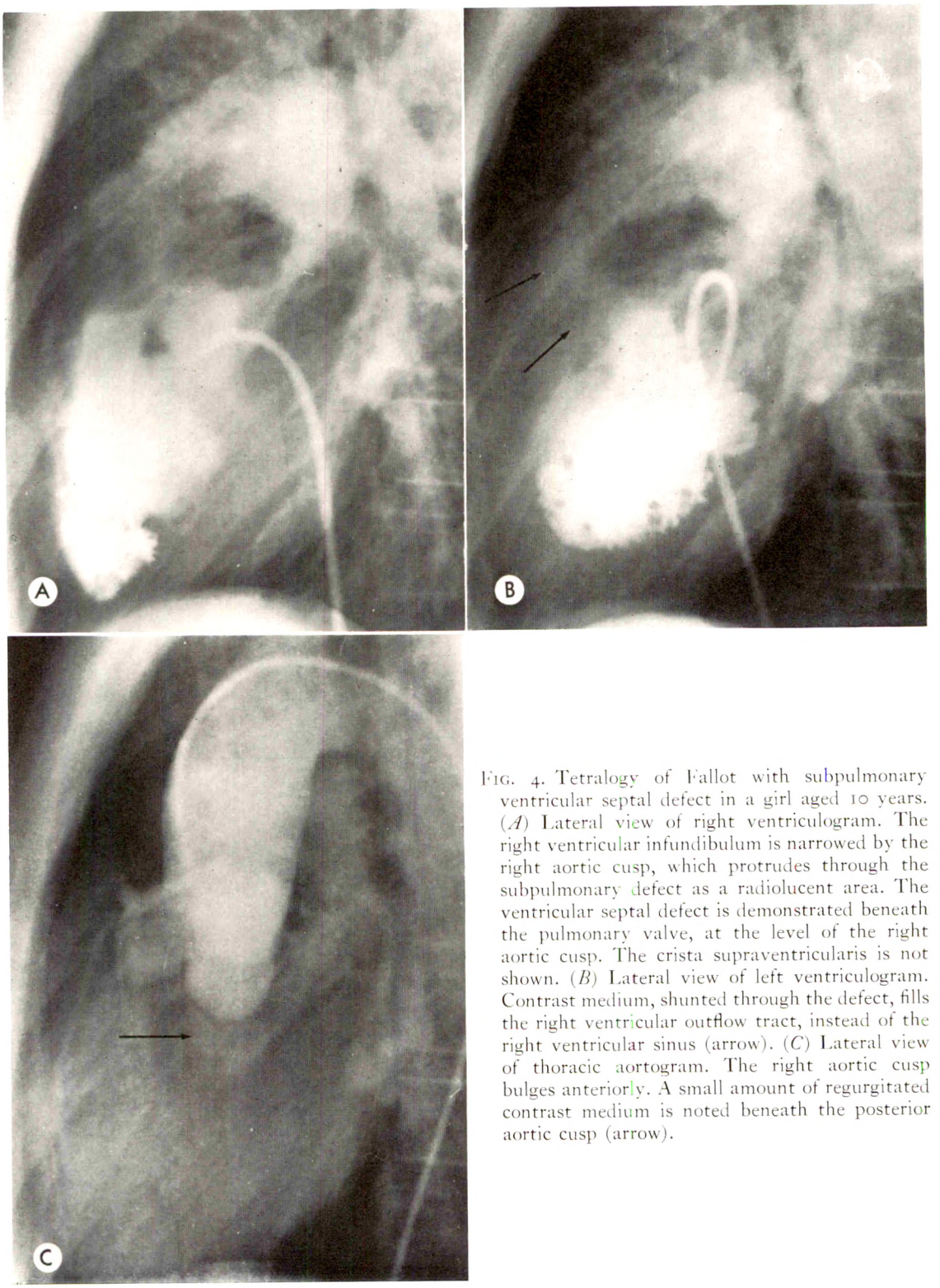


FIG. 4. Tetralogy of Fallot with subpulmonary ventricular septal defect in a girl aged 10 years. (*A*) Lateral view of right ventriculogram. The right ventricular infundibulum is narrowed by the right aortic cusp, which protrudes through the subpulmonary defect as a radiolucent area. The ventricular septal defect is demonstrated beneath the pulmonary valve, at the level of the right aortic cusp. The crista supraventricularis is not shown. (*B*) Lateral view of left ventriculogram. Contrast medium, shunted through the defect, fills the right ventricular outflow tract, instead of the right ventricular sinus (arrow). (*C*) Lateral view of thoracic aortogram. The right aortic cusp bulges anteriorly. A small amount of regurgitated contrast medium is noted beneath the posterior aortic cusp (arrow).

the relaxation phase to clarify the relationship between the aortic cusps and the defect in the anteroposterior view. In the former phase, the contrast medium is ejected from the right ventricle directly into the aorta via the defect. While the contrast medium tends to flow in the center of the aortic root in cases of infracristal defect, it flows along the left wall of the aorta. This indicates a subpulmonary defect, in which the defect is situated beneath the commissure between the right and left aortic cusps. During the relaxation phase, however, the adjacent structures are rather well delineated in the lateral view, while the precise position of the defect is difficult to locate in the anteroposterior view.

Twelve cases underwent left heart angiography. In cases of the infracristal defect, the contrast medium filled the right ventricular sinus. In 2 cases of the subpulmonary defect, however, 1 case showed filling of the ventricular sinus, which is indicative of an infracristal defect. In the other case, the contrast medium filled only the right ventricular infundibulum through the subpulmonary defect, where the right aortic cusp bulged. Thus, filling of the right ventricular sinus in left ventriculography may not be a reliable sign as reported by Baron *et al.*[1] in the isolated ventricular septal defect. Whether the defect is localized beneath or at the level of the right aortic cusp should be cautiously studied in the lateral view. In tetralogy of Fallot, left ventriculography gives complementary findings in determining the location of the ventricular septal defect.

The high ventricular septal defect in tetralogy of Fallot may cause a herniation of the right or posterior aortic cusp, resulting in aortic regurgitation and a pressure gradient between the main pulmonary artery and the body of the right ventricle.[7,10,13] In thoracic aortography, if the right aortic cusp is involved in the isolated ventricular septal defect, a step-like bulge can be seen between the dilated upper part of the right aortic cusp and the ascending aorta.[3] Similarly, aortic regurgitation may result from malformations of the aortic valve in patients with tetralogy.[5] Thoracic aortography demonstrates clearly the anatomy of the aortic branches and the systemic arterial supply to the lungs as reported by Rees and Somerville.[12] Furthermore, it serves to delineate aortic regurgitation caused by herniation or malformation of the aortic cusps.

SUMMARY

The ventricular septal defect in 60 cases of tetralogy of Fallot was examined by right ventriculography. Left heart angiography was performed in 12 cases.

The infracristal defect was demonstrated in 45 cases and the subpulmonary defect in 15 cases.

The diagnosis of infracristal ventricular septal defect is established without difficulty, when the crista supraventricularis forms the upper outline of the defect in the lateral view, and when the crista separates the 2 ventricular outflow tracts as a tongue-like radiolucency in the anteroposterior view. In cases where the crista is not clearly shown, it is necessary to examine the defect in relation to the aortic cusps and pulmonary valve.

Left heart angiography gives the complementary findings in determining the location of the ventricular septal defect.

One case of subpulmonary defect is reported in which herniation of the right aortic cusp partially filled the defect.

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CONGENITAL ABSENCE OF THE PULMONARY VALVE IN TETRALOGY OF FALLOT AND ORIGIN OF THE LEFT PULMONARY ARTERY FROM THE AORTIC ARCH*

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TETRALOGY of Fallot complicated by the absence of the pulmonary valve is a rare condition. Reviewing the literature from 1908 to 1964, Ruttenberg and co-workers commented on 25 cases.[6,7] From 1964 to 1969 we found another 15 observations.[1,3,6]

The purpose of this communication is to describe an additional case which has the associated anomaly of the left pulmonary artery originating from the aorta. This complex malformation has—to our knowledge—not been reported previously.

REPORT OF A CASE

A 2 day old male infant was admitted to our hospital because of cyanosis and a roentgenologically prominent paracardial mass on the right. The pregnancy and delivery were uneventful; the birth weight was 2,620 gm. On admission the newborn showed marked cyanosis; the respiratory rate was slightly elevated; the liver was not enlarged; and no signs of congestive failure were present. The right ventricle was hyperactive. On auscultation the first sound was normal; an early systolic ejection click was present over the lower left sternal border; the second sound was loud and no splitting was detectable. A high-pitched Grade 3 pansystolic ejection murmur and a loud Grade 2 early to mid-diastolic murmur were heard over the left lower sternal border, giving the impression of a to-and-fro type murmur.

Roentgenograms of the chest (Fig. 1, *A* and *B*) showed a moderately enlarged heart, a tumor-like bulge at the right hilar region and diminished peripheral vascular markings. The left upper cardiac border was somewhat prominent.

Phonocardiogram (Fig. 2) confirmed the findings of auscultation.

Electrocardiogram showed a frontal axis of 190°, sinus rhythm and right ventricular hypertrophy which was more than usual for the normal newborn.

Cardiac catheterization (Table 1) performed on the second hospital day revealed a normal pressure in the right atrium and a normal right ventricular end-diastolic pressure. The systolic pressure in the right ventricle was at systemic level. The pulmonary artery was not entered; aortic pressure, however, was normal. There was a peripheral arterial desaturation of 67 per cent.

Angiocardiography. Cineangiocardiography from injection into the right ventricle (Fig. 3, *A* and *B*) shows a well-trabeculated chamber with a markedly hypertrophic crista supraventricularis. The orifice of the infundibulum is very narrow and the pulmonary artery shows considerable stenosis. There is simultaneous filling of a dextroposed ascending aorta through a ventricular septal defect. The right pulmonary artery is aneurysmally dilated, and its peripheral branches seem small. No left pulmonary artery is seen. There is diastolic regurgitation of contrast material from the pulmonary artery into the right ventricle.

Aortography by retrograde umbilical arterial catheterization (Fig. 4, *A* and *B*) reveals a slightly dilated ascending aorta. At the site of the ductus Botalli a vessel originates from the aortic arch and supplies the left lung. This vessel has approximately half the diameter of the descending aorta.

Diagnosis of tetralogy of Fallot, absent pulmonary valve and origin of the left pulmonary artery from the aortic arch was made.

Course. The child did fairly well, no heart failure or severe hypoxia developed and the patient could be discharged 10 days later. At a control in the outpatient clinic 2 months later

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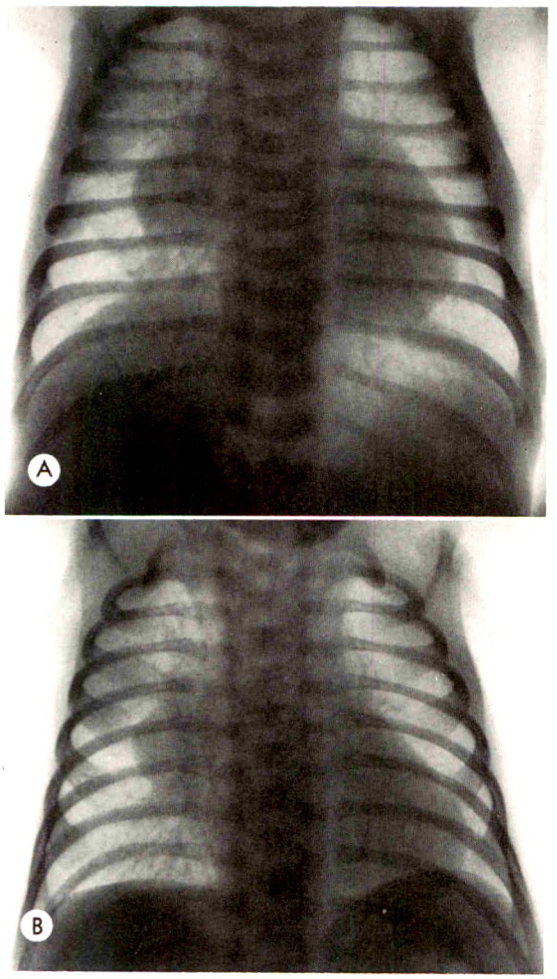


FIG. 1. (*A* and *B*) Chest roentgenograms on Day 2 and Day 14 at different respiratory phases. In *A* the "tumor" like bulge on the right is seen lateral to an opacity identified later (Fig. 4) as the ascending aorta. Here and in *B* also note the prominence of the left upper cardiac border. The peripheral pulmonary vasculature is noticeably diminished throughout.

the child was noted to have virtually no more cyanosis. The other physical findings were unchanged and the general condition was good.

DISCUSSION

The clinical appearance of this patient with a loud to-and-fro murmur at the lower left sternal border and moderate cyanosis already suggests the diagnosis of pulmonary insufficiency in tetralogy of Fallot. This impression is further supported by the roentgenologic features; *i.e.*, cardiomegaly, marked dilatation of the pulmonary artery and diminished peripheral vascularity. A prominent bulge at the upper left cardiac border (the dilated right ventricular outflow tract) is another helpful sign for roentgenologic identification of this lesion.[6,7] The definite confirmation of an absent pulmonary valve can only be obtained by surgery or necropsy. The diagnosis, however, based on the presented data can be presumed to be correct since full correlation between auscultatory, angiocardiographic and anatomic findings was established.[1,2,7]

In tetralogy of Fallot with an absent pulmonary artery on one side, this anomaly always occurred on the left.[4] This phenomenon was explained by the fact that the defect of the bulbus cordis affects not only the infundibulum but may also influence the development of the main and left pulmonary arteries. In cases with an absent left pulmonary artery the blood supply to

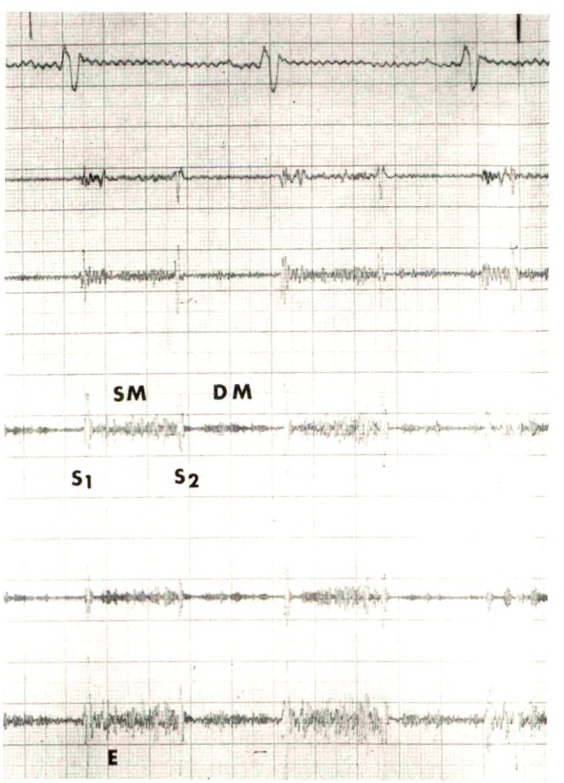


FIG. 2. Phonocardiogram on Day 2, taken at the lower left sternal border. Top: ECG-Lead II. S_1 = first sound; S_2 = second sound; SM = systolic murmur; DM = diastolic murmur; E = ejection click (seen best in high frequencies and auditory range).

Table I

CATHETERIZATION DATA

Vessel	Saturations (per cent)	Pressures (mm. Hg)
SVC	67	—
RA	67	a=6, m=1
RV	71	66/5
Ao. asc.	67	72/48, m=56

SVC=superior vena cava; RA=right atrium; RV=right ventricle; Ao. asc.=ascending aorta.
a=average; m=mean.

the affected lung was generally through bronchial arteries or a vessel arising from the aortic arch. Embryologically, such a vessel represents the persistent dorsal and ductal-like portion of the pulmonary arch. A blood supply from the ascending aorta was found only in the cases with an absent right pulmonary artery.[3,4]

Based on the previous reports on tetralogy with absent pulmonary valve, there seems to be a poor prognosis. Hypoxia and more commonly intractable right heart failure as a result of massive pulmonary valve insufficiency were the causes of death.[1,6,7] Respiratory distress from tracheobronchial obstruction caused by the dilated pulmonary artery and its branches has also been reported.[1,6] Surgical closure of the ventricular septal defect and removal of the infundibular obstruction, however, was performed successfully, and the remaining pulmonary valve insufficiency seemed not to have detrimental effect on these cases.[2,7] Anastomosis of the superior vena cava to the right pulmonary artery was recently reported to have relieved the respiratory tract obstruction in 1 infant.[6] In our patient, the present good clinical condition seemed not to warrant any surgical intervention. The mentioned problems in the course of tetralogy and pulmonary insufficiency are further complicated by the additional anomaly in our case. This ductal type vessel supplying the left lung seemed rather small as did its branches. It may be of some importance, however, that angiography was performed on the fourth day of life when the pulmonary vascular resistance might still have been elevated.[5] In fact, the falling resistance to flow to the left lung might very well have caused the disappearance of

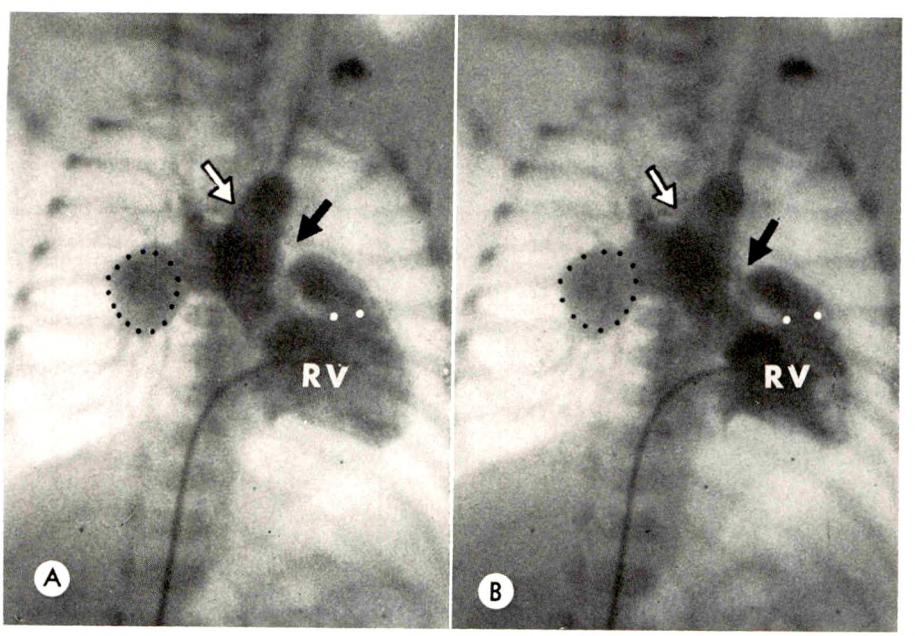


Fig. 3. (*A* and *B*) Cinecardiography on Day 3. Tip of catheter in right ventricle (RV). Beside the details given in text note narrowing of right ventricular outflow tract (. .) and pulmonic stenosis (⬟); on the right the aneurysmally dilated pulmonary artery ◌. ⬠=dextroposed ascending aorta.

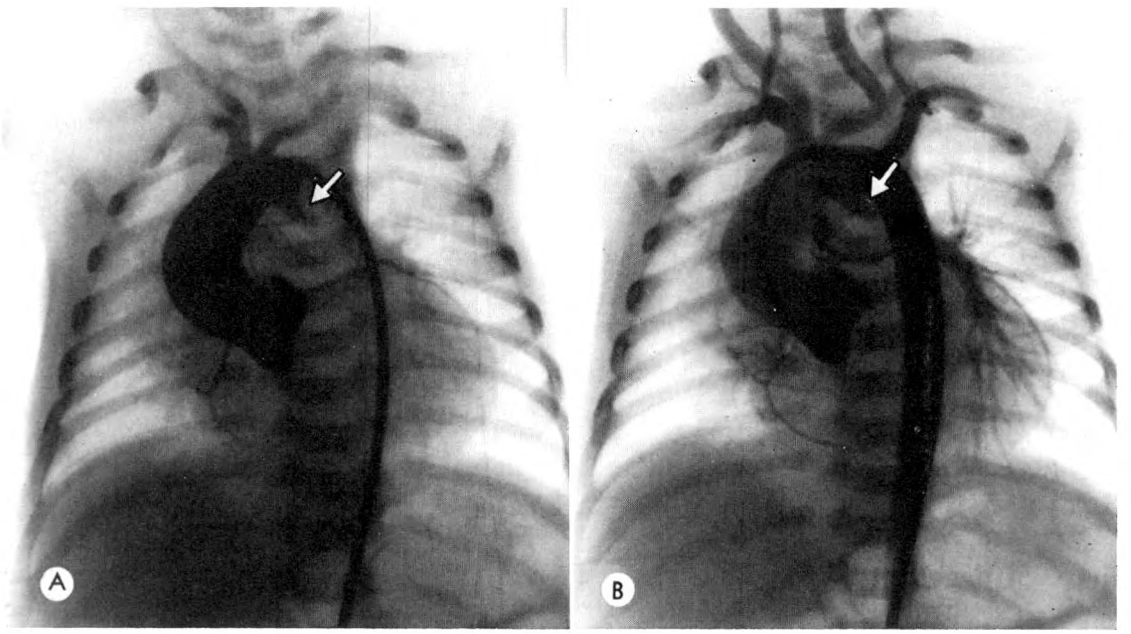


FIG. 4. (*A* and *B*) Transumbilical aortogram on Day 4 of life. (*A*) Early phase. Opacification of a dilated ascending aorta and both coronary arteries. Note the filling of a "ductus" like structure (⬋). (*B*) Through the latter, there is in this later phase filling of the entire left pulmonary artery.

cyanosis in our patient at follow-up several weeks later.

This increasing aorticopulmonary shunt may result in left heart failure. On the other hand, diminished pulmonary blood flow and cyanosis could be the consequence of pulmonary vascular disease caused by long standing pulmonary hypercirculation. Any of these two mechanisms may well play a decisive role in the ultimate course of this hemodynamically complicated malformation.

SUMMARY

A case of tetralogy of Fallot complicated by the absence of the pulmonary valve is reported.

The case had the associated anomaly of origin of the left pulmonary artery from the aortic arch.

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MALPOSITION OF PULMONARY ARTERIES (CROSSED PULMONARY ARTERIES) IN PERSISTENT TRUNCUS ARTERIOSUS*

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AN UNCOMMON arterial anomaly is that in which the ostium of the left pulmonary artery lies to the right of and above that of the right pulmonary artery. From these positions, the 2 pulmonary arteries cross each other as they proceed to their respective lungs.

Jue and associates[2] proposed the name "crossed pulmonary arteries" for this condition and described an example in a newborn infant with the trisomy 18 syndrome. In addition to the pulmonary arterial malformation in question, their case exhibited an atrial septal defect of the so-called sinus venosus type associated with anomalous connection of the right upper pulmonary vein to the right atrium. The great arteries were otherwise normally developed.

The present report deals with 3 cases, each with persistent truncus arteriosus, in which the ostia of the pulmonary arteries were malpositioned. In 2 of the cases, the pulmonary arteries crossed each other and there was interruption of the aortic arch; in the third case, there was atresia of the aortic arch, but without crossing.

Knowledge of the occurrence of "crossed pulmonary arteries" in the presence of persistent truncus arteriosus associated with malformations of the aortic arch may aid in the interpretation of angiocardiograms in appropriate cases.

DESCRIPTION OF SPECIMENS

An analysis was made of 13 specimens with persistent truncus arteriosus, in each of which the pulmonary arteries arose from the truncus (Types I and II).[1]

In 10 of these cases, the left and right pulmonary arteries arose next to each other, the ostium of the left artery lying to the left of the ostium of the right (Fig. 1).

In 3 cases, there were abnormalities in origin of the pulmonary arteries and, in 2 of these, in the courses that all the arteries took. These 3 cases form the basis for the descriptions below.

ILLUSTRATIVE CASES

CASE I. The specimen was from a newborn infant, of whom no other information was available to us. The heart exhibited a classic example of persistent truncus arteriosus, Type I.[1] The branches of the aortic arch were normal. The segment of the aorta between the left subclavian artery and the ductus arteriosus was represented by an atretic strand. The pulmonary arterial ostia which were malpositioned arose from the pulmonary aspect of the persistent truncus. The orifice of the left pulmonary artery lay 2 mm. superior and to the right of the orifice of the right pulmonary artery (Fig. 2; and 3, *a* and *b*). From these origins, the 2 pulmonary arteries crossed each other as they coursed toward their respective lungs. In crossing, the left pulmonary artery was superior to the right. The ductus arteriosus was patent although narrow. Its pulmonary end appeared to begin from the superior aspect of the pulmonary aspect of the truncus near the left pulmonary artery.

CASE II. A newborn female infant died at the age of 1 week following a surgical procedure for congenital atresia of the duodenum. The heart showed a Type I persistent truncus arteriosus, with complete interruption of the aortic arch. The pulmonary portion of the truncus was dilated and communicated directly through a

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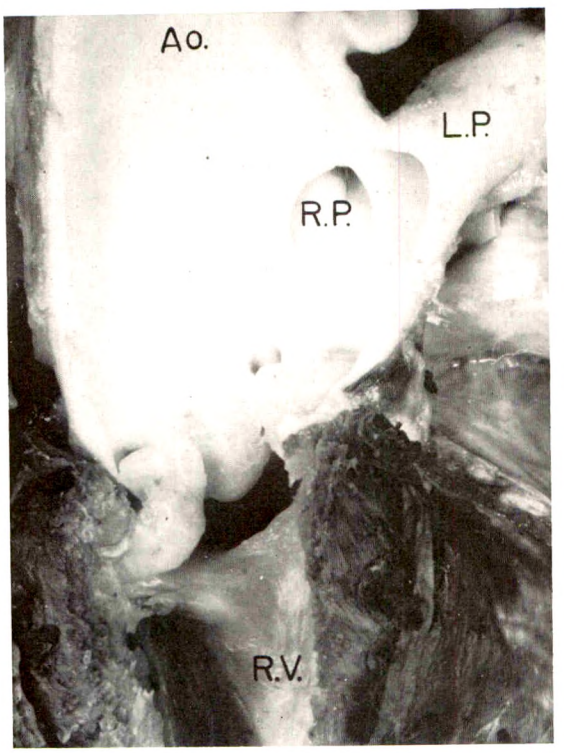


FIG. 1. An example of normally oriented pulmonary arteries in persistent truncus arteriosus (Type II). The ostia of the pulmonary arteries lie next to each other, with the ostium of the left pulmonary artery (L.P.) to the left of that of the right pulmonary artery (R.P.). R.V. = right ventricle; Ao. = ascending aorta beyond truncus.

widely patent ductus arteriosus with the descending aorta. The left subclavian artery arose from the descending aorta opposite the ductus arteriosus. The ostia of each of the pulmonary arteries were separated by 9 mm. The orifice of the left pulmonary artery was located superiorly and slightly to the right of the ostium of the right pulmonary artery. There was, as in Case I, a typical pattern of "crossed pulmonary arteries," with the left pulmonary artery crossing superior to the right (Fig. 4; and 5, *a* and *b*).

CASE III. A female infant, born at 32 weeks gestational age, died 1 hour after birth with multiple congenital malformations associated with the trisomy 18 syndrome. The heart showed a classic example of persistent truncus arteriosus, classified as Type I and associated with interruption of the aortic arch. There was no continuity between the walls of the aortic arch and descending aorta. The short pulmonary trunk, after its origin from the persistent truncus, was dilated and connected through a patent ductus arteriosus with the descending aorta. The left subclavian artery originated from the descending aorta, immediately distal to the orifice of the ductus arteriosus. Each of the 2 main pulmonary arteries originated from the pulmonary portion of the truncus. The orifice of the left pulmonary artery, which was narrow, was positioned superior to that of the right pulmonary artery (Fig. 6, *a* and *b*). From these origins, the vessels proceeded to their respective lungs without crossing.

DISCUSSION

Although congenital anomalies of major pulmonary arteries are uncommon, a variety of entities come under this category. Included are such conditions as: (1) localized stenosis or hypoplasia of 1 or both branches; (2) absence of 1 or each artery; (3) anomalous origin of either 1 or both branches from a vessel other than the pulmonary trunk; and (4) malposition of the origins of the pulmonary arteries, either

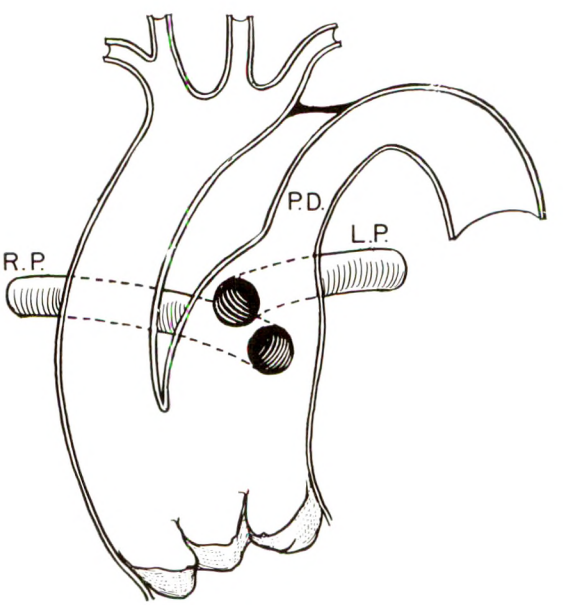


FIG. 2. Case 1. Schematic drawing of vascular abnormalities. Persistent truncus arteriosus with atresia of aortic arch and patent ductus arteriosus (P.D.). The ostium of the left pulmonary artery (L.P.) lies to the right of and above that of the right pulmonary artery (R.P.). The branches cross as each proceeds to its respective lung.

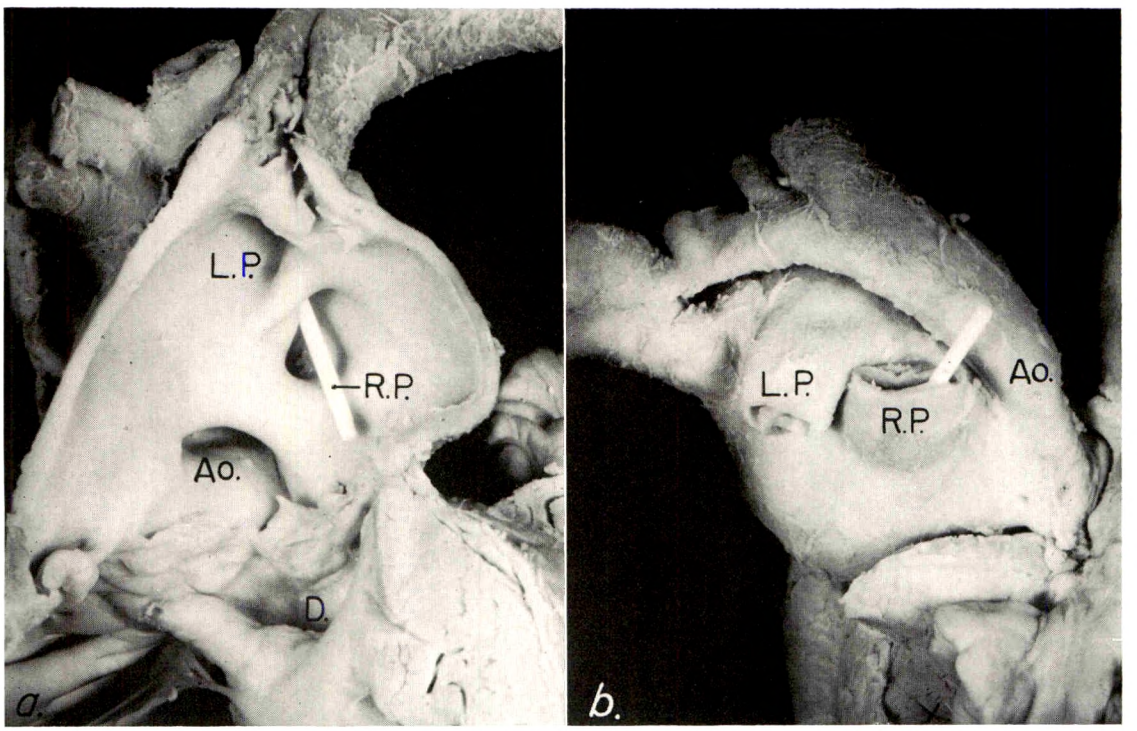
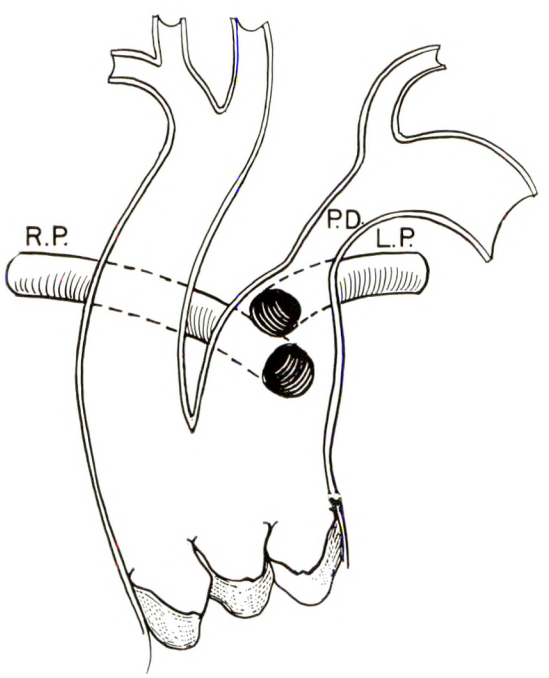


Fig. 3. Case 1. (*a*) Interior of persistent truncus arteriosus. Vessel overrides a ventricular septal defect (D). The orifice of the left pulmonary artery (L.P.) lies to the right and above that of the right pulmonary artery (R.P.). Ao. = ascending aorta. (*b*) Posterior view of exterior of great vessels showing the left pulmonary artery (L.P.) crossing superiorly to the right pulmonary artery (R.P.). Ao. = ascending aorta.



from the pulmonary trunk or from a persistent truncus arteriosus.

In the case with the latter condition, reported by Jue and associates,[2] each pulmonary artery arose from the pulmonary trunk. The origin of the left pulmonary artery lay to the right of and above the orifice of the right pulmonary artery. Each artery crossed the other as it coursed to its respective lung. Jue and his group[2] proposed the term "crossed pulmonary arteries" for this anomaly.

In our study, there were 3 cases in which the pulmonary arteries arose anomalously

Fig. 4. Case II. Schematic drawing of vascular abnormalities. Persistent truncus arteriosus with interruption of aortic arch and patent ductus arteriosus (P.D.). The left (L.P.) and right (R.P.) pulmonary arteries show abnormalities similar to that in Case 1.

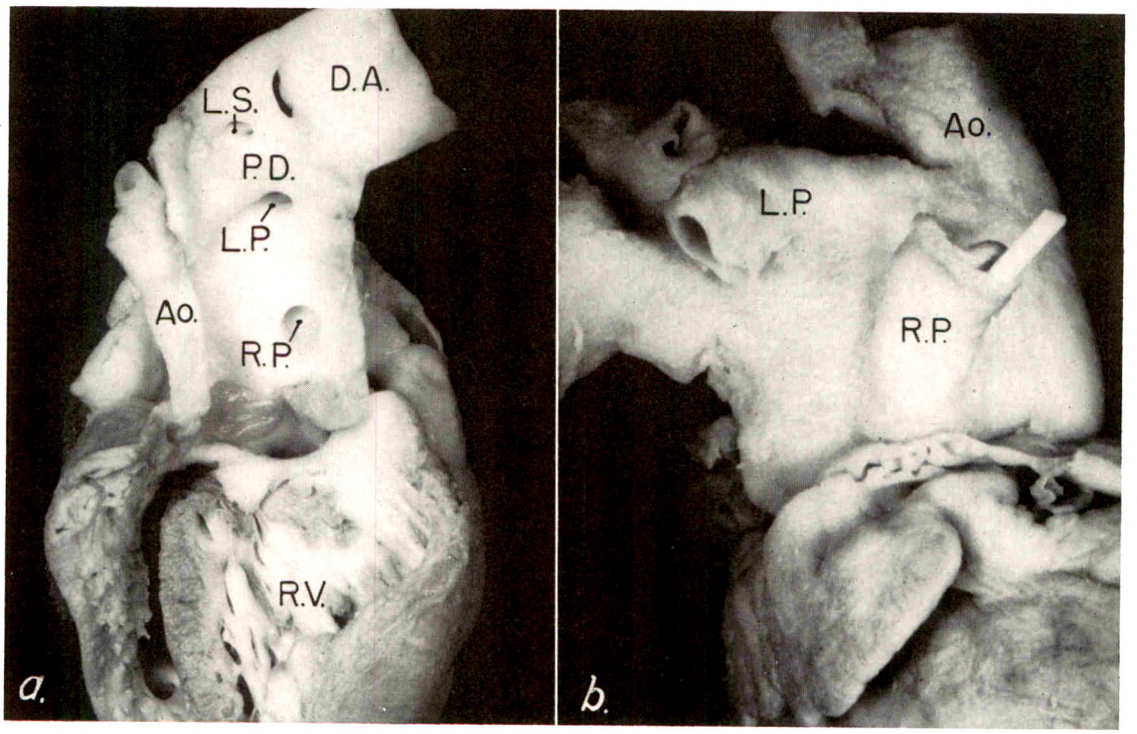


FIG. 5. Case II. (*a*) Right ventricle (R.V.) and great vessels. The ascending aorta (Ao.), which arises from the truncus arteriosus, ends by dividing into the innominate and left common carotid arteries. The ostium of the left pulmonary artery (L.P.) lies slightly to the right of and above that of the right pulmonary artery (R.P.). A patent ductus arteriosus (P.D.) leads from the pulmonary portion of the persistent truncus arteriosus to the descending aorta (D.A.). The left subclavian artery (L.S.) arises from the latter segment (artifact beyond subclavian ostium). (*b*) Posterior view of great vessels showing crossing of the left pulmonary artery (L.P.) superiorly to right pulmonary artery (R.P.). Ao. = ascending aorta.

and, in 2 of these, the pulmonary arteries crossed each other. These cases were found among 13 specimens with persistent truncus arteriosus (Types I and II). In the 1 case with anomalously positioned ostia but without crossing (Case III), the malformation is considered a lesser form of classical "crossed pulmonary arteries." It is of interest that, in each of our cases of persistent truncus with this pulmonary arterial anomaly, interruption or atresia of the aortic arch occurred.

To our knowledge, there have been no reported cases of "crossed pulmonary arteries" in persistent truncus arteriosus, although an anomalous position of the orifices of the pulmonary arteries has been described in this condition. In several reports, a superiorly located orifice of the left pulmonary artery has been mentioned.[4,5] Keith and associates[3] described, in persistent truncus arteriosus, the angiocardiographic phenomenon of a high arching left pulmonary artery. Van Praagh and Van Praagh[5] attributed this sign to the superior position of the orifice of the left pulmonary artery in persistent truncus. Although mentioning that the left pulmonary artery may arise above the right, the latter authors did not indicate how common this change was among the 57 specimens with persistent truncus that they reviewed. They did not mention the phenomenon of crossing. Victorica and associates,[6] in a clinical study of 14 cases of persistent truncus arteriosus in infancy, mentioned that, in roentgenograms, an abnormally high left hilum was an un-

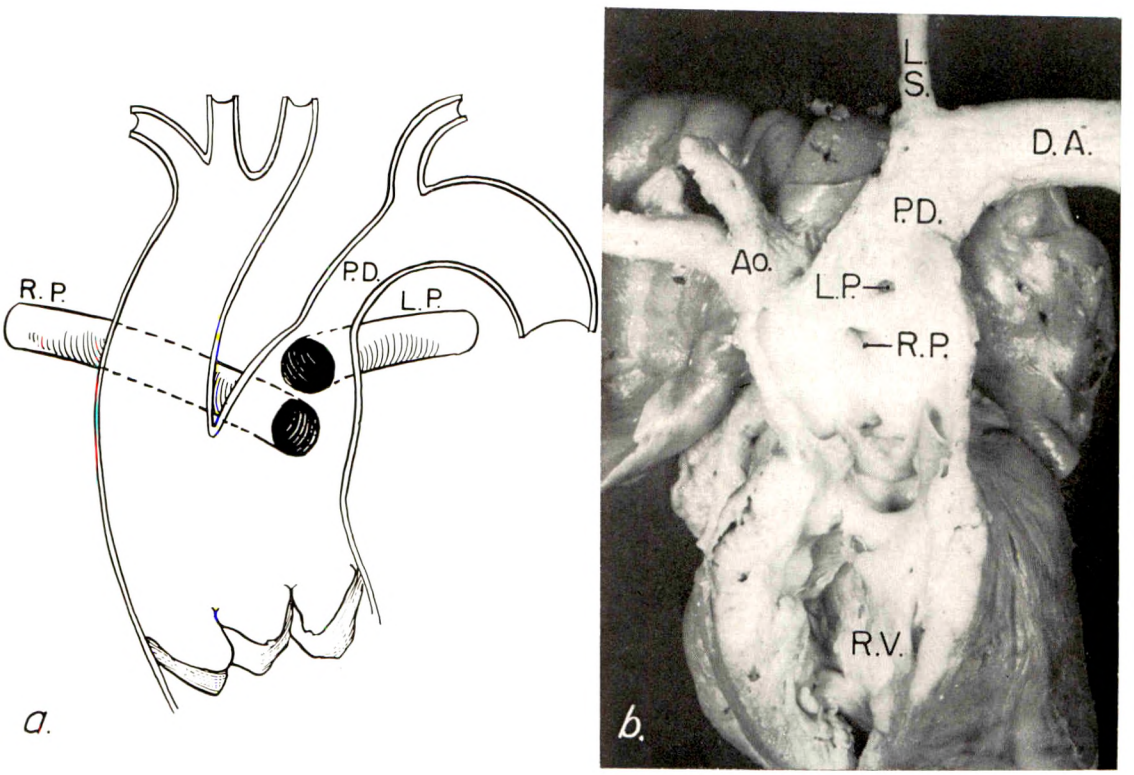


Fig. 6. Case III. (*a*) Schematic drawing showing persistent truncus arteriosus with interruption of aortic arch and patent ductus arteriosus (P.D.). (*b*) Interior of right ventricle (R.V.), persistent truncus arteriosus, patent ductus (P.D.) and descending aorta (D.A.). The left pulmonary arterial ostium (L.P.) is narrow and arises above the ostium of the right pulmonary artery (R.P.). Ascending aorta (Ao.) arises from persistent truncus and ends by branching into the innominate and left common carotid arteries. Left subclavian artery (L.S.) arises from descending aorta.

common finding. This could indicate the rarity of malpositioned orifices of the pulmonary arteries. It is of interest that no cases with interrupted aortic arch were encountered in their series. In each of our cases with crossed pulmonary arteries, interruption or atresia of the aortic arch was present.

The developmental basis for the anomalous position of the ostia of both pulmonary arteries is not certain. Jue and associates[2] suggested that this condition could result from faulty differential growth during the process of partitioning of the truncus arteriosus into the aorta and pulmonary trunk.

Although the condition of "crossed pulmonary arteries" is of no hemodynamic significance, knowledge of its occurrence, whether or not in combination with persistent truncus, may aid in the interpretation of certain peculiarities encountered in the angiograms.

SUMMARY

Among 13 specimens with persistent truncus arteriosus, there were 3 cases with malposition of the pulmonary arteries. In 2 specimens, the ostium of the left pulmonary artery lay to the right of and above that of the right pulmonary artery. The 2 pulmonary arteries crossed each other as they proceeded to their respective lungs: "crossed pulmonary arteries." The third specimen presented anomalously positioned ostia of the pulmonary arteries but without crossing. This condition is considered to be a lesser form of classical "crossed pulmo-

nary arteries." Each of these cases also exhibited interruption or atresia of the aortic arch.

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COARCTATION OF THE ABDOMINAL AORTA*

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COARCTATION of the abdominal aorta is uncommon. The disease entity may be a developmental anomaly, although the prevailing theory in its etiology is an arteritis similar to a variety of Takayashu's aortitis.[6,8]

The purpose of this paper is to report 2 points, the benefits of surgical therapy and prognosis.

REPORT OF CASES

Case 1. L.Y. An asymptomatic 16 year old white female was first noticed to have systolic hypertension during a physical examination at 7 years of age. She was referred to the Yale-New Haven Hospital at 12 years of age for persistent systolic hypertension, a heart murmur and weak femoral pulses.

Physical examination. The blood pressure in the right arm was 140/90 and about 80 mm. Hg in the legs. There was no cardiomegaly. A left ventricular impulse was palpable. A harsh ejection systolic murmur was best heard over the second right intercostal space and was conducted towards the neck. No murmur was heard over the back, lumbar area or over the abdomen. The radial pulses were strong and easily palpable. The femoral pulses were very weak.

Except for left axis deviation, the electrocardiogram was normal.

Roentgenologic findings. Roentgenograms of the chest showed normal heart size and pulmonary vasculature. No rib notching was seen.

Venous angiocardiogram with injection of the contrast medium into the right atrium and antegrade aortogram via brachial artery were obtained (Fig. 1, only the lateral projection is currently available). Both studies demonstrated coarctation of the abdominal aorta extending from the mid-portion of D12 for approximately $7\frac{1}{2}$ cm. The renal arteries, the superior mesenteric and the celiac axis showed post-stenotic dilatations as they originated from the coarcted segment. Just distal to the coarctation was minimal post-stenotic dilatation proximally but the distal abdominal aorta was diffusely small.

Surgery. At surgery the abdominal aorta showed a narrow coarcted segment, 7–8 cm. long. The narrowing was conspicuous at the center where the external diameter was about

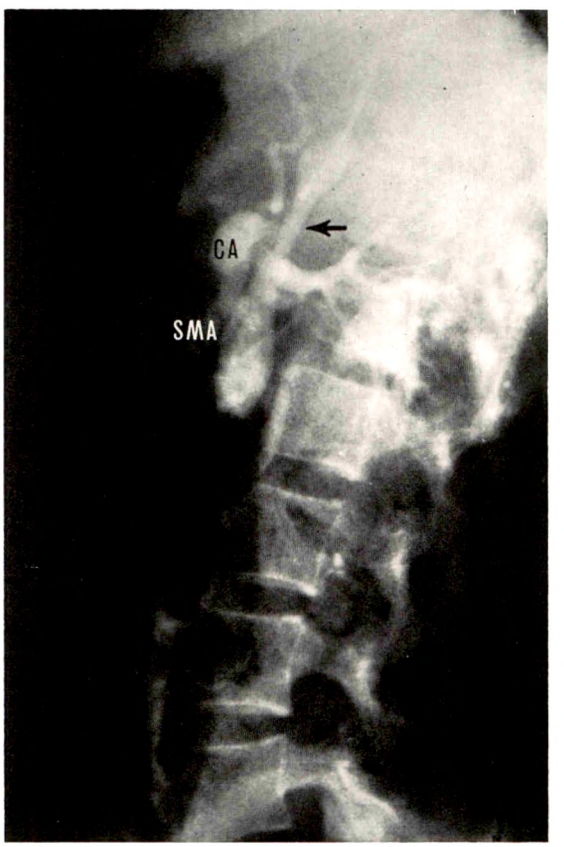


Fig. 1. Case 1. Preoperative aortogram in the lateral projection demonstrating a diffuse narrowing of the abdominal aorta (7.5 cm. long) at the level of mid D12 to mid L1 (arrow). The proximal celiac axis (CA), superior mesenteric artery (SMA), and aorta immediately distal to the coarctation show post-stenotic dilatation. However, the distal abdominal aorta appears diffusely narrow.

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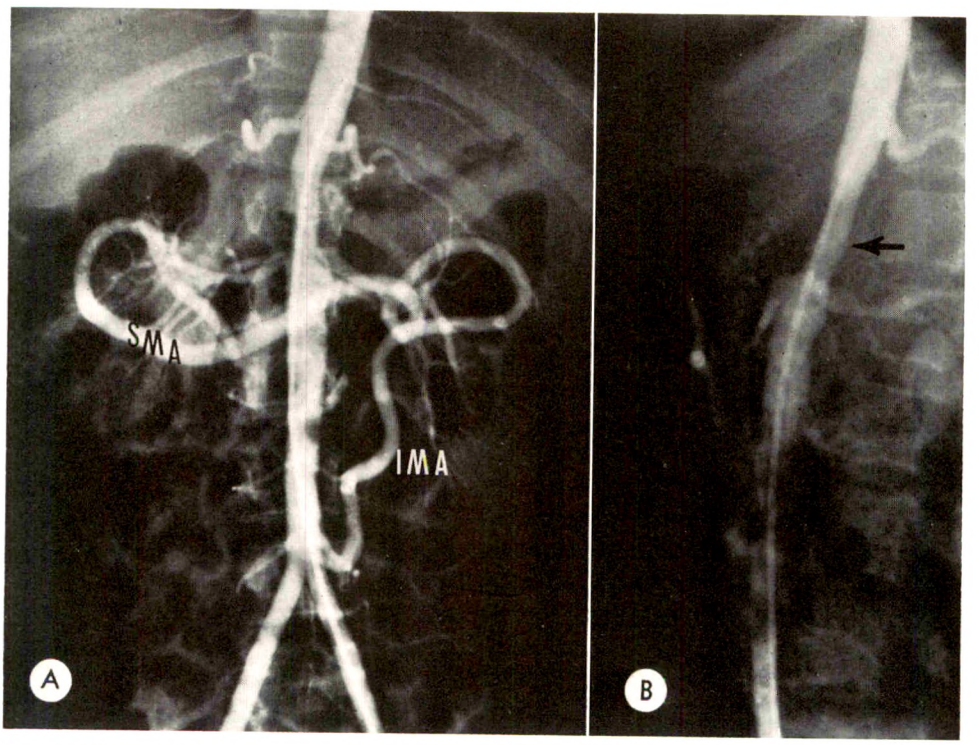


FIG. 2. Case 1. (*A* and *B*) Biplane aortograms after patch grafting. The coarcted segment is twice the lumen diameter compared to the preoperative state. The distal abdominal aorta is of normal caliber. The inferior mesenteric artery (IMA) is dilated and fills in retrograde fashion the superior mesenteric artery (SMA).

4–5 mm. A membrane was present in the center of the coarcted segment. This was excised and a patch dacron graft was sutured to the aortic wall to restore a normal sized lumen.

Follow-up. Retrograde aortography (Fig. 2, *A* and *B*) demonstrated improvement in the coarcted lumen to at least twice the diameter of the preoperative state. Four years after surgery, she was asymptomatic with blood pressures of 130/88 mm. Hg in the upper and lower extremities.

CASE II. K.P. A 19 year old white female was admitted to the Bridgeport Hospital. Chief complaints of numbness and pain of both lower extremities of 1 year's duration followed moderate to severe exertion. She had vague upper abdominal pains. At 16 years, she was hypertensive. An extensive study at this time showed no abnormality in the blood chemistries, urinary catecholamines, 5-hydroxy-indole-acetic acid, and aldosterone levels, intravenous pyelogram, electrocardiogram and roentgenograms.

Physical examination. The blood pressure in the right arm was 209/130, left arm 200/120 mm. Hg and could not be obtained in the lower extremities. There was no cardiomegaly. An early ejection systolic murmur was heard over the apex with maximal intensity over the mid-abdominal area. No murmur was heard at the back. The femoral pulses could not be felt. The radial pulses were strong.

The electrocardiogram was normal. Right heart catheterization was normal. The left brachial artery pressure was 230/140 and the lower abdominal aortic pressure was 168/125 mm. Hg.

Roentgenologic findings. Roentgenograms of the chest demonstrated normal heart size and pulmonary vasculature. Upper gastrointestinal studies and cholecystography were normal. The right 9th and 10th ribs included in the gallbladder roentgenograms showed minimal notching.

Retrograde aortogram (Fig. 3) showed abrupt narrowing of the abdominal aorta at the level of the origin of the celiac axis. The entire abdominal aorta and proximal common iliac arteries were hypoplastic. There was stenosis of both proximal renal arteries, left more than right. The inferior mesenteric artery was dilated

and opacified retrograde to the right colic branches of the superior mesenteric artery. The lower intercostal and lumbar arteries were dilated and tortuous.

Surgery. At surgery (Dr. Michael DeBakey), she had a bypass graft from the descending thoracic aorta to the abdominal aorta and a bypass graft from the abdominal aortic graft to both renal arteries performed. The pulses of the lower extremities were restored in the postoperative period. Blood pressure in the arms now is 140/80 mm. Hg.

Follow-up. In 1 year she underwent aortography for evaluation of the graft and abdominal aorta and its run-off (Fig. 4, *A* and *B*). She has remained symptom free since the operation.

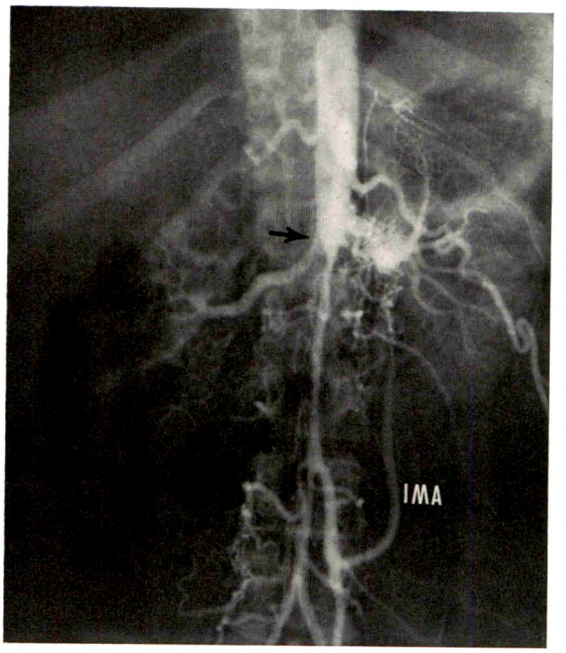


FIG. 3. Case II. Preoperative retrograde aortogram in a 19 year old hypertensive female. The study demonstrated an abrupt coarctation of the upper abdominal aorta at the level of the first lumbar vertebral body. The origins of the celiac axis, superior mesenteric and both renal arteries were involved. The abdominal aorta distal to the coarcted segment was very narrow which extended to the common iliac arteries. The inferior mesenteric artery (IMA) was dilated and serial roentgenograms revealed subsequent opacification of the superior mesenteric artery. The other roentgenograms showed dilated and tortuous low intercostal arteries.

DISCUSSION

This condition has a female sex predominance and affects frequently children and young adults.[6,8] Variable symptoms may occur directly from the lesion in the form of leg cramps, easy fatigue of the lower extremities and occasionally abdominal angina. Indirectly the patients may manifest signs and symptoms of congestive heart failure and cerebrovascular accident.

Hypertension is commonly present in the upper half of the body and frequently to an alarming degree. This may be ascribed to a purely obstructive lesion of the aortic blood flow which results in elevation of the pressure in a confined aortic location. However, a better explanation is the formation of humoral factors from the kidneys due to low perfusion pressure of the kidneys. This may be secondary to coarctation of the abdominal aorta or actual involvement of one or both proximal renal arteries.[1]

One interesting facet in both cases presented is the lowering of the blood pressures to normal after surgery.

Abdominal pain may be vague and minor as in our Case II or may be catastrophic.[5] The mechanism of the gastrointestinal symptomatology is due to involvement of the origins of the celiac, superior and inferior mesenteric arteries adjoining the coarcted segment of the abdominal aorta. There results a low perfusion and ischemia of the gastrointestinal tract. Another reason for the visceral pain may be diversion of the blood flow of the viscera to the lower extremities by way of collateral circulation (viscus to somatic steal).

Proper abdominal palpation may elicit a strong aortic pulse in the upper abdomen which diminishes in intensity or disappears distally. This maneuver is valuable in differentiating coarctation of the abdominal aorta from that of the thoracic aorta. Our Case II demonstrated a systolic murmur in the mid abdomen which is ascribed to the turbulence in the aortic flow at the coarctation.[4]

A diagnostic point is illustrated in Case II by the erosions of the right 10th and 11th ribs which were discovered by accident in

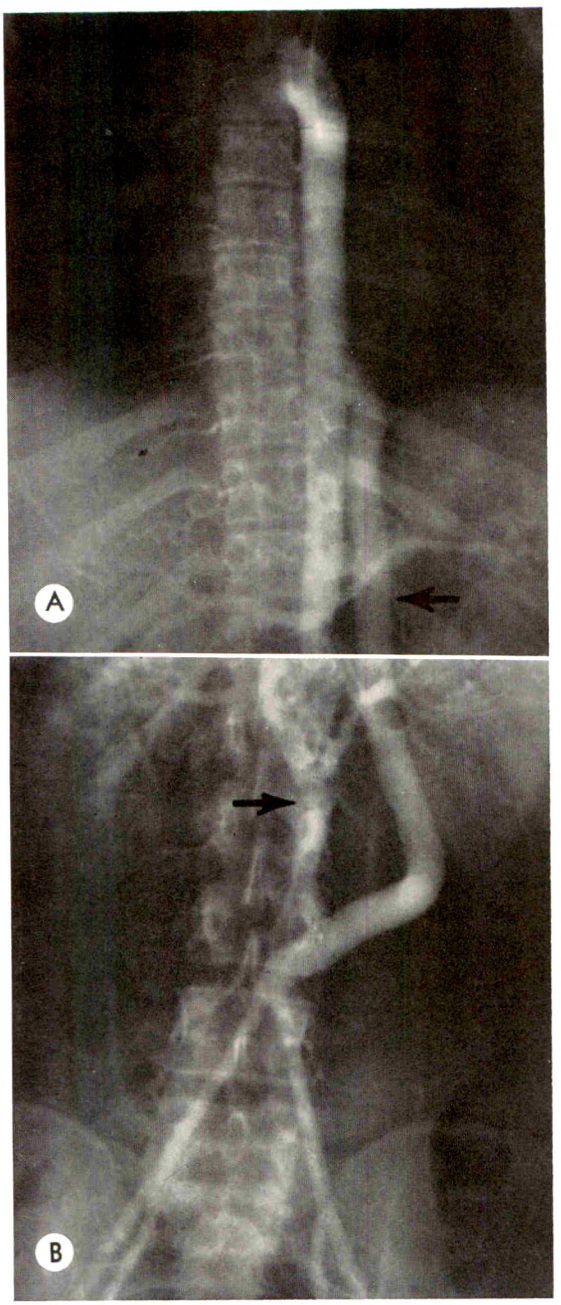


FIG. 4. Case II. (*A* and *B*) Postoperative aortograms via left axillary approach 1 year after surgery. There is wide patency of the graft (arrow in *A*) from the mid-thoracic aorta to the distal abdominal aorta as well as the branching graft (arrow in *B*) from the distal graft to both renal arteries. The common iliac arteries are now of normal size.

cholecystograms. This finding is due to the pressure effects of the dilated and tortuous low intercostal arteries which majorly participate in the collateral circulation to the lower part of the body.

Coarctation of the low thoracic or upper abdominal aorta must be considered strongly in the presence of upper extremity hypertension and a disappearing or absent pulsation of the abdominal aorta. The chest roentgenograms reveal absence of roentgenologic findings of coarctation at the aortic arch level. Closer scrutiny of the lower ribs may demonstrate rib notching in the 10th to 12th ribs (Case II).

Aortography[6–8] is necessary in the diagnostic evaluation of this disease. The size, site and extent of aortic obstruction can be determined. The involvement of visceral vessels and the degree of collateral circulation are identified. These factors, along with the symptomatology, will help determine the type and scope of vascular surgery.[3]

A simple excision of a localized coarctation and subsequent anastomosis of the aorta may suffice. If the lesion is somewhat small and adjacent to origins of the visceral arteries, a patch graft may be added for proper restoration of the aortic caliber (Case I). When the lesion is fairly extensive, prosthetic grafts are necessary to divert the aortic flow from the low thoracic to the abdominal aorta or iliac arteries (Case II). Another graft is applied to the renal arteries to enhance the perfusion of the kidneys.

The pathologic lesion consists of increased density in the periaortic connective tissue. There is marked intimal proliferation with accumulation of elastic and collagenous tissue in the media. The first case shows another interesting finding of a membrane in the center of the coarcted segment.

In both cases, the distal abdominal aorta and iliac arteries were extremely narrow in the preoperative aortograms. Originally, this was thought to be hypoplasia of the vessels rendering judgment of the extent of the lesion very difficult. Aortograms after prosthetic grafting (Case II) demonstrated restoration of the iliac arteries to normal

caliber. The plausible explanation of the initial observation is now ascribed to the low volume and diminished intravascular pressure due to the coarctation rather than diffuse extension of the lesion.

If the lesion is not corrected surgically, the patient may die of cardiac decompensation from prolonged and severe hypertension, cerebrovascular hemorrhage or visceral ischemia.[1]

SUMMARY

Two cases of coarctation of the abdominal aorta are reported to illustrate the salient diagnostic points and the improvement following surgery.

Coarctation of the low thoracic or abdominal aorta must be considered strongly in young female patients who have upper extremity hypertension, strong upper abdominal aortic pulse which disappears distally or no aortic pulse in the abdomen and a murmur or bruit in the epigastrium. Chest roentgenograms show lack of findings of coarctation of the aorta at the arch level, but associated rib notching of the 10th to 12th ribs. Aortography is essential in making the diagnosis and as a guide to the therapeutic approach. Vascular surgery will alleviate the condition.

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THE CALYCEAL CRESCENT—A ROENTGENOGRAPHIC SIGN OF OBSTRUCTIVE HYDRONEPHROSIS*

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IN SOME infants and children with obstructive hydronephrosis, a series of crescent-shaped shadows of increased density appears in the early part of the urographic examination. These crescent-shaped shadows disappear in the later phase of the examination as the distended pelvicalyceal system becomes opacified by excreted contrast medium. The occurrence of these shadows in the first 20 to 30 minutes of excretory urography is so strongly indicative of severe obstructive hydronephrosis that the examination should be continued for as long as 24 hours if necessary, with or without the use of a second intravenous injection of contrast medium (Fig. 1, *A* and *B*).

THEORETIC CONSIDERATIONS

We believe that the calyceal crescents are produced by the collecting tubules which, in the course of being stretched, distorted and displaced by the progressive distention of the calyx, become realigned so that they lie parallel to the everted margin of the calyx, rather than perpendicular to it.[9] The evidence for this is as follows:

1. The normal collecting tubules are frequently visualized during excretory urography as a "brush effect" extending as a fan-like increase in density from the renal papilla outwards (Fig. 2, *A* and *B*).[7] That this shadow does represent the normal collecting tubules containing contrast medium has been confirmed experimentally.[6]
2. It is known that water resorption occurs in the tubules. In hydronephrosis with increased intrapelvic pressure, passage of the urine through the collecting tubules is slowed and water resorption may be increased. It may, therefore, be inferred that the increase in concentration of the contrast medium is enhanced in the collecting tubules relative to its concentration in the normal subject.[3]
3. Histologic examination of kidneys removed because of obstructive hydronephrosis clearly shows that the collecting tubules are indeed realigned so as to lie parallel rather than at right angles to the interface between the renal parenchyma and the distended calyx (Fig. 3, *A–C*).[10]
4. As excretion into a dilated collecting system occurs, the heavy contrast material sinks to the most dependent portion of the distended calyx or pelvis. This can be demonstrated by changing the position of the patient and showing corresponding displacement of the contrast material, and by using horizontal beam roentgenography in any position (with the patient upright, lying on the side, or supine) when a fluid-fluid level appears (Fig. 4, *A–C*).[8] The crescentic shadows, however, remain fixed in position with changes in the posture of the patient; they remain in the renal parenchyma in all positions, and show no tendency with horizontal beam roentgenography to formation of fluid-fluid levels (Fig. 5).
5. The timing of the appearance and

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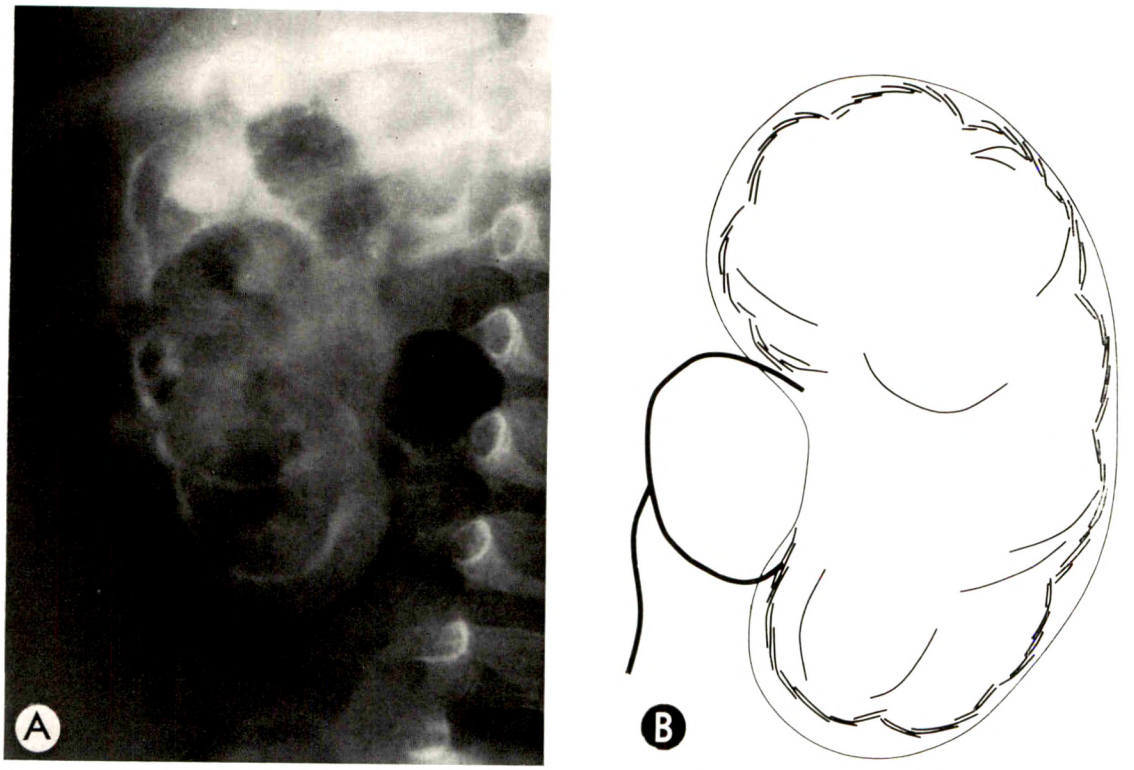


Fig. 1. (*A*) Typical crescents during urography. (*B*) Schematic illustration of the collecting tubular visualization in hydronephrosis.

disappearance of the crescent shadows, which are present frequently within one-half hour of injection and are followed by opacification of the pelvicalyceal system, is in favor of our postulate (Fig. 6, *A* and *B*).

6. The density of the crescentic shadows is greater than that shown by the nephrogram of the normal or hydronephrotic kidney (Fig. 7, *C* and *D*; Case III) and approximately equal to maximum opacification anticipated and achieved by intrarenal concentration of the contrast medium.
7. Intermediate stages of the abnormality are sometimes observed; in these, shadows are present which appear to be collecting tubules undergoing change in configuration from the normal "brush effect" to calyceal crescents. This occurs in hydronephrosis which is mild to moderate in degree (Fig. 8).

Experimental Animal and Clinical Observations

We have attempted to reproduce this phenomenon in experimental animals by artificially created chronic obstructive hydronephrosis, but have not yet succeeded. We have, however, been able to duplicate Fleischner and associates'[6] results in showing the normal collecting tubules experimentally, by sacrificing the animal during excretory urography and roentgenographing slices of the kidney removed at autopsy.

A few measurements in the operating room of intrapelvic pressures, just before corrective surgery or nephrectomy, were done in kidneys whose urographic investigation had shown calyceal crescents. The measurements as yet are too few to be conclusive, but suggest that the intrapelvic pressure is raised (in 5 patients the intrapelvic pressure was 10–20 mm. Hg). So far as we know, however, the spectrum of

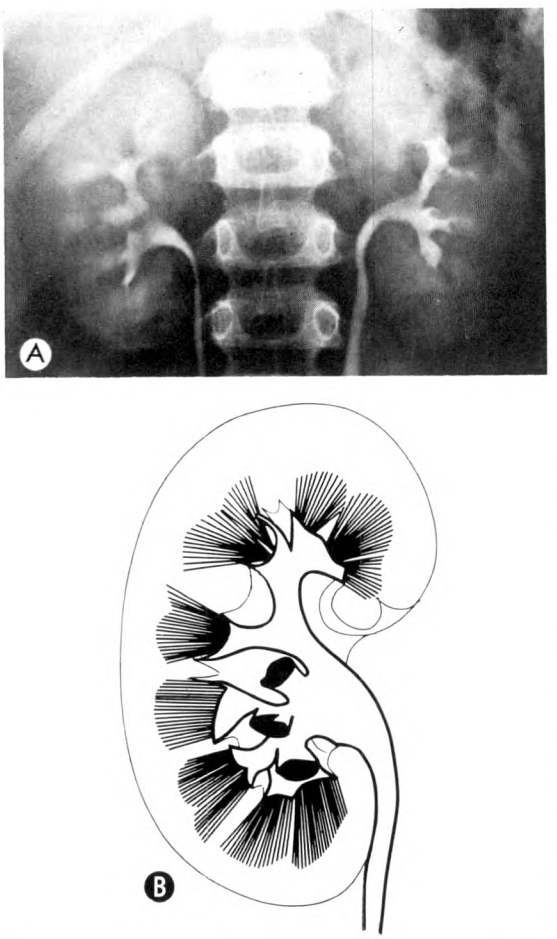


FIG. 2. (*A*) Normal collecting tubular opacification during urography. (*B*) Schematic illustration of the normal tubular filling.

normal values of resting intrapelvic pressures in children is not known.[5]

When the obstruction causing hydronephrosis has been successfully relieved by catheterization of the renal pelvis, nephrostomy, or corrective surgery, the "calyceal crescent" sign may disappear, without collecting tubule visualization on subsequent urographic examinations. If corrective surgery improves drainage of the collecting system and the hydronephrotic distention of the pelvicalyceal system undergoes marked change toward normal, it can sometimes be demonstrated that the collecting tubules—as shown by the "brush effect"—become reorientated and are again normally demonstrable during excretion of contrast medium (Fig. 9, *A–C*; Case IV).

The "calyceal crescent" sign has been shown in our material with di-iodo and tri-iodo contrast medium, using low dose and high dose urography.[4] We do not use dehydration of the patient as a part of preparation for urography since it is potentially dangerous.[12] Therefore, we cannot express any opinion as to what effect dehydration might have on the frequency of this sign.

We have not observed the "calyceal crescent" sign in acute hydronephrosis of a few hours' duration—such as that frequently caused by calculi, post-traumatic blood clot, transient postoperative obstruction of mild degree, or intermittent hydronephrosis. It thus appears that not only must obstruction with increased intrapelvic pressure be present, but it must be present for some time, during which the collecting tubules presumably gradually become realigned, possibly stretched and distorted, but without loss of their function. The earliest documented appearance of calyceal crescent sign in our material is 10 days following obstruction (Fig. 7, *A* and *B*; Case III). The preservation of the function of the collecting tubules with, in particular, their ability to absorb water from the excreted urine just before it enters the calyceal cavity, appears to be essential for crescents to form.

In patients who have vesicoureteric reflux and long-standing dilatation, elongation and tortuosity of the ureter, with subsequent functional obstruction and elevated resting pressure in the collecting system, calyceal crescents may also be demonstrated.

Thus "calyceal crescents" may appear in hydronephrosis due to functional or mechanical obstruction.

Since we first reported this finding in 1959, it has also appeared under the name "ring sign."[2]

PROGNOSTIC EVALUATION

We have attempted, in reviewing our material, to reach an opinion as to whether

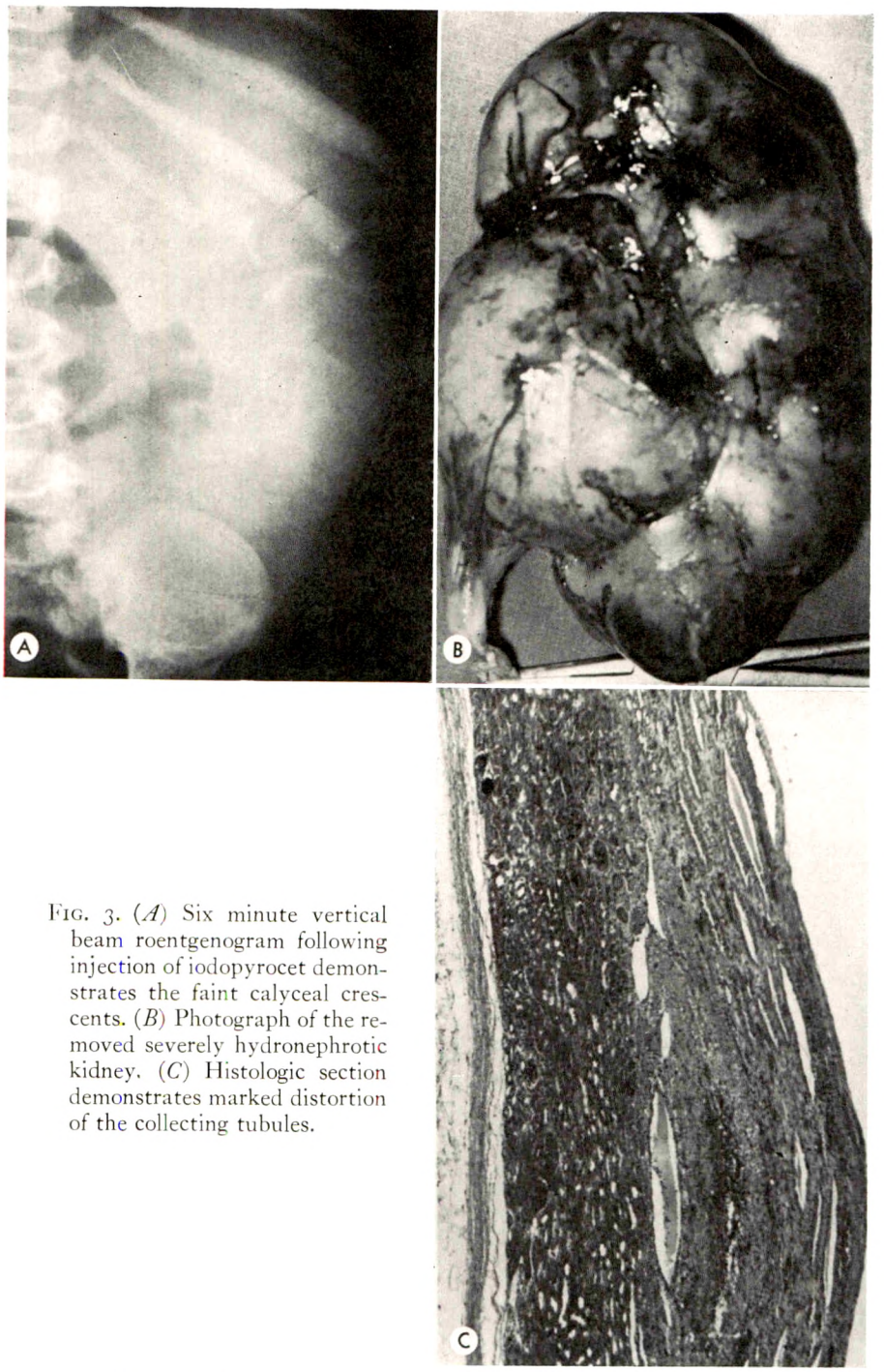


Fig. 3. (*A*) Six minute vertical beam roentgenogram following injection of iodopyrocet demonstrates the faint calyceal crescents. (*B*) Photograph of the removed severely hydronephrotic kidney. (*C*) Histologic section demonstrates marked distortion of the collecting tubules.

the appearance of the "calyceal crescent" sign gives any indication about surgical salvageability of the kidney. This sign by itself is insufficient; visualization of the renal parenchyma by nephrogram,[1] and often fluorographic evaluation of the pelviureteric function are necessary to establish a prognosis.

REPORT OF CASES

Case 1. G.A., No. 325931. A 3 year old boy was transferred from another hospital

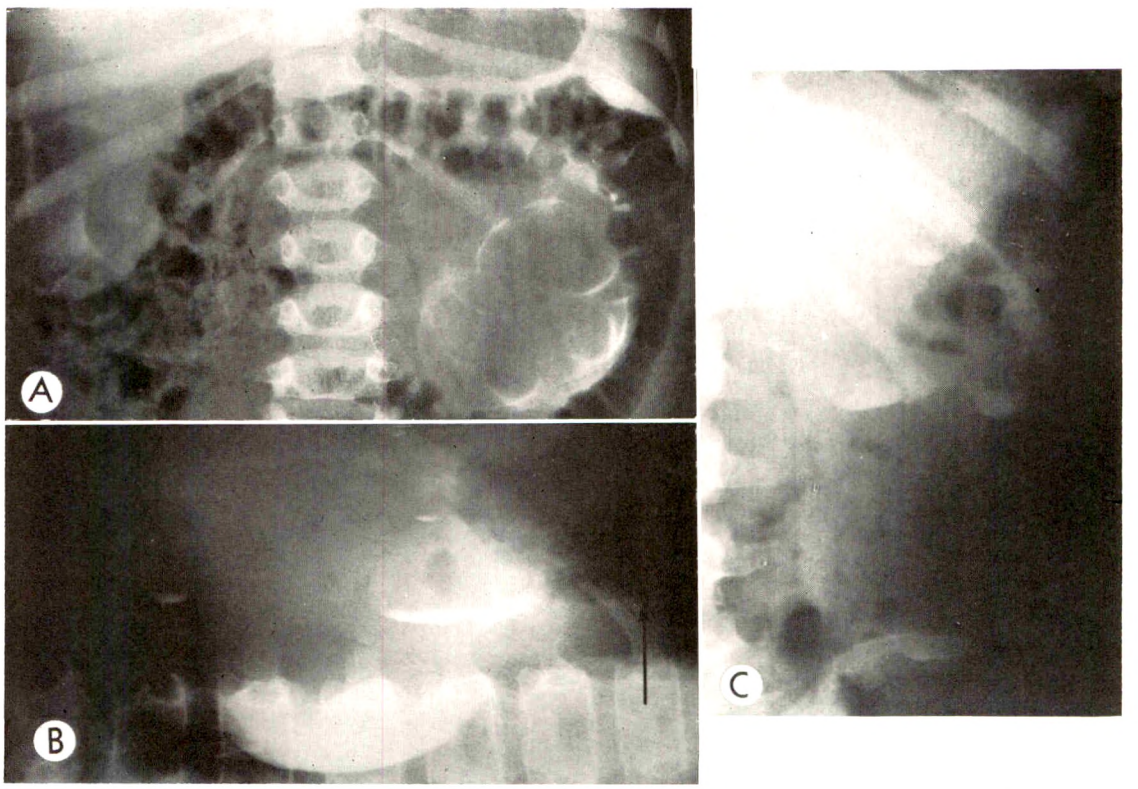


FIG. 4. (*A*) Calyceal crescents demonstrated on the 3 minute vertical beam roentgenogram. (*B*) Case IV. Horizontal beam roentgenogram (9 min.) in a patient lying on her side. (*C*) Twenty minute roentgenogram with the patient erect.

where he was investigated for vomiting and abdominal pain. Severe right-sided hydronephrosis was found on urography, secondary to pelviureteric junction obstruction.[4] There were no urinary signs or symptoms prior to this hospitalization. The child had a known absence of the left pericardium. On physical examination, a right-sided abdominal mass was palpated. There were no other significant findings.

During urography, calyceal crescents were shown and a thin rim of functioning parenchyma was demonstrated by nephrogram (Fig. 6, *A* and *B*). At operation a tense right-sided hydronephrosis was found due to pelviureteric junction obstruction. The renal parenchyma was extremely thin. Before nephrectomy the intrapelvic pressure was measured and found to be 10 mm. Hg. The content of the hydronephrotic sac was 350 ml. Right nephrectomy was done.

CASE II. J.R., No. 377013. This 2 month old boy was found to have a right-sided abdominal mass on routine physical examination. Apart from this mass, no abnormality was noted and the laboratory findings were noncontributory. Urography showed calyceal crescents and 2–3 mm. thick renal parenchyma on the right side. From the secondary signs a diagnosis of severe pelviureteric junction obstruction was made. On the basis of the roentgenologic examination, the kidney was not thought to be salvageable.

At surgical exploration, a large hydronephrosis was found with a thin cap of parenchyma. The intrapelvic pressure was 12 mm. Hg, and the content of the hydronephrotic sac was 245 ml. Nephrectomy was done.

CASE III. R.H., No. 54733. This girl was first hospitalized at the age of 2 months for bladder extrophy, abdominal hernia and prolapse of the rectum. In the first 2 months of life she had been seen and treated elsewhere. Her first intravenous urography in our hospital was done at the age of 3 years, prior to ureterosigmoidostomy. The kidneys and the upper collecting systems were normal and no collecting tubular filling occurred during urography (Fig. 7*A*).

Ten days following ureterosigmoidostomy,



Fig. 5. Intravenous urogram obtained before adequate drainage of obstructive hydronephrosis has been established. Air has been introduced into the upper collecting system via a nephrostomy tube. Calyceal crescents are shown corresponding to the anatomic site of the collecting tubules on the roentgenogram made with a horizontal beam.

moderately severe hydronephrosis was demonstrated bilaterally with flattening of the papillae and eversion of the calyces. Calyceal crescents were demonstrated bilaterally during urography. The hydronephrosis was secondary to ureterosigmoid junction obstruction. Subsequent to this postoperative urography, the patient was followed in our hospital and 5 urographic examinations were done over a period of 10 years. The partial ureterosigmoid obstruction persisted and the calyceal crescents were demonstrated on every examination, with progressive eversion of the minor calyces (Fig. 7, *B–D*).

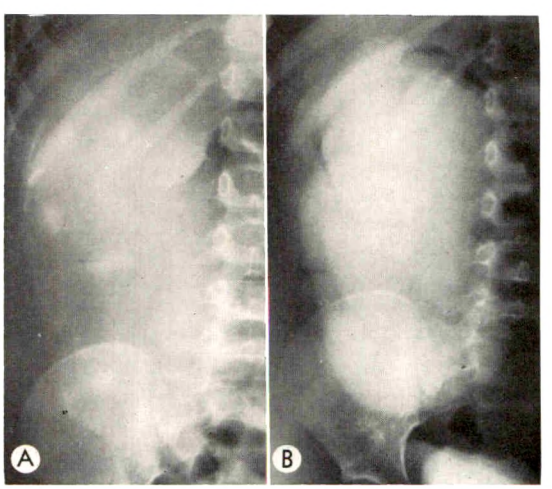


Fig. 6. Case 1. (*A*) Vertical beam roentgenogram 3 minutes after injection of meglumine diatrizoate. (*B*) Two hours following second injection, the large hydronephrotic sac is outlined. Roentgenologic diagnosis was pelviureteric junction obstruction.

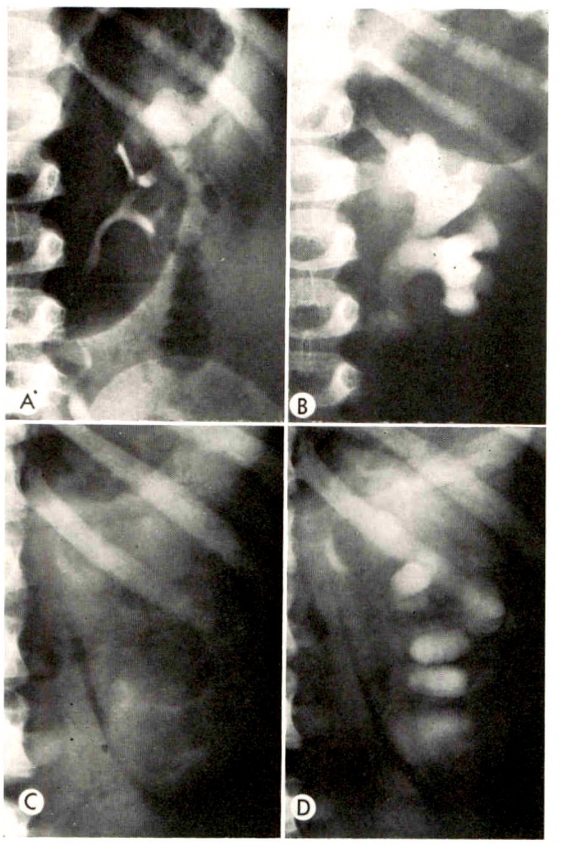


Fig. 7. Case III. (*A*) At 3 years of age, the kidneys and their collecting systems are normal. (*B*) Ten days following ureterosigmoidostomy the distorted collecting tubules are opacified during urography. (*C* and *D*) Urography 5 years following ureterosigmoidostomy shows deterioration of hydronephrosis and obvious calyceal crescents. (*C*) Three minute and (*D*) 15 minute roentgenograms. Note the layering of the contrast medium in the dependent portion of the dilated calyces.

At the age of 11 years, ileal conduit diversion was done because the patient continued to have recurrent pyelonephritis. Two months after the diversion via the ileal conduit, the calyceal crescents were still demonstrated but they were less obvious and fewer in number than on the preoperative examination.

Subsequent intravenous urographies from 11 to 18 years of age showed gradual decrease of hydronephrosis and gradual realignment of the collecting tubules with disappearance of the calyceal crescents roentgenologically. The drainage from the ileal conduit was satisfactory during this period of time.

Case IV. J.M., No. 256175. A 10 year old girl was admitted after her fourth episode in 1 month

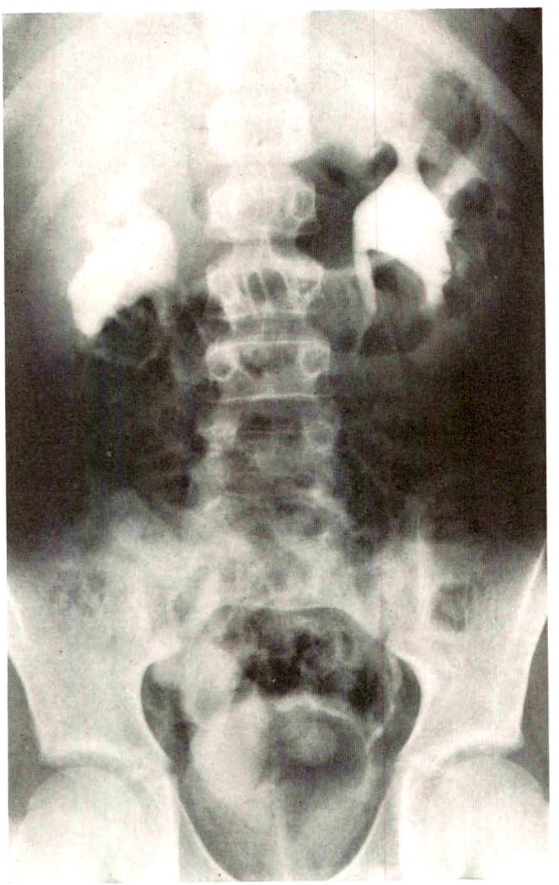


FIG. 8. A case of obstructive hydroureter and hydronephrosis. The hydronephrosis is mild, the calyces are in the early stages of ballooning and eversion, and the calyceal crescent sign in an intermediate stage is present, particularly in relation to the superior and middle calyces.

of generalized abdominal pain, vomiting and diarrhea. She had recurrent abdominal pain for 5 years prior to admission. On physical examination, generalized abdominal tenderness and a possible mass in the left upper quadrant were found. Urography on admission showed an enlarged left kidney with well preserved renal parenchyma. Calyceal crescents were shown in the early phase of urography (Fig. 4 *B*; and 9*A*). The hydronephrosis was due to pelviureteric junction obstruction.

A Hines-Anderson pyeloplasty was performed to relieve the ureteropelvic junction obstruction. Following operation, the drainage of the left renal pelvis was adequate and with subsequent urographic examinations, gradual realignment of the distorted collecting tubules was documented over a period of 6 years (Fig. 9, *B* and *C*).

Pathologic findings supported the diagnosis in all patients who had nephrectomy.

DIFFERENTIAL DIAGNOSIS

The nephrographic effect of increase in density of renal parenchyma contrasted against the water density of the urine in the unopacified distended collecting system may resemble the calyceal crescent sign. However, the nephrographic density is less than that produced by the crescent sign and the crescent shadows are usually separated from the renal margin by recognizable renal tissue partially opacified by the nephrographic effect. Thus, the calyceal crescent is almost always surrounded or followed by a nephrogram. When there is doubt, therefore, it is likely that one is not encountering the calyceal crescent sign, but rather thinned renal parenchyma. Another entity which must be distinguished from the calyceal crescent sign is that produced by total body opacifications—a common and important finding in infants in whom high dose urography is used, as first pointed out by O'Connor and Neuhauser.[11] This phenomenon manifests itself by increase in opacification of all body tissue which is vascularized, and, conversely, by nonopacification of nonvascularized masses or fluid collections. Thus a cystic, fluid-filled mass may, in the course of excretory urography, show thin strands of vascularized tissue which have become increased in density as part of a "total body opacification" and are contrasted against the nonvascularized fluid content of the cyst. This phenomenon is of particular importance in differential diagnosis of the so-called multicystic kidney, which often presents as a nonfunctioning flank mass replacing or simulating the kidney in the newborn period.

In diagnosis and differential diagnosis, a further possibility must be emphasized. A

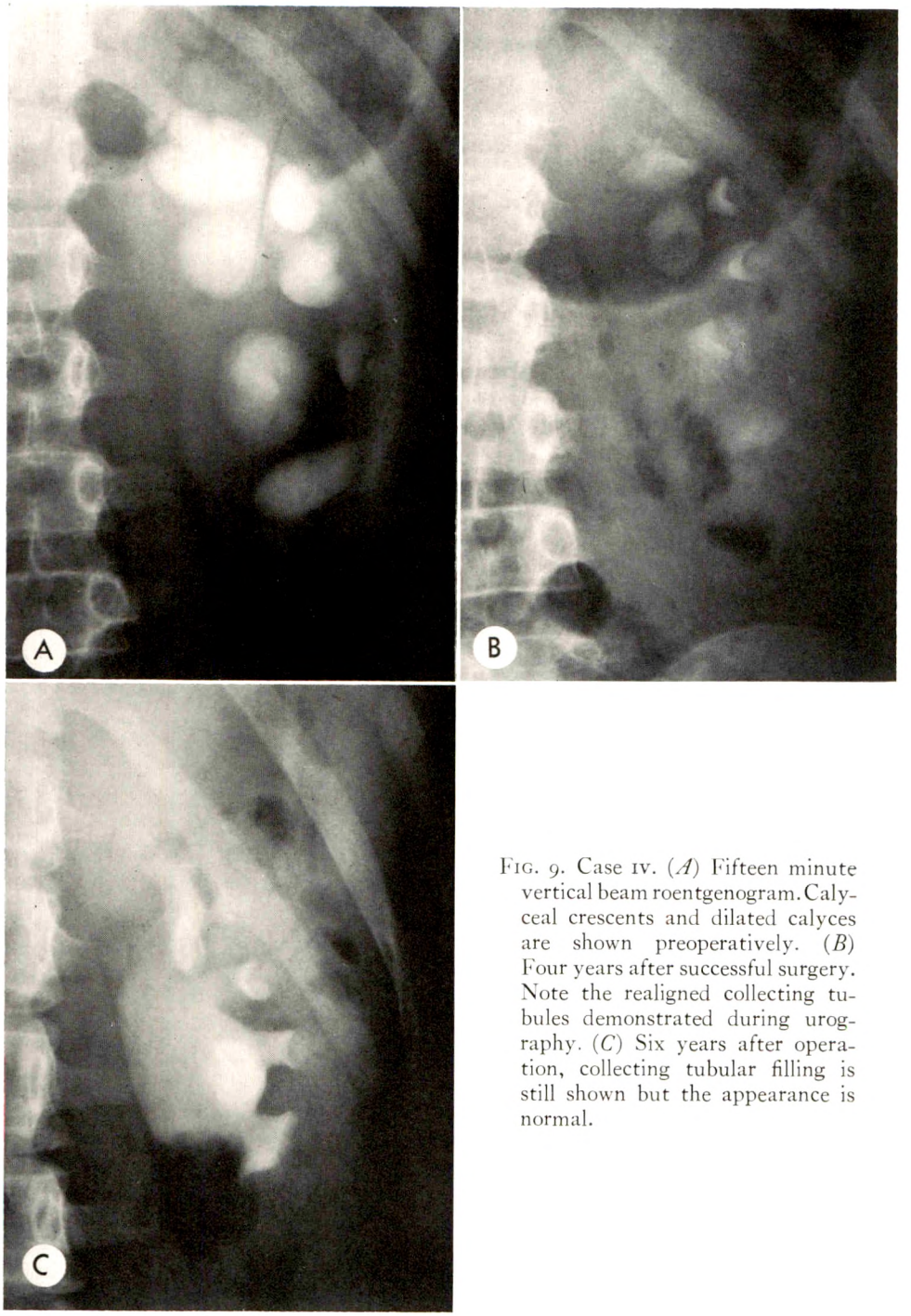


Fig. 9. Case IV. (*A*) Fifteen minute vertical beam roentgenogram. Calyceal crescents and dilated calyces are shown preoperatively. (*B*) Four years after successful surgery. Note the realigned collecting tubules demonstrated during urography. (*C*) Six years after operation, collecting tubular filling is still shown but the appearance is normal.

renal tumor such as Wilms' tumor may, in the course of invading and destroying the kidney, cause obstruction of all or part of the upper collecting system, and thus may produce localized calyectasis; during excretory urography the calyceal crescent sign may appear. It should be evident that in any case of suspected or demonstrated hydronephrosis involving part or all of the collecting system, the

investigation should be so complete as to define not only the hydronephrotic kidney but the cause of the hydronephrosis.

Overlying gas shadows having crescentic margins or interfaces might be mistaken for calyceal crescents.

SUMMARY

"Calyceal crescents" are the distorted collecting tubules filled with concentrated contrast medium during the early phase of urography in hydronephrosis due to obstruction.

Following adequate drainage of the collecting system, return to normal collecting tubular alignment may be documented.

The recognition of "calyceal crescents" is helpful in diagnosing hydronephrosis secondary to obstruction (functional or mechanical). The differential diagnosis is discussed.

A few pressure measurements before surgery and urography, following drainage of the collecting system, strongly suggest that for occurrence of the "crescent" phenomenon the resting intrapelvic pressure must be increased.

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RADIOLUCENT FOLDS IN THE UPPER URINARY TRACT*

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RADIOLUCENT folds or striations of the renal pelvis and ureters as a sign of urinary tract inflammation were first described in 1963.[9] Sporadic mention has since been made of this roentgenographic finding and its association with acute or chronic upper urinary tract infection.[2,5,7,10] Despite the fact that the pathologic significance of folds has recently been questioned,[3] we feel that their presence is a reliable roentgenographic finding of chronic upper urinary tract infection, correlating well with underlying pathologic changes. Their significance is further enhanced by the fact that in our experience they have proven to be potentially reversible changes that frequently precede the irreversible cortical scars which heretofore have been considered the earliest roentgenographic manifestations of chronic infection.[6]

METHOD

The only requisite for inclusion in this study was the demonstration of radiolucent folds in the calyces, infundibula, pelves or ureters by intravenous pyelography. Forty-five cases have been studied since 1962, the majority in the last 4 years. The complete medical record of each patient was reviewed, including history, laboratory data, roentgenographic procedures, and surgical and pathologic specimens when available. An attempt was made to evaluate the results of therapy from a roentgenographic approach whenever follow-up studies were available.

RESULTS

The 45 patients in this series all demonstrated smooth longitudinal radiolucent filling defects (folds) in the calyces, infundibula, pelves and/or ureters, that could best be identified with partial filling, particularly on the prone or drainage roentgenogram. With careful inspection folds could frequently be demonstrated to extend the entire length of the collecting system. The incidence of the finding cannot be estimated on the basis of this study as no attempt was made to review all intravenous pyelograms, or even those obtained specifically for evaluation of urinary tract infection.

All of the patients in the study were children, the oldest 16 years, the youngest 1 month of age. Only 2 patients were over 12 years of age, the average age was 5 years, and 70 per cent of the patients were female. All 45 patients had urinary tract infection documented by history and laboratory findings including cultures. The most common organisms cultured in decreasing order of frequency were *E. coli*, *Klebsiella aerobacter*, *Proteus* and mixed infections. We have not observed folds in any patient evaluated for purposes unrelated to infection in the urinary tract.

Two of the 45 patients had a history compatible with a urinary tract infection for the first time. Both were confirmed by cultures, treated medically and lost to follow-up. One of these patients was a 1 month old infant with sepsis in whom the infection was undoubtedly acute. The remaining patient had cortical scarring suggestive of chronic infection which could not be documented.

Forty-three patients in the study had

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TABLE I

PATIENTS WITH CHRONIC URINARY TRACT INFECTION

	Total Patients (43)	Folds Only (15)	Folds Plus C.A.P.* (28)
Cultures	42/43	14/15	28/28
positive	37/42 (88%)	13/14 (93%)	24/28 (86%)
negative	5/42	1/14	4/28
Negative Cultures on Therapy	5/5	1/1	4/4
Cystography	40/43	12/15	28/28
reflux	39/40 (96%)	12/12 (100%)	27/28 (96%)
normal	1/40	0/12	1/28
bilateral	19/40 (47%)	7/12 (58%)	12/27 (44%)
Surgery	25/43	5/15	20/28
pathology	22/25 (88%)	4/5 (80%)	18/20 (90%)
negative	3/25	1/5	2/20

* C.A.P. = chronic atrophic pyelonephritis.

documented chronic or recurrent urinary tract infection, the majority of them having been referred for further evaluation following a diagnostic work-up and therapeutic trial at other institutions. The success with which persistent infection and vesicoureteral reflux has been managed at our institution prompted these referrals, providing us with the unique opportunity of studying a large number of children with this problem. A composite of the pertinent findings is listed in Table 1.

Cultures were obtained on all but 1 patient, 38 in our own institution and 4 elsewhere. Thirty-seven of the 42 cultures were positive, and the 5 patients with negative cultures were all on antibiotic therapy at the time the specimens were collected.

Forty of the 43 patients had cystourethrography in addition in intravenous pyelography, all but 3 of the examinations having been performed in our own institution. Thirty-nine of the 40 patients demonstrated vesicoureteral reflux which was bilateral approximately one-half of the time. With unilateral reflux there was a slight preponderance on the right side. In each instance the radiolucent folds and the vesicoureteral reflux were ipsilateral except for 2 patients in whom bilateral reflux was demonstrated but only unilateral radiolucent folds were evident.

All 43 patients were treated medically for infection. Because of persistent infection and vesicoureteral reflux, 25 patients were subjected to ureteral reimplantation. The resected ureteral segments provided the material for pathologic correlation. Evidence of variable degrees of inflammation was found in 22 of the 25 specimens. This was characterized by slight to moderate edema of the ureteral tunica propria and the infiltration of chronic inflammatory cells in the tunica and occasionally in the interstices of the muscularis. The muscularis showed variable degrees of hypertrophy which was interpreted as secondary to reflux. Contraction of muscle of the resected segment produced longitudinal invagination of the edematous mucosa. These are best demonstrated in cross sections (Fig. 3, *A–D*), and are presumed to represent the

Table II

PATIENTS WITH FOLLOW-UP EXAMINATIONS

	Total Patients (26)	Folds Only (8)	Folds Plus C.A.P.* (18)
Improvement	12/26 (46%)	3/8 (38%)	9/18 (50%)
Progression	14/26 (54%)	5/8 (62%)	9/18 (50%)
Medical Management Only	7	5	2
improvement	1/7 (14%)	1/5 (20%)	0/2 (0%)
progression	6/7 (86%)	4/5 (80%)	2/2 (100%)
Combined Medical-Surgical Management†	19	3	16
improvement	11/19 (58%)	2/3 (67%)	9/16 (56%)
progression	8/19 (42%)	1/3 (33%)	7/16 (44%)

* C.A.P. = chronic atrophic pyelonephritis.
† Surgical management implies ureteral reimplantation.

pathologic corollary of the folds seen roentgenographically.

Fifteen of the 43 patients had radiolucent folds as the only roentgenographic abnormality, whereas 28 of the 43 patients had coexistent changes indicative of chronic atrophic pyelonephritis. These well documented changes consisted of lack of normal growth and development of the affected kidney, often with hypertrophy of the uninvolved kidney or portion thereof; focal areas of cortical atrophy with eventual smooth clubbing of the adjacent calyx as the atrophy progressed; uneven distribution of the focal areas of scarring in generalized atrophy; asymmetric involvement when the disease was bilateral except when the process was very severe; and symmetric concentration.[6]

A comparison of the two groups, those with radiolucent folds as the only demonstrable abnormality as opposed to those with coexistent chronic atrophic pyelonephritis, reveals no appreciable difference in either history, culture, reflux, or pathologic confirmation (Table I).

Twenty-six of the 43 patients had follow-up examinations which enabled roentgenographic evaluation of therapeutic results, an adequate time interval of at least 1 year from the initial study having been arbitrarily chosen. Roentgenographic improvement implies diminution or complete disappearance of radiolucent folds, elimination of further cortical scarring and resumption of normal growth of the involved kidney (Table II). Of particular interest in this group of 26 patients are those in whom radiolucent folds were the only abnormality initially demonstrable by intravenous pyelography. There were 8 such patients, 3 of whom improved roentgenographically, with complete disappearance of folds, and 5 of whom progressed roentgenographically with the development of focal cortical atrophy. Thus it is apparent that radiolucent folds are potentially reversible roentgenographic findings that not infrequently precede the development of chronic atrophic pyelonephritis.

Some illustrative cases will be presented.

Report of Cases

Case 1. A.S., a 10 year old white male, was admitted for evaluation of left flank pain and dysuria. He had had multiple documented urinary tract infections in the past, all treated medically, and was on antibiotic therapy at the

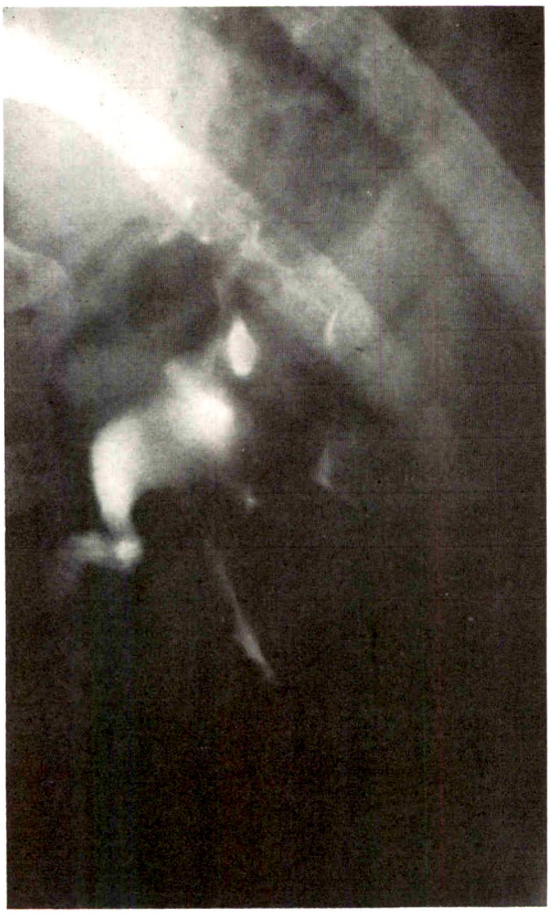


FIG. 1. Case I. Intravenous pyelogram. The left kidney is of normal size and the cortex of normal width. There is no evidence of focal scarring or calyceal clubbing, but the pelvis and ureter contain multiple linear radiolucent filling defects which parallel their long axis. The margins of the pelvis and ureter are slightly serrated in appearance.

time of admission. Physical examination was unremarkable except for increased severity of the left flank pain on deep palpation. Cystoscopy revealed changes consistent with chronic cystitis. Cystourethrography revealed Grade 2 vesicoureteral reflux on the left side and intravenous pyelography showed multiple smooth longitudinal radiolucent filling defects or folds in the left upper collecting system and ureter (Fig. 1); the left kidney was otherwise normal roentgenographically. No abnormalities of the right kidney or ureter were demonstrated. Laboratory studies on this admission were within normal limits. The patient was discharged on a medical regimen under the supervision of his private physician.

Comment. Reflux is graded on the basis of its severity as follows: Grade 1, transient, intermittent reflux into the distal ureter without associated dilatation or loss of peristalsis; Grade 2, reflux into the upper collecting system of the kidney without associated dilatation or loss of peristalsis; Grade 3, reflux into the upper collecting system which is associated with dilatation but peristalsis, although it may be diminished, is still preserved; and Grade 4, the most severe reflux is usually massive, the bladder and ureter sometimes acting as one continuous reservoir, the collecting system is dilated, the ureter is often tortuous and kinked, and peristalsis is absent. In each instance in which we have observed radiolucent folds in the upper urinary tract, infection and vesicoureteral reflux have been associated findings.

CASE II. I.V., a 7 year old white male, was admitted with a history of recurrent urinary tract infections since the first year of life. Bilateral vesicoureteral reflux, Grade 3, had been demonstrated 1 year prior to admission, along with chronic atrophic pyelonephritis. Dysuria was noted 1 month prior to admission, with the subsequent development of chills and fever. Physical examination, aside from a low grade fever, was negative. Five to 10 white blood cells and 10 to 12 red blood cells per high power field were noted in the urine, from which *Proteus vulgaris* (over 100,000 organisms) was cultured. Grade 3 vesicoureteral reflux was still present bilaterally and radiolucent folds could be identified the entire length of both collecting systems on cystography as well as intravenous pyelography (Fig. 2). Bilateral ureteral reimplantations were performed. The resected ureteral segment displayed marked polypoid edema of the tunica propria with a sparce scattering of chronic inflammatory cells here and in the markedly hypertrophied muscularis (Fig. 3*A*).

Comment. The marked exaggeration of mucosal folds and the extreme muscular hypertrophy are quite evident when this ureter is compared with a normal ureter removed at autopsy from a child of the same age (Fig. 3*B*). The resected ureteral segments of Case III (Fig. 3*C*) and Case V (Fig. 3*D*) are illustrated in the same compos-

ite arrangement to facilitate comparison with the normal.

Case III. K.K. was initially admitted at the age of 5 years with a history of intermittent episodes of fever, chills, abdominal pain, frequency and dysuria over a $3\frac{1}{2}$ year interval. Antibiotic therapy had been employed with each symptom flare-up. Physical examination revealed only suprapubic discomfort to palpation. Laboratory findings on admission were normal. Cystography, however, revealed Grade 2 vesicoureteral reflux bilaterally, and intravenous pyelography demonstrated bilateral radiolucent folds as the only abnormality in the upper urinary tract. The patient was discharged on a medical regimen to be readmitted the following summer for bilateral ureteral reimplantations. Cystography and pyelography on the second admission (7 months later) revealed changes similar to those noted initially, but in addition focal areas of cortical scarring were apparent in both kidneys for the first time (Fig. 4). Ureteral reimplantation was done bilaterally at this time. The resected short ureteral segments displayed concentric hypertrophy of the ureteral muscularis with moderate mucosal edema and polypoid invagination (Fig. 3*C*).

Follow-up examinations in the second, sixth, and sixteenth postoperative months revealed no evidence of vesicoureteral reflux and gradual diminution of the radiolucent fold. Two years following surgery there was again no evidence of reflux and the radiolucent folds had completely disappeared (Fig. 5). Focal areas of cortical scarring persisted without change, and the kidneys had undergone normal growth and development, which is probably conclusive proof of the success of long term therapy. The patient had remained asymptomatic.

Comment. This patient is 1 of 16 children who were treated by the combined medical-surgical approach in the presence of folds and atrophic pyelonephritis (Table II). Nine such patients have shown improvement by the criteria previously outlined, of which this is a typical example.

Case IV. K.B., a white female, was seen in our outpatient department at the age of 5 years. She had received intermittent medical therapy for recurrent urinary tract infections throughout most of her life. Cystography at that time revealed Grade 2 vesicoureteral reflux on the left side only, and intravenous pyelography showed radiolucent folds in the left collecting system without associated cortical or calyceal abnormalities. The right kidney and ureter were normal (Fig. 6). She was discharged to the care of her private physician on long term medical therapy. Urine samples remained sterile and the child was asymptomatic over the next 1 year interval. When re-evaluated roentgenographically, reflux was still evident on the left side. Intravenous pyelography, however, revealed continued growth of the left kidney without the development of atrophic pyelonephritis, and the radiolucent folds were no longer demonstrable (Fig. 7).

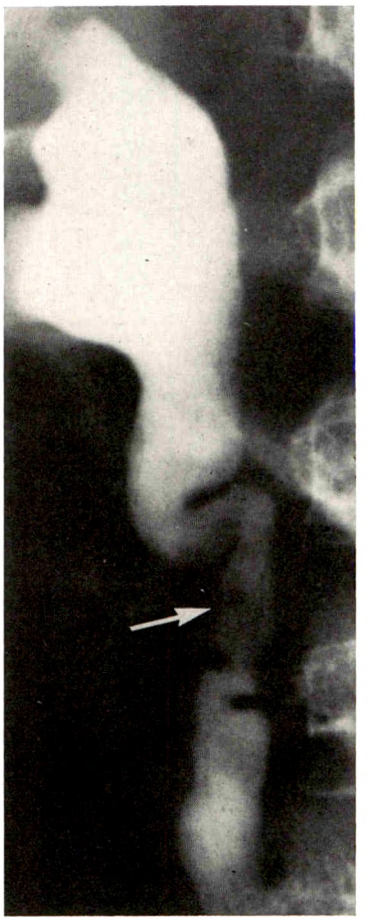


Fig. 2. Case II. Cystogram. There is moderate dilatation of the right pelvis and ureter. Multiple linear radiolucent defects paralleling its long axis can be seen within the tortuous ureter. The smooth round radiolucency (arrow) is an air bubble inadvertently introduced during cystography which refluxed into the ureter along with the contrast material. Similar linear filling defects were evident in the left ureter.

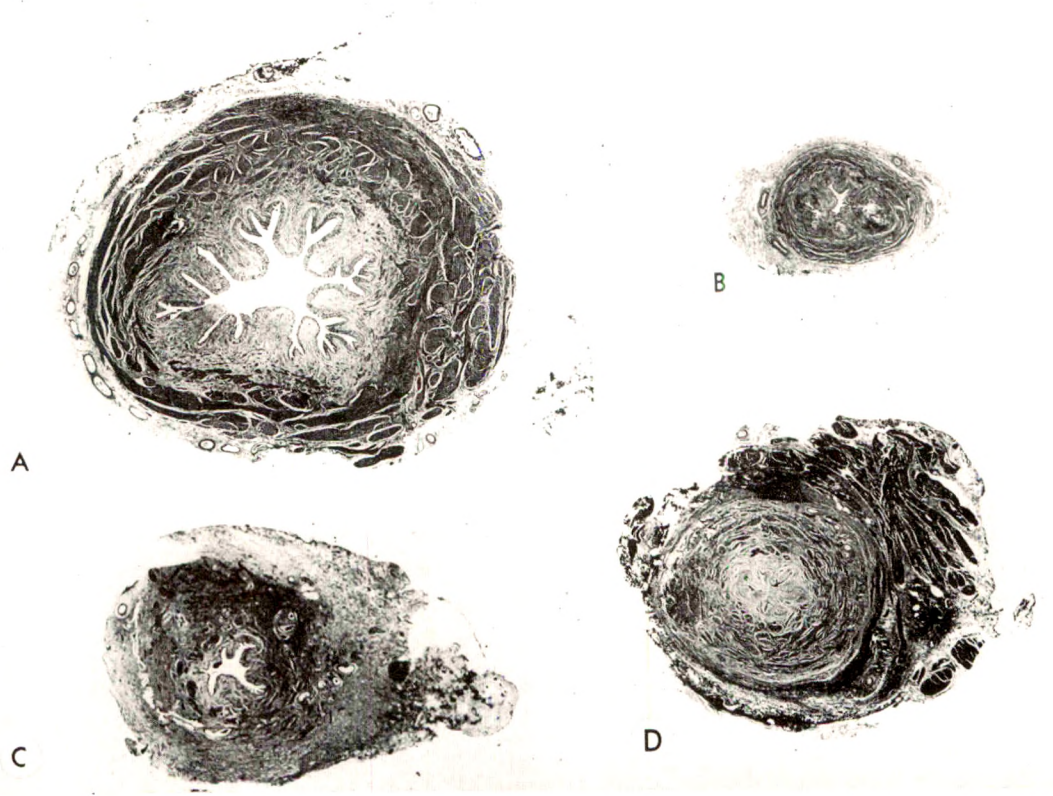


FIG. 3. Photomicrographs of resected ureters. (*A*) Case II. (*B*) Normal control. (*C*) Case III. (*D*) Case V. Polypoid edematous invagination of the mucosa, responsible for the longitudinal striations seen roentgenographically. Marked muscular hypertrophy is also evident as well as periureteral fibrosis in *C*. (11×. Masson–trichrome stain.)

Comment. This is the one patient in this group of chronically infected children who demonstrated improvement by roentgenographic criteria on a purely medical regimen (Table II). The reversibility of these roentgenographic folds is again emphasized.

CASE V. E.A., a white male, was initially evaluated at the age of 2½ years because of intermittent fever and dysuria. Urinalysis at that time was positive with 20 to 30 white blood cells and 5 to 10 red blood cells per high power field. The leukocyte count was 15,400 with a shift to the left. Blood cultures were negative but over 100,000 organisms per ml. of *Proteus vulgaris* were grown from the urine. Cystourethrography at that time revealed Grade 3 vesicoureteral reflux bilaterally, the intravenous pyelogram was normal (Fig. 8). The patient was treated medically with symptomatic improvement, and on re-evaluation 3 months later Grade 2 reflux was demonstrated on the left side only. Intravenous pyelography at that time showed minimal radiolucent folds in the left upper collecting system without cortical or calyceal changes (Fig. 9). After 6 months on continuous drug therapy the roentgenographic changes were unchanged except for the suggestion of early cortical scarring of the left kidney. Twelve months following the initial examination there was persistent Grade 2 reflux on the left side. Intravenous pyelography, on the other hand, revealed more prominent folds within the collecting system of the left kidney, with definite focal areas of cortical atrophy (Fig. 10). Throughout this interval the patient had been on continuous drug therapy and the right kidney was normal. The left ureter was reimplanted at that time. The resected ureteral segment displayed a significant degree of periureteral fibrosis with an infiltration here and in the mucosa of chronic

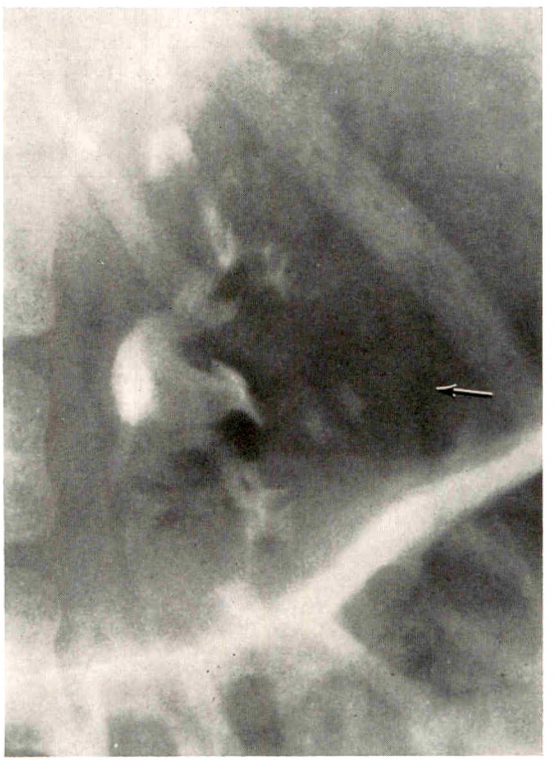


Fig. 4. Case III. Intravenous pyelogram. Multiple radiolucent folds can be identified in the infundibula, pelvis, and proximal ureter of the left kidney. A small focal area of cortical atrophy can be seen on the anterolateral aspect of the kidney (arrow). More obvious cortical atrophy is seen in the upper pole with associated calyceal clubbing. Similar changes were present in the right kidney.

inflammatory cells (Fig. 3*D*). Muscular hypertrophy was less marked than in the other cases but polypoid edema of the mucosa was quite evident.

Comment. This patient was lost to follow-up after his surgical procedure. He has been included, therefore, among the 5 patients who initially demonstrated only folds roentgenographically and showed progressive renal deterioration on medical management alone (Table II). This represents a typical example of the radiolucent folds preceding the development of cortical atrophy in chronic urinary tract infection.

Case VI. J.P., a white female, was initially evaluated at the age of $3\frac{1}{2}$ years for urinary tract infection. Both kidneys and ureters were completely duplicated. Grade 3 vesicoureteral reflux into the lower poles of the duplex systems was present bilaterally. Chronic atrophic pyelonephritic scarring of both lower poles was evident along with prominent radiolucent folds within the collecting systems. Despite aggressive medical and surgical treatment which included bilateral ureteral reimplantations on two separate occasions, persistent reflux into both lower poles and radiolucent folds had persisted over a 5 year interval (Fig. 11). Progressive scarring of both lower poles had taken place with compensatory hypertrophy of the normal upper poles bilaterally (Fig. 12). Minimal dilatation of the ureters draining the

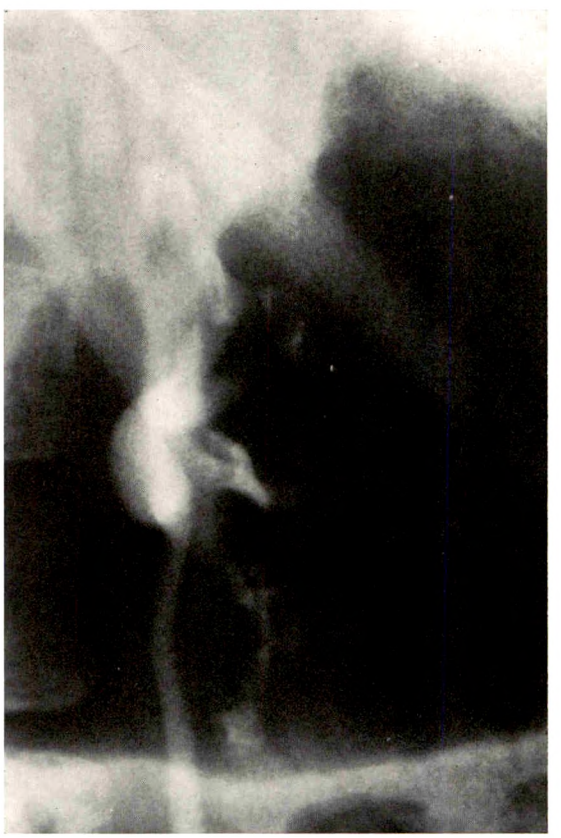


Fig. 5. Case III. Intravenous pyelogram. Two years after ureteral reimplantation the kidneys show evidence of commensurate growth and development with the child. Only the left kidney is utilized for illustration, although improvement was present bilaterally with complete disappearance of the radiolucent folds. The focal pyelonephritic scars persist without progression.



Fig. 6. Case iv. Intravenous pyelogram. Multiple radiolucent folds are present within the left pelvis and proximal ureter. The calyces and renal cortex are normal roentgenographically.



Fig. 7. Case iv. Intravenous pyelogram. After 1 year of medical therapy the left kidney is normal roentgenographically. No folds are identifiable.



Fig. 8. Case v. Intravenous pyelogram. Both kidneys were normal roentgenographically on the first examination, the left is shown for illustration. There is minimal tortuosity of the proximal left ureter.

upper poles had persisted after the first reimplantation. Microscopic examination of the resected ureteral segments from the second reimplantation revealed both ureters to be enclosed within the same sheath (Fig. 13). The ureter from the upper, *i.e.*, normal, pole shown on the left (Fig. 13) displayed minimal mucosal

edema and invagination but to an insignificant degree as compared to the lower pole ureter on the right. Here massive invagination of the mucosa is apparent associated with marked hypertrophy of the muscular coat.

Comment. This patient is included because of the excellent demonstration of the striking roentgenographic and histologic difference between the normal and the chronically infected kidney and collecting system, not only within the same patient but within the same organ system bilaterally.

DISCUSSION

The criteria by which the diagnosis of chronic atrophic pyelonephritis can be made roentgenographically have been well documented.[6] These changes consist primarily of focal areas of cortical scarring associated with clubbing of adjacent calyces. Despite the frequency with which this disease is encountered in children, there is a paucity of early roentgenographic signs which might enable one to establish a definitive diagnosis before the onset of irreversible cortical scars. Little mention has been made in the past of the changes found in the calyces, infundibula, pelves or ureters in the presence of chronic infection. We are persuaded that many children with vesicoureteral reflux and chronic infection do in fact show quite definite ureteral and pelvic changes in the form of prominent longitudinal radiolucent filling defects as identified by intravenous pyelography. Examination of resected specimens in which these folds have been demonstrated reveals a thickened ureter with an edematous mucosal pattern.[9] Microscopically we have confirmed the presence of an edematous and thickened mucosa. The infiltration

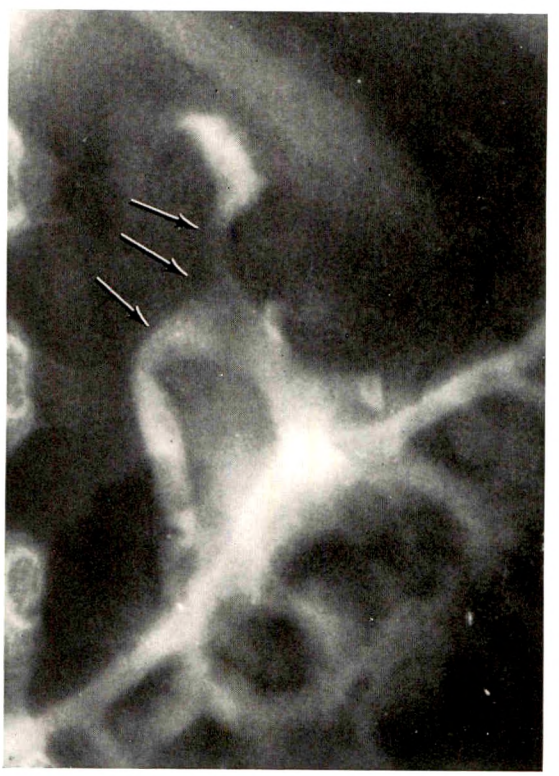


Fig. 9. Case v. Intravenous pyelogram. After 3 months of medical therapy in the face of persistent left vesicoureteral reflux, the first evidence of radiolucent folds can be found in the upper infundibulum and pelvis of the left kidney (arrows). No calyceal or cortical abnormalities can be identified.

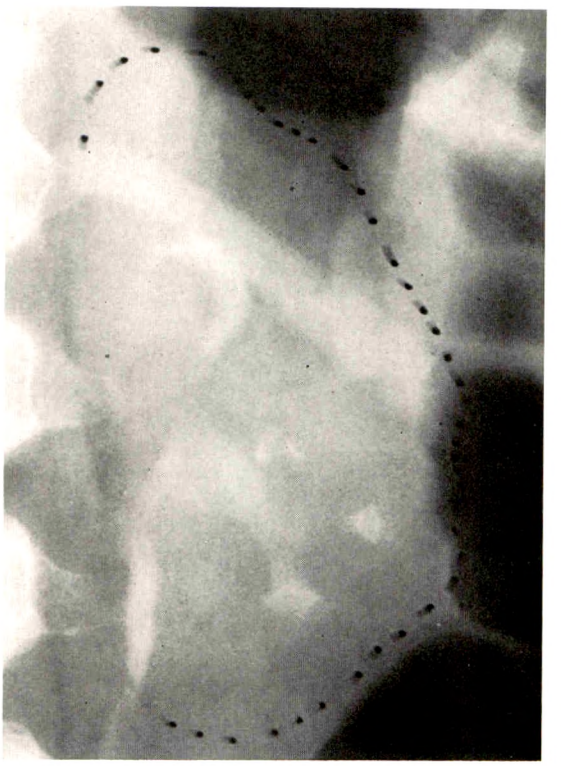


Fig. 10. Case v. Intravenous pyelogram. After 1 year of medical management multiple focal areas of cortical atrophy are present (re-touched) with early calyceal changes. Prominent radiolucent folds are evident throughout the entire length of the collecting system.

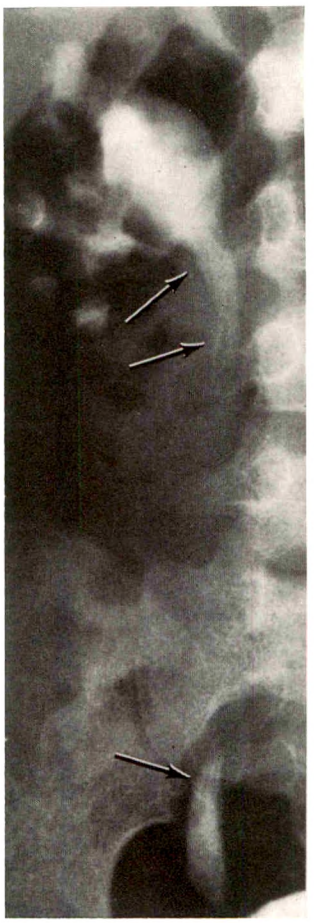


FIG. 11. Case VI. Cystogram. Persistent vesicoureteral reflux into both lower poles is present 5 years after the initial examination; the right is shown for illustration. The calyces are blunted and the pelvis is dilated. Longitudinal radiolucent folds are present throughout the entire length of the ureter (arrows).

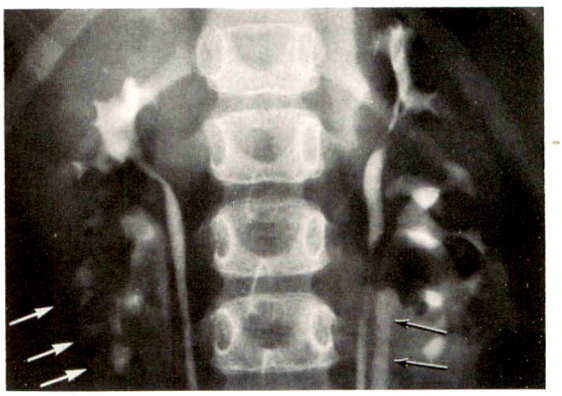


FIG. 12. Case VI. Intravenous pyelogram. Both collecting systems are duplicated completely. Severe chronic atrophic pyelonephritis is obvious in both lower poles of the duplex systems (left arrows) with compensatory hypertrophy of the upper halves of the duplex systems. Radiolucent folds are present in the lower pelves bilaterally (right arrows), although less apparent in this illustration than are those within the ureter (Fig. 11).

of chronic inflammatory cells in the tunica and occasionally in the interstices of the muscularis has been identified, the predominant cell type in all cases examined being the lymphocyte. Hypertrophy of the ureteral muscularis is a constant finding. It is felt that the thickened, polypoid, edematous and invaginating mucosa as well as the hypertrophy of the underlying muscularis are the major contributing factors responsible for the radiolucent filling defects or folds that are seen roentgenographically.

These radiolucent folds have only been demonstrated in children with urinary tract infection and vesicoureteral reflux in our experience. They can occur in the absence of other roentgenographic criteria of pyelonephritis which are by their definition chronic in nature.[6] We have demonstrated that folds are potentially reversible roentgenographic changes, and that their presence can precede the development of chronic atrophic pyelonephritis.

The differential diagnosis of radiolucent filling defects in the collecting system should

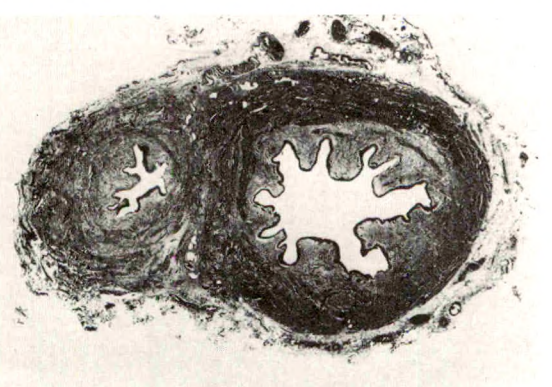


FIG. 13. Case VI. Photomicrograph of double ureter. The right ureter displays marked polypoid invagination and marked muscular hypertrophy. It apparently drained the lower pole of the kidney, whereas the left ureter showing very minimal change drained the upper pole. For size compare with Figure 3*B*. (11 ×. Masson—trichrome stain.)

include pyelitis or ureteritis cystica and renal leukoplakia, both of which are relatively rare but do occur in children.[1,4,8] Pyeloureteritis cystica is characterized by multiple small subepithelial cysts which produce filling defects which are oval in shape and marginate the mucosa of the renal pelvis or ureter.[8] These should not be confused with the linear longitudinal filling defects which we have described. The filling defects of leukoplakia are usually more irregular than the smooth linear folds we have found, and the common occurrence of urinary tract infection as opposed to the extreme rarity of leukoplakia in children is of considerable help in distinguishing the 2 entities.[1,4]

CONCLUSION

The intravenous pyelogram of any child who presents with urinary tract infection and vesicoureteral reflux should be carefully scrutinized for the presence of radiolucent filling defects or folds in the renal pelvis and ureter. These folds indicate the presence of chronic inflammatory changes, changes that are potentially reversible roentgenographically. One should be alerted to the fact that such a child is a candidate for atrophic pyelonephritis and aggressive treatment should be directed towards the hopeful prevention of irreversible cortical scarring.

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URETERAL ECTOPIA INTO A WOLFFIAN DUCT REMNANT (GARTNER'S DUCTS OR CYSTS) PRESENTING AS A URETHRAL DIVERTICULUM IN TWO GIRLS*

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URETHRAL diverticula are common, acquired lesions in multiparous females, but are probably nonexistent as congenital lesions in little girls. In Williams' experience, "a urethral diverticulum in a girl has been the lower end of an ectopic ureter, the upper end of which is narrow or actually obliterated."[2]

Two cases of ureteral ectopia in apparent urethral diverticula are the object of this report. An explanation of this peculiar presentation based on developmental anatomy is suggested.

REPORT OF CASES

CASE I. (J. H. H. No. 1130745). Baby girl J. Q. was born September 28, 1964, the product of an uncomplicated pregnancy and delivery. In December, 1964 the patient was examined in the Outpatient Department because of recurrent fevers. A urinary tract infection was found to be the cause of the fevers. An excretory urogram obtained on December 11, 1964, was of poor quality. A voiding cystourethrogram 4 days later (Fig. 1) showed an apparent diverticulum on the posterior aspect of the urethra. Because antibiotic treatment was ineffective, the lower urinary tract was reinvestigated March 25, 1965. During catheterization of the urethra the tip of the catheter entered the "diverticulum." Injection of an iodinated water soluble contrast agent in this locale demonstrated a widely dilated and tortuous ureter draining the upper pole of the left kidney into the "diverticulum." Some of the contrast agent refluxed into the bladder and secondarily into the normal ureter draining the lower pole of the left kidney (Fig. 2; and 3). Review of the excretory urogram of December 11 showed that no intrarenal collecting system had been demonstrated in the upper pole of the left kidney. Subsequently, the patient underwent a left heminephrectomy and left ureterectomy to the level of the bladder. In September, 1968, because of recurring urinary tract infection, the distal stump of the ectopic left ureter was excised. Urinary tract infections have not recurred since then.

CASE II. (UCMC No. 348343). Baby girl B. was born on July 5, 1969, the product of an uncomplicated pregnancy and delivery. Shortly after birth an imperforate anus with rectoperineal fistula was discovered. Investigation of this patient's urinary tract was undertaken to define possible associated anomalies. An excretory urogram on the day of birth disclosed a low ectopic right kidney and a dilated proximal collecting system on the left side (Fig. 4, *A* and *B*). Voiding cystourethrography on the same day disclosed a "diverticulum" on the posterior aspect of the urethra (Fig. 5*A*). In order to demonstrate the connection between the dilated left collecting system and the "diverticulum," the excretory urogram and the voiding cystourethrogram were repeated. On July 11th during excretory urography, the patient was fluoroscoped and the distal left ureter traced to a level below the trigone (Fig. 6, *A* and *B*). On July 17th a voiding cystourethrogram showed reflux of contrast agent from the "diverticulum" into the left ureter (Fig. 5*B*). No surgical procedures have yet been attempted on this child.

DISCUSSION

In each instance the ectopic ureter did not enter the urethra directly, rather it drained into an apparent diverticulum off the urethra. This is evident by the difficulty encountered in obtaining filling of this ectopic ureter, *not* at the posterior wall of the urethra, but at the apex of a "diverticulum."

A brief review of the embryologic devel-

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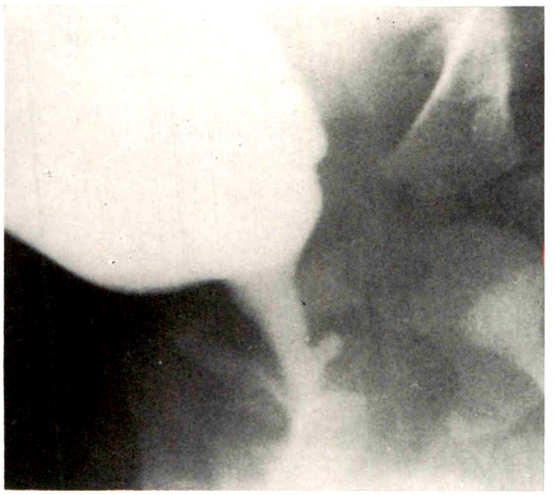


Fig. 1. Case 1. Voiding cystourethrogram, showing "diverticulum" projecting posteriorly from the mid urethra.

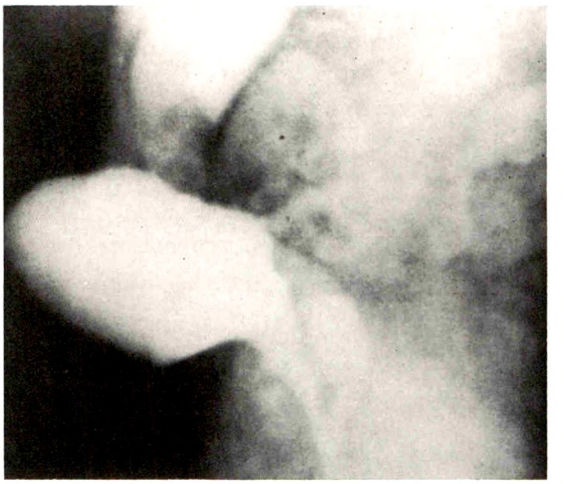


Fig. 2. Case 1. Repeat voiding cystourethrogram, showing dilated ureter filled from the "diverticulum."

opment of the ureter in its relationship with the wolffian duct[1] is necessary to discover an explanation for this finding. Figure 7, *A–G*, is a graphic representation of the evolutionary process to be discussed. The ureter originates as a cephalad growing bud from the lower end of the mesonephric duct (wolffian duct). Incorporation of the caudal portion of the wolffian duct into the posterior wall of the cloaca, while the latter is being divided by the downward growth of the urorectal septum, results in separation of the ureter from the wolffian duct. The ureter and the mesonephric duct have separate orifices entering the posterior wall of the primitive bladder. Subsequent differential growth of the urogenital sinus further separates the ureteral orifices from the wolffian duct orifices. In the male, the orifices of the wolffian ducts are the openings of the ejaculatory ducts at the lateral aspect of the verumontanum. In the female, the wolffian duct system degenerates and its remnants are known as Gartner's ducts or cysts.

When the ureteral bud originates from the mesonephric duct in an unusually high position, incorporation of the ureteral orifice into the posterior wall of the urogenital sinus is delayed. The ureteral orifice remains attached to the wolffian duct during



Fig. 3. Case 1. Lateral roentgenogram of the abdomen during repeat voiding cystourethrography. A widely dilated ureter drains the upper pole of the left kidney to a point below the bladder neck. A normal sized lower pole collecting system can be seen posterior to the proximal redundancy of the dilated ureter.

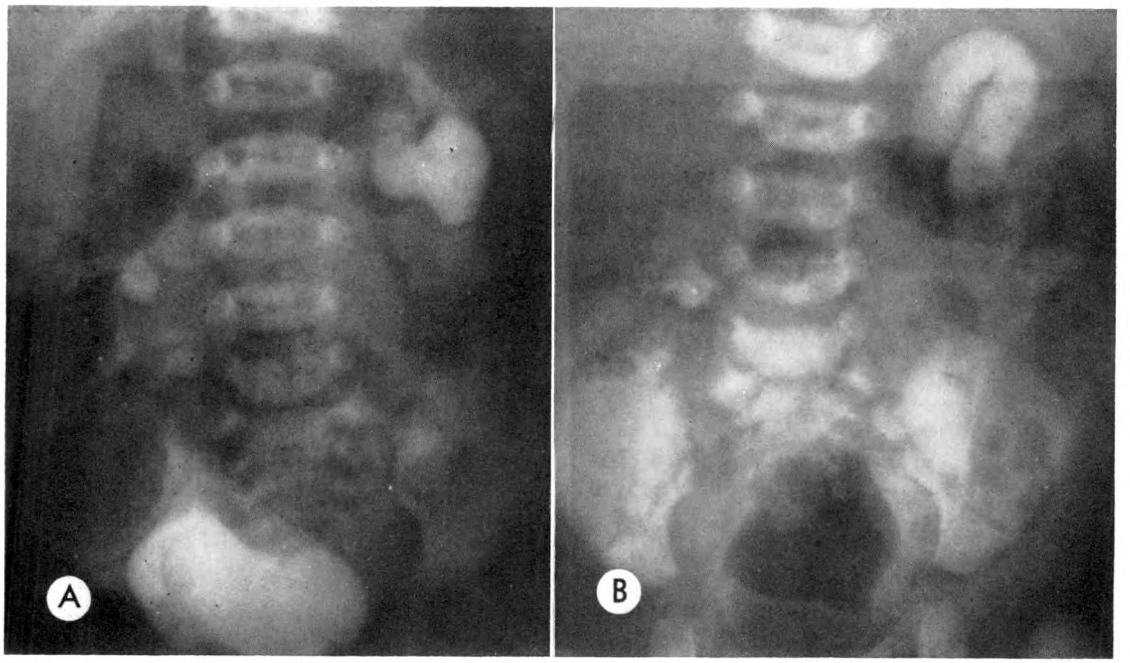


FIG. 4. Case II. Intravenous pyelograms on day of birth. (*A*) Anteroposterior projection. (*B*) Posteroanterior projection. The right kidney is low ectopic. The left kidney is drained by a single dilated ureter.

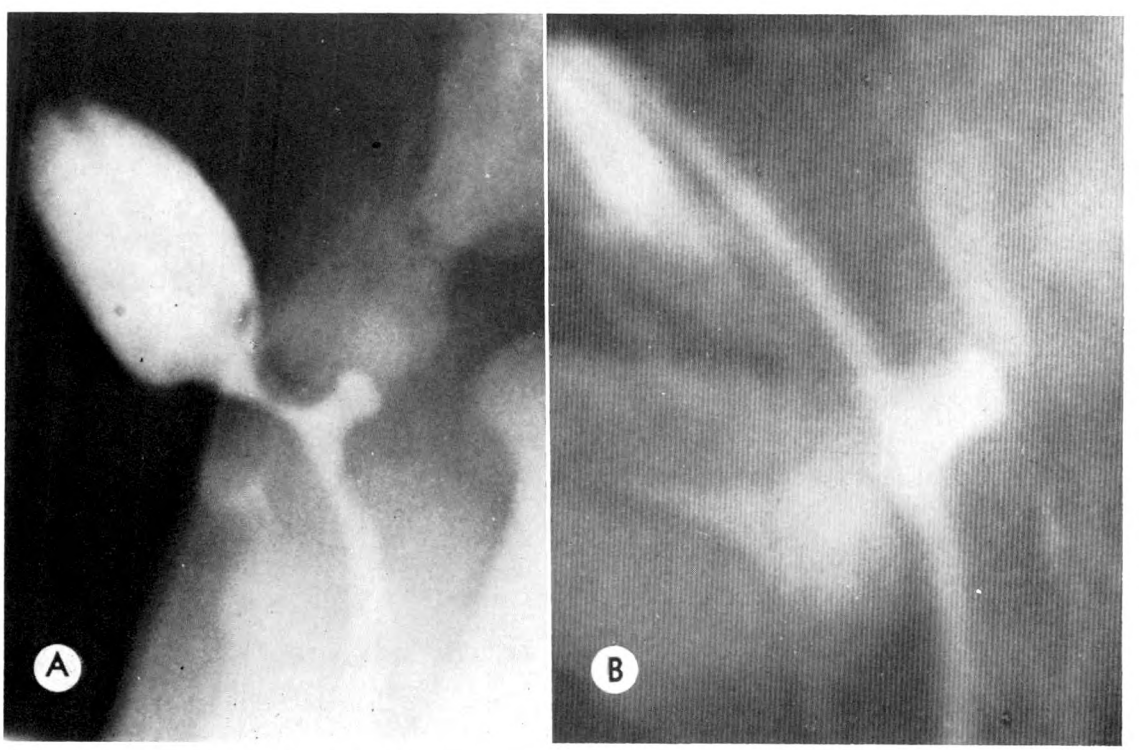


FIG. 5. Case II. (*A*) Voiding cystourethrogram on day of birth, showing "diverticulum" projecting posteriorly from the mid urethra. (*B*) Repeat voiding cystourethrogram, showing reflux from "diverticulum" into ureter.

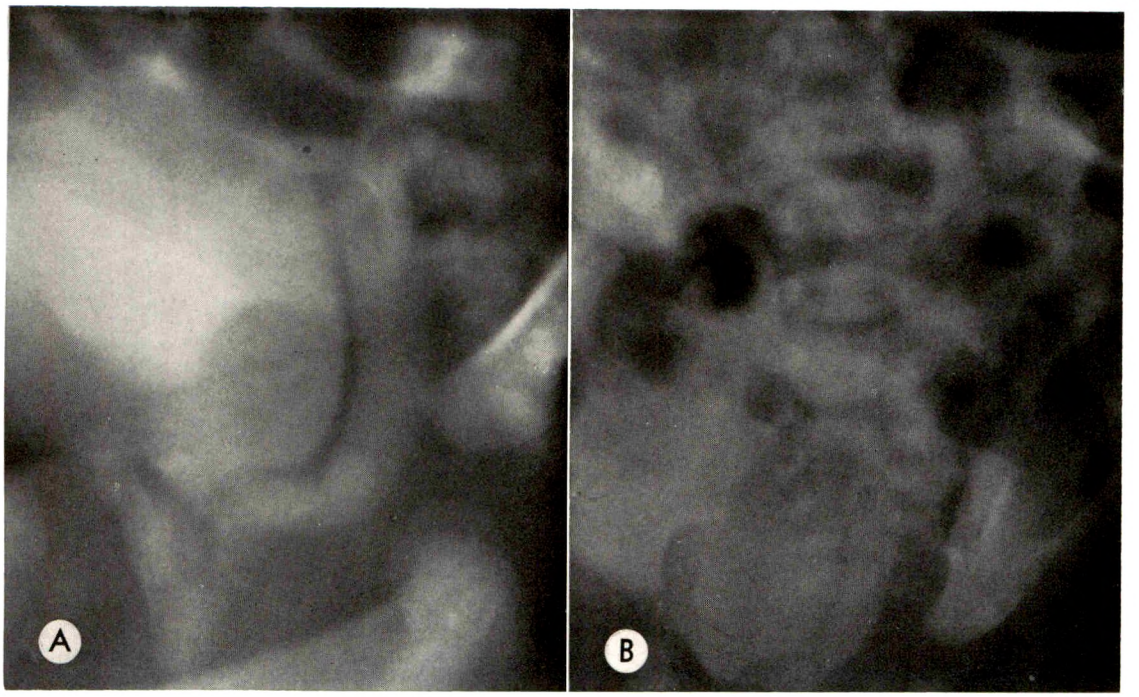


Fig. 6. Case 11. (*A* and *B*) Fluorographic spot films obtained during repeat excretory urography. The dilated left ureter clearly descends below the bladder neck.

the latter's downward growth and eventually obtains its own orifice in the urogenital sinus in a lower than normal position (single ectopic ureter).

In cases of ureteral duplication, 2 ureteral buds grow off the mesonephric duct. One bud (draining the lower renal pole) originates in a relatively normal position; the other (draining the upper renal pole) grows from a more cephalad position. The end result of this duplication is a lower pole ureter in normal position in the bladder and the second ureter opening at a point below and medial to the first orifice.

When a ureteral bud originates from an even higher point along the mesonephric duct, incorporation to the point of separation of the ureteral orifice from the wolffian duct becomes impossible. In the male, the ureter opens into the ejaculatory duct, the seminal vesicles or the vas deferens.

In the female, a similar high origin of the ureter off the mesonephric duct should logically result in a persistence of the caudal portion of the wolffian duct (Gartner's duct or cyst) with the ectopic ureter draining into it. A change from mesonephric duct to ureter at the apex of the "diverticulum" appears a likely explanation for the fact that the difficulty in retrograde filling of the ureter occurs at the apex of the "diverticulum."

In both patients the location of the "diverticulum" in the urethra is identical and compares with the position of the ejaculatory duct orifice in the male urethra.

We propose that the anomaly just described represents in the female the equivalent of a ureter opening in an ejaculatory duct in the male.

We were unable to find any *bona fide* reports of true congenital diverticula of the urethra in little girls. The demonstration of an apparent diverticulum should therefore induce the examiner to make every possible attempt to verify the presence of an ectopic ureter entering such a "diverticulum."

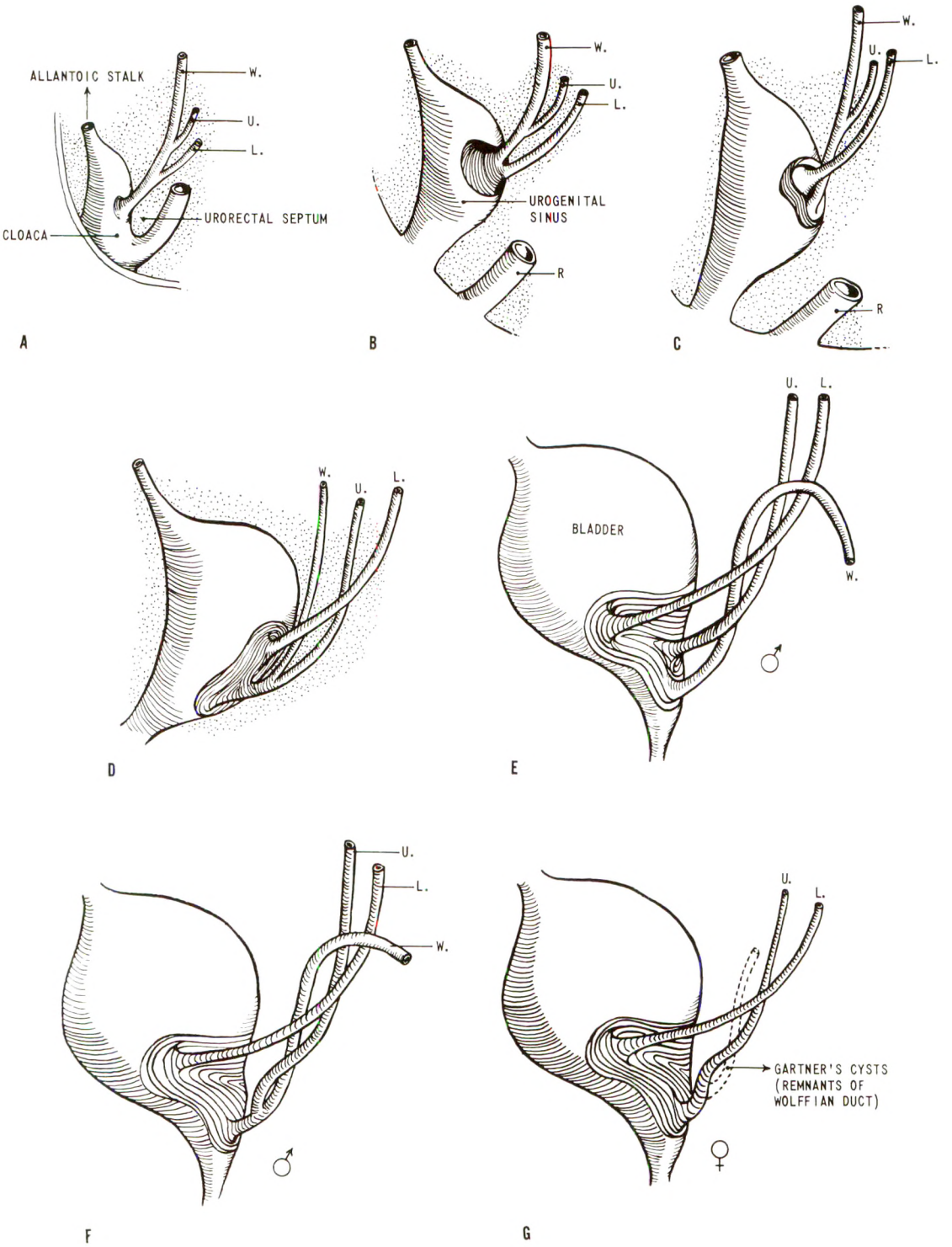

ALLANTOIC STALK
W.
U.
L.
UROGENITAL SINUS
UROGENITAL SINUS
R
CLOACA
UROGENITAL SINUS
UROGENITAL SINUS
R
A
B
C
W.
U.
L.
D
BLADDER
E
F
GARTNER'S CYSTS
(REMNANTS OF
WOLFFIAN DUCT)
G

Fig. 7. (*A*) Primitive stage: Two ureteral buds originate from the wolffian duct. Upper ureter would not be present in case of single normal ureter. It would be the only ureter present in case of single ectopic ureter. (*B*) The caudal end of the wolffian duct is being incorporated in the posterior wall of the urogenital sinus. (*C*) Incorporation of the caudal end of the wolffian duct has progressed to the point where the lower ureter has gained a separate orifice in the posterior wall of the urogenital sinus while the wolffian orifice continues on in a caudad and medial migration. (*D*) Differential growth in the posterior wall of the urogenital sinus moves the orifice of the wolffian duct further caudad. The upper pole ureter is still attached to the wolffian duct. (*E*) End stage of development resulting in common variety of ureteral duplication with ectopia.

(*F*) *Male*. When the upper ureter originates from the wolffian duct in an exceptionally high position, not enough of the caudal end of the wolffian duct can be incorporated in the posterior wall of the urogenital sinus and the high ureter remains forever attached to the wolffian duct, which by the end of the evolutionary process has become the genital duct system. The ureter enters the ejaculatory duct, the seminal vesicle or even the vas deferens.

(*G*) *Female*. Under circumstances similar to *F* in the male, the exceptionally high ureter continues to drain in a wolffian duct which is doomed to degeneration. Continued drainage of urine in the distal end of the wolffian duct keeps this distal end from degenerating.

W. = Wolffian duct. U. = Upper pole ureter (not present in case of single normal ureter). L. = Lower pole ureter (normal ureter when duplication is not present). R. = Rectum.

SUMMARY

Two cases of ectopic ureters opening at the apex of an apparent urethral diverticulum are described in girls.

An attempt is made to provide an explanation for this anomaly based on developmental anatomy. It is suggested that the "diverticulum" is a persistent segment of wolffian duct, making this anomaly in the female the counterpart of a ureter opening into the ejaculatory duct in the male.

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BILATERAL NEPHROMEGALY IN A CHILD WITH HEMIHYPERTROPHY*

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THE association of Wilms' tumor with a variety of congenital malformations is well documented. These include hemihypertrophy, aniridia, defects of the genitourinary system and hamartomatous lesions.[1] Fraumeni *et al.*[2] reported 5 cases of Wilms' tumor associated with congenital hemihypertrophy and stated that a total of 26 cases with this association had been cited.

This paper reports a 2 week old infant with hemihypertrophy and bilaterally palpable abdominal masses due to markedly enlarged kidneys. On the basis of intravenous urography, a diagnosis of bilateral Wilms' tumor was made. Open renal wedge biopsies revealed normal renal tissue; the child has remained healthy on a 3 year follow-up.

REPORT OF A CASE

P. W., a 14 day old white girl, was admitted for evaluation of a left-sided abdominal mass and enlargement of the left lower extremity. The patient was the product of a 38 week pregnancy in a 35 year old gravida 9, para 8, abortus 1 woman and weighed 8 lb., 3 oz. at birth. The gestation, labor and delivery were uncomplicated. When the child was 3 days old, a mass was felt in the left abdomen. An intravenous urogram was reportedly normal although of poor quality. The patient was discharged on the fifth day. At home, the mother noted that the left lower extremity was longer than the right. Upon re-examination, the hypertrophy of the left leg was confirmed by the patient's physician who noted no change in the size of the abdominal mass.

The patient's parents and 7 siblings were in good health without a family history of significant disease or congenital abnormalities.

On admission to this hospital, the patient appeared alert and healthy, weighing 3,900 grams and measuring 53 cm. in length. A hemangioma was present over the bridge of the nose and another at the nuchal crest. No café-au-lait spots were seen. A 4×7 cm. firm, noncystic, smooth mass was palpable on the right.

The following extremity measurements were obtained: right upper extremity was 1 cm. longer and had a ½ cm. greater circumference at biceps than the left; right lower extremity 19 cm. (hip to medial malleolus), circumference of mid-thigh 15 cm. and of calf 11 cm.; left lower extremity 20.5 cm. (hip to medial malleolus), circumference of mid-thigh 16 cm. and of calf 12.5 cm.

Routine laboratory work and chest roentgenogram were normal. Roentgenographic examination of the extremities revealed hypertrophy of both the upper and lower extremity on the left with increased length, diameters and skeletal maturation (Fig. 1). The left innominate bone was larger than the right.

Intravenous urography demonstrated bilateral enlargement of the kidneys, the right measuring 7.5×4.0 cm. and the left 7.0×3.8 cm. (Fig. 2, *A–D*). The right kidney was 7 standard deviations and the left 6 standard deviations above the mean for a patient of this size.[3] The calyceal anatomy was intrinsically distorted. The roentgenographic appearance was interpreted as being compatible with bilateral Wilms' tumor. Other less likely possibilities were adult type polycystic disease, partial renal vein thrombosis or multiple hamartomata. Preoperative radiation therapy in a single 500 r/d. dose to the most involved kidney (right) was recommended but not carried out. An open biopsy of both kidneys was performed prior to any therapy. At celiotomy large kidneys were the only abnormality found. No thrombus or abnormality of the inferior vena cava or renal veins was detected. Bilateral lower pole wedge biopsies were made with frozen section report of "unremarkable kidneys." The biopsy specimen from the left kidney measured 11 mm.×5 mm.×1 mm. and the specimen from the right kidney measured 7 mm.×4 mm.×2 mm. Microscopic examinations of both specimens were reported as "normal immature parenchyma" (Fig. 3, *A* and *B*).

The postoperative course was uneventful and the patient was discharged. Follow-up intravenous pyelography 1 month after surgery showed persistent renal enlargement with im-

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provement in the anatomy of the collecting system. The child has subsequently done well and a repeat intravenous pyelography at 27 months of age (Fig. 4, *A* and *B*) showed persistent although less marked enlargement of both kidneys with less intrinsic distortion of the collecting systems. The patient has remained well for 3 years although the kidneys are still palpably enlarged.

DISCUSSION

The differential diagnosis of abdominal masses of renal origin in the newborn infant is rather extensive and depends primarily on the roentgenographic appearance of the kidneys.[4] In this patient both kidneys were large with somewhat distorted calcyes. Although we felt that bilateral Wilms' tumor was the likely diagnosis, the gross and microscopic anatomy and course excluded this possibility. Confusion over what constitutes a Wilms' tumor exists in neonates.[1,6] A lengthy discussion of this problem is beyond the scope of this report; however, readers are referred to the excellent review by Wigger.[10] Other conditions also felt to be excluded are: (1) the adult form of polycystic disease which is occasionally seen in infants;[5] (2) renal vein thrombosis;[7] (3) multiple hamartomata; and (4) involvement by tumors other than Wilms' tumor, such as leukemia or lymphoma *versus* various infiltrative processes—amyloid, glycogen storage disease, etc.

Hemihypertrophy is an unusual malformation of obscure origin which is not infrequently associated with a variety of disorders other than Wilms' tumor. These include mental deficiency, skin abnormalities (nevi, telangiectasia, hemangiomas, and café-au-lait spots), neurocutaneous disorders or phakomatoses,[10] adrenocortical carcinoma, hepatoblastoma[2] and neuroblastoma. It is extremely rare in the latter tumor. The actual frequency and distribution of neoplasia among individuals with hemihypertrophy is unknown.

In their review of 104 confirmed cases of hemihypertrophy and 25 cases of segmental hypertrophy, Ringrose *et al.*[9] reported 5 cases with enlargement of the internal organs on the involved side. One case of Wilms' tumor on the involved side was included. The actual incidence of internal organ enlargement in this condition is unknown, since examination of these organs has been rare in the absence of tumors. In 25 cases of hemihypertrophy associated with Wilms' tumor where laterality was specified, hemihypertrophy was ipsilateral in 13, contralateral in 8 and crossed (involving different anatomic areas on both sides of the body) in 4 children.[2]

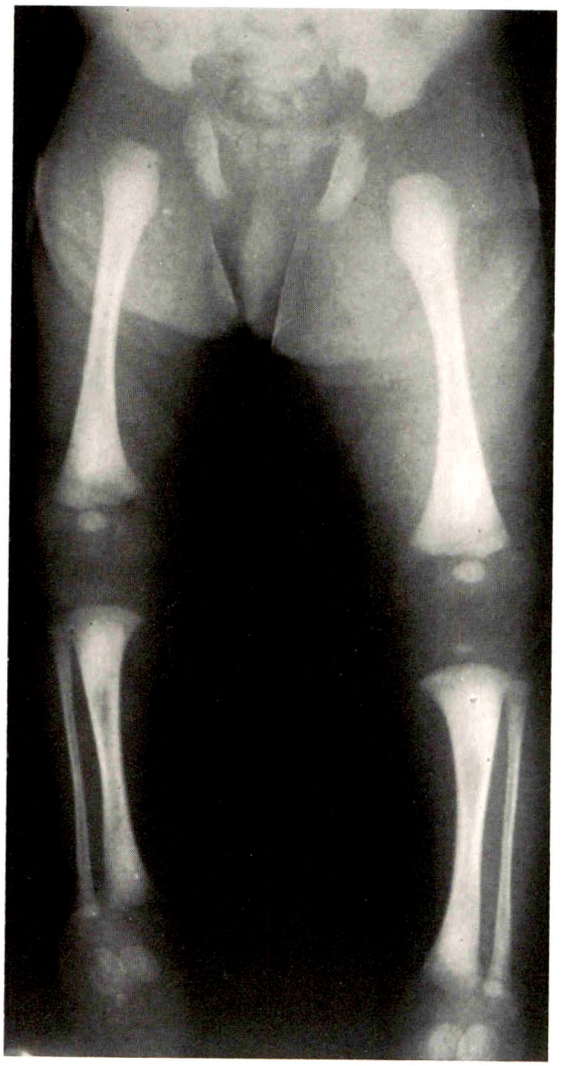


Fig. 1. Lower extremities showing left-sided hypertrophy.

The possible relationship between hemihypertrophy and Wilms' tumor (or other childhood neoplasia) is uncertain; however, Fraumeni *et al.*[2] speculate that "the association between hemihypertrophy and Wilms'

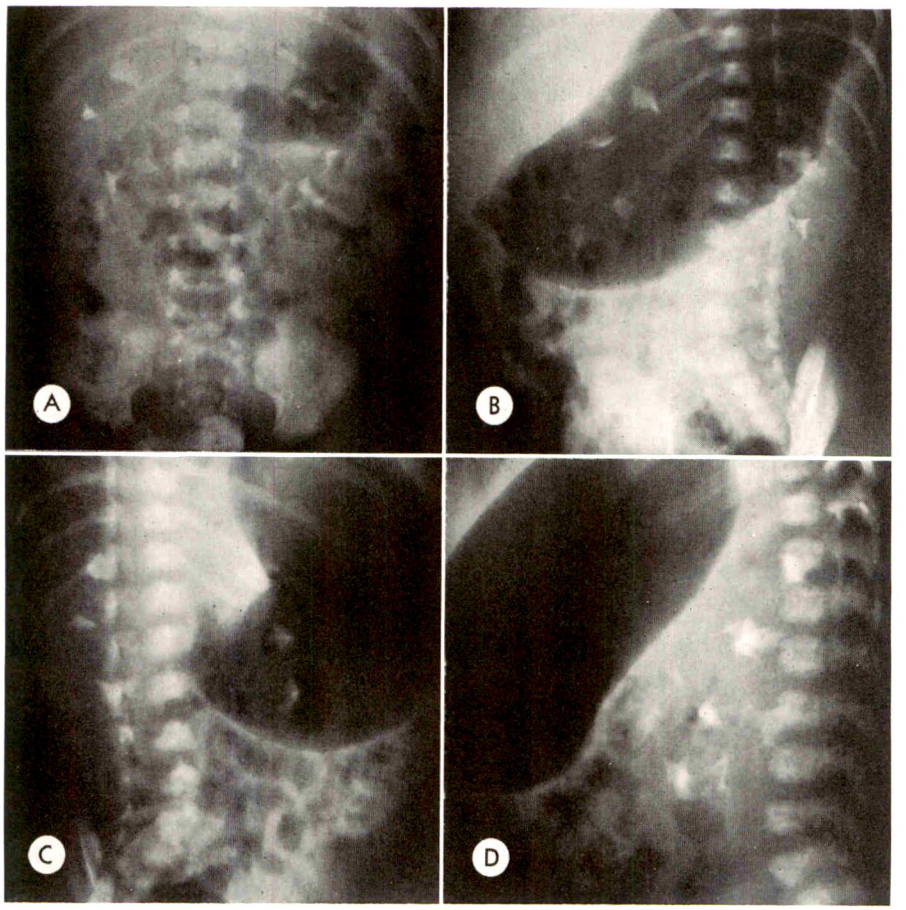


FIG. 2. Intravenous urograms at 2 weeks of age showing bilaterally enlarged kidneys with intrinsic calyceal distortion. (*A*) Anteroposterior, (*B*) oblique right kidney, (*C*) oblique left kidney, and (*D*) lateral views.

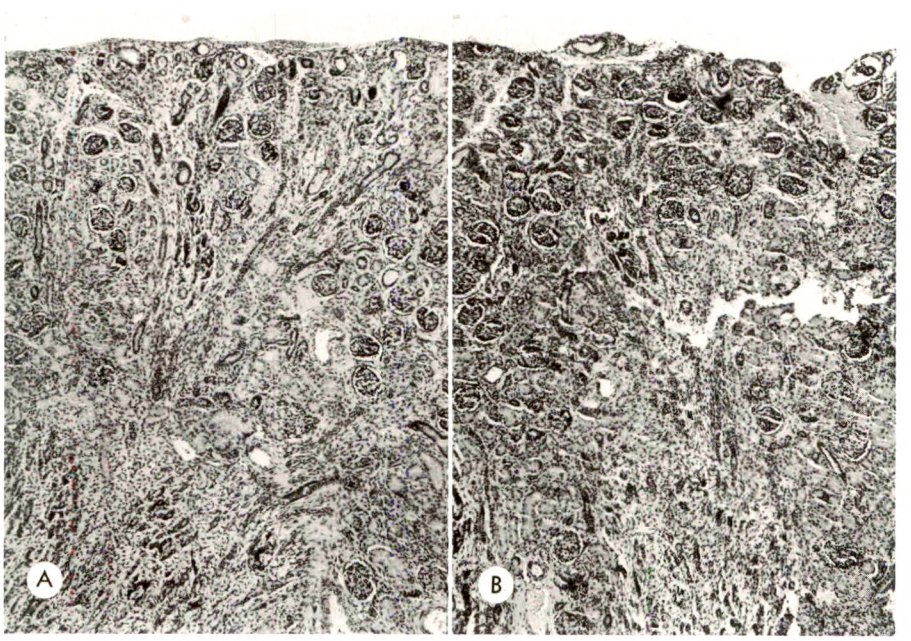


FIG. 3. Low power photomicrographs of biopsies of (*A*) right and (*B*) left kidneys showing histologic appearances normal for a newborn term infant. (Both × 90.)

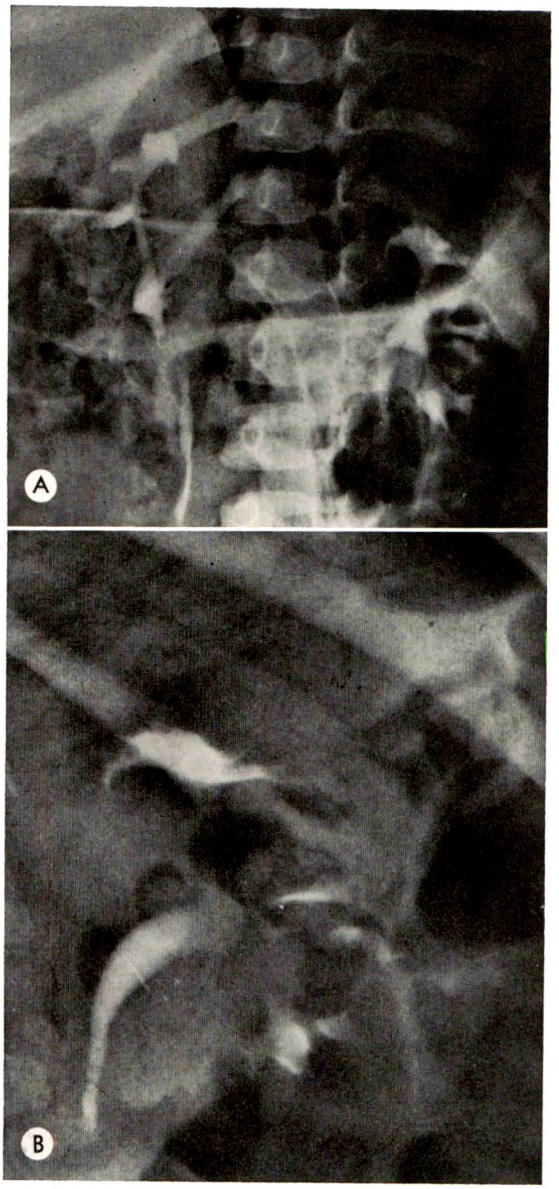


Fig. 4. Intravenous urograms at 27 months of age. (*A*) Oblique view of the right kidney, and (*B*) anteroposterior view of the left kidney showing persistent enlargement and calyceal distortion.

tumor may reflect either common etiologic factors or a preneoplastic anlage in the hemihypertrophic kidneys." They also suggest that "since a number of kidney anomalies coexist with Wilms' tumor, one might expect occasionally to see congenital defects instead of Wilms' tumor as the renal expression in hemihypertrophy." The possibility that the renal abnormalities seen in our patient with hemihypertrophy may represent such an expression adds to the interest in the interrelationship of these 2 conditions.

SUMMARY

A newborn with hemihypertrophy and bilateral palpable abdominal masses due to markedly enlarged kidneys is reported.

The kidneys showed intrinsic distortion of the collecting systems by intravenous urography and a preoperative diagnosis of Wilms' tumor was made. Open wedge biopsies of each kidney evinced normal tissue. Despite large kidneys the patient has remained well for 3 years.

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AN UNUSUAL BONE DYSPLASIA: PARASTREMMATIC DWARFISM

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ONE of the best known examples of dysostotic dwarfism with excessive shortness of the spine is Morquio's disease. This rare disorder has been strictly defined in recent years by a pattern of clinical, roentgenologic and biochemical characteristics.[7,10] With the use of rigid diagnostic criteria it has become possible to differentiate from Morquio's disease a number of bone dysplasias such as diastrophic dwarfism,[6,8] metatropic dwarfism,[11] the congenital[15] and the tarda[9,12] type of spondyloepiphyseal dysplasia.

The purpose of this paper is to present another condition that has been confused with Morquio's disease. Since one of the major clinical features of the disease is an exceptionally severe distortion of the lower extremities, we suggest the term "parastremmatic dwarfism," from the Greek parastremma meaning "distorted limb."

REPORT OF CASES

CASE 1. Anette K., born in 1954, was treated in the Orthopedic Hospital of Hannover in 1956 and 1967 for multiple skeletal deformities.

Her father was born in 1910 and measured 159 cm. The mother was born in 1915 and measured 155 cm. Both were living and well. There was no indication of parental consanguinity. Two sisters, born in 1947 and 1950, measured 156 and 155 cm., respectively, and were clinically normal. The patient was the product of a full term gestation, a spontaneous delivery and her birth weight was 3,385 gm. Immediately after birth a "stiffness" of the spine and a mild scoliosis were noted. Subsequently, bowing of the legs developed, and a diagnosis of rickets was made. This was later changed to chondrodystrophy. The patient learned to walk by the age of 2 years. Her mental milestones were normal.

Physical examination in 1956 revealed a $2\frac{1}{2}$ year old dwarfed girl measuring 79 cm. (<3 per cent). The clinical features are shown in Figure 1. Briefly, they consisted of a brachycephaly with a high forehead and temporal bulge, a pectus carinatum, S-shaped kyphoscoliosis, short extremities with bilateral genu valgum and a varus deformity of the thighs and shanks. There was an osseous enlargement of the knees. There were multiple contractures of the large joints and the girl walked with her hips and knees flexed. Roentgenograms are reported to have demonstrated flattened, irregularly structured vertebral bodies, a kyphoscoliosis, deformed pelvic and bowed tubular bones, metaphyseal widening and irregular epiphyseal and metaphyseal bone texture.

The patient was again seen at the age of 13 years.

Physical examination revealed a severely dwarfed and deformed girl without any complaints (Fig. 2). The body measurements are given in Table 1.

The head was brachycephalic. There was a fixed kyphoscoliosis with its apex in the mid

TABLE I

BODY MEASUREMENTS OF CASE I

	Patient (cm.)	Normal for Age[16] (cm.)	Normal for Height[16] (cm.)
Height	99	156	
Sitting height	59	82	56
Length of legs	l. 49 r. 41	88	51
Length of upper arms	18	30	17
Length of forearms	24	23	14
Hand length	12	17	11

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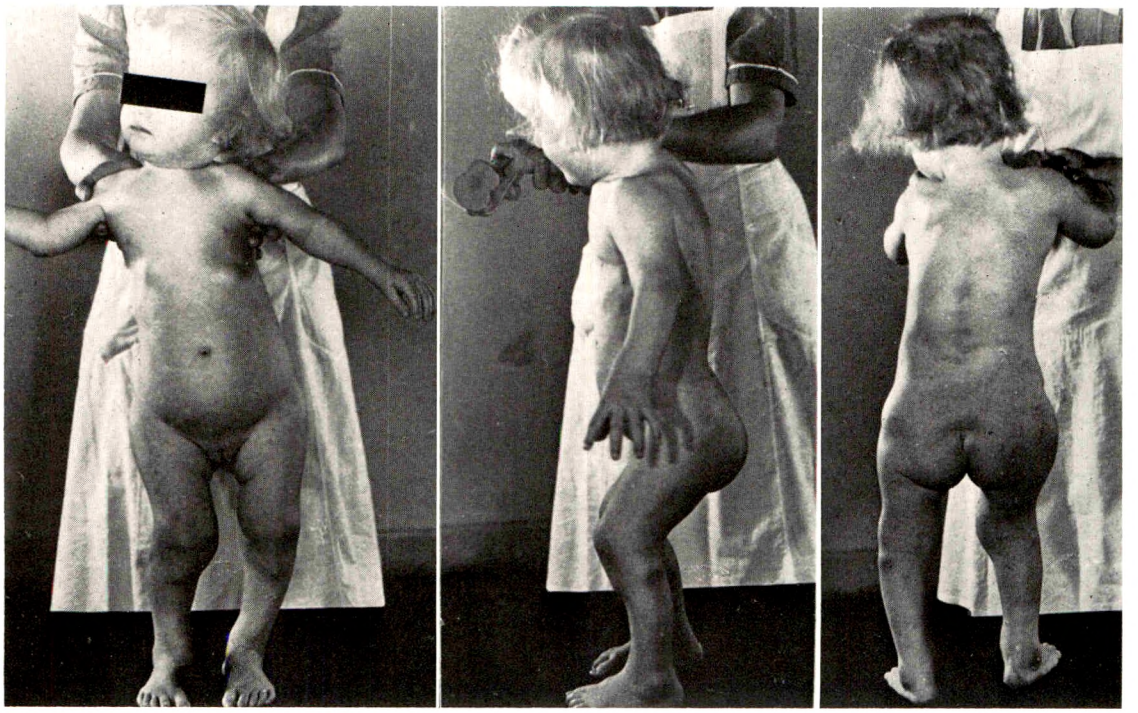


Fig. 1. Case 1. Two and one-half years.

thoracic region. Craniad the scoliosis extended into the cervical spine forcing the head into a left tilt. The lower part of the thoracic cage was everted. There was a mild varus deformity of the upper arm. The hands and feet were short and stubby. The right leg was 8 cm. shorter than the left. There was a grotesque, 55° valgus deformity of the right leg and a milder genu valgum on the left. Motion about the shoulders was limited in all planes. Extension of the el-

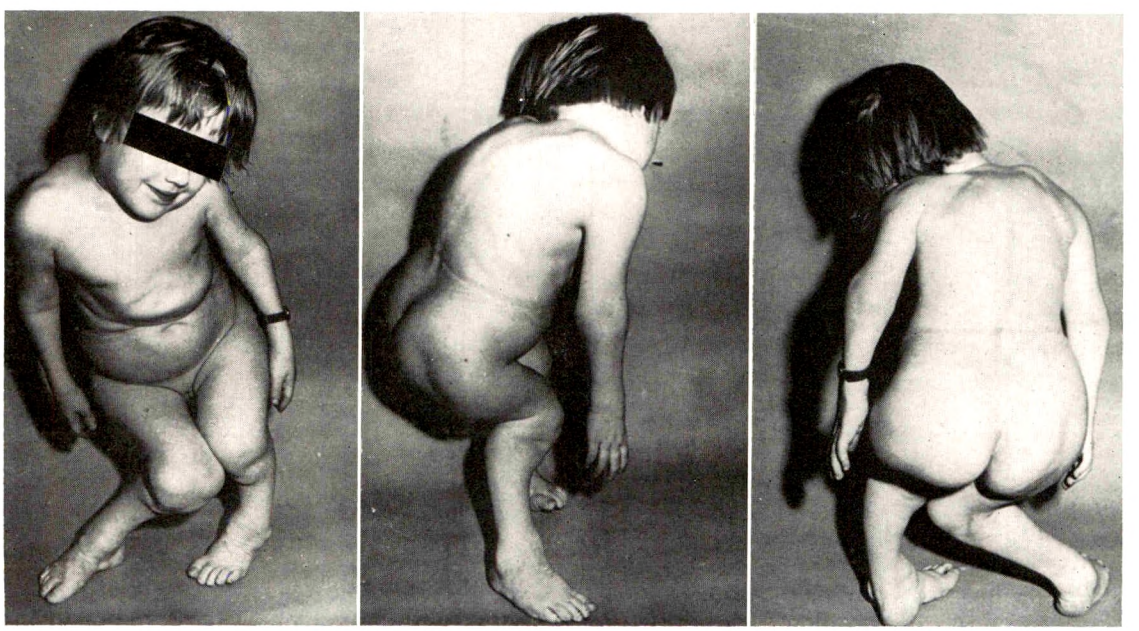


Fig. 2. Case 1. Thirteen years.

bows and rotation of the forearm were moderately restricted. Abduction of the hips was limited to 10°, extension to 160° and rotation to 40°. Extension contractures were present in the talocalcaneal joints. Hyperextensibility was not noted. The patient had a waddling gait with her hips and knees flexed. Fundoscopy and slit-lamp examination of the corneae were within normal limits. The sclerae were white; the teeth, fingernails, hair, skin and internal organs were normal.

Laboratory findings: Ca, 9.9 mg. per cent; P, 4.5 mg. per cent; alkaline phosphatase 46.5 mU (normal 10–40 mU); K, 4.9 mEq/l. There was normal urinary excretion of acid mucopolysaccharides.

The roentgenographic findings in this case are discussed in the Section of Roentgenographic Features.

CASE II. Sharon S., born in 1945, was admitted to the San Francisco unit of the Shriner's Hospital in 1955 for correction of a recurrent valgus deformity of the right knee.

There was no family history of congenital deformities or dwarfism. Two sisters were reported to be normal.

The patient was the product of a full term gestation and a spontaneous delivery. Generalized stiffness was noted at birth and persisted. She sat up at 9 months, talked and walked at 12 months. By the age of 8 months a tentative diagnosis of vitamin D resistant rickets was made. This diagnosis was changed subsequently to a variant of achondroplasia. Bilateral tibial osteotomies were done for a valgus deformity of the right knee and a varus deformity of the left knee.

Physical examination revealed a 10 year old, white, dwarfed girl measuring 100 cm. (<3 per cent). There was a marked kyphoscoliosis of the dorsal spine with severe limitation of flexion, extension and lateral bending. The left leg was essentially straight, but on the right side a 30° genu valgum was present. Abduction and internal rotation were limited in both shoulders. Both elbows lacked 40° of extension. In the lower extremities there was a 20° flexion contracture and severe abduction contracture of the hips.

Laboratory examination disclosed a normal white blood cell count, urinalysis, bleeding time, coagulation time and a vital capacity of 950 cm.

The roentgenographic findings are discussed in the Section of Roentgenographic Features.

CASE III. Grace O., born in 1956, was examined in the Chicago Unit of the Shriner's Hospital in 1966 for evaluation of multiple skeletal abnormalities.

The father was 37 years old and 170 cm. in height. The mother was 37 years old and measured 165 cm. There was no evidence for parental consanguinity. The patient was the fourth in a sibship of 6. Two sisters and 3 brothers were of normal height and without bone abnormalities. The mother had 2 abortions, 1 at 2 months and 1 at 5 months.

The patient was the product of a full term gestation and a spontaneous delivery. She appeared normal at birth, measuring 49.5 cm. She was able to stand at 10 months and started to walk at 13 months. From the beginning her gait seemed "funny." Roentgenograms taken at the age of 15 months were interpreted as showing signs of achondroplasia with bowing of the legs and club hips.

Physical examination revealed a 10 year old dwarfed girl measuring 91 cm. (<3 per cent), who complained of occasional pain in the mid-back. The head appeared normal, the neck was short, the anteroposterior diameter of the thorax was increased. There was a gibbus about the level of D-11 and a slight right low dorsal scoliosis with limited motion of the back. The hands were short and stubby with some hyperextensibility of the metacarpophalangeal joint of the index finger. The lower extremities showed marked internal bowing of the tibiae which approximated 45°.

Motion was limited in all major joints.

Ophthalmoscopic examination showed the fundi to be normal. No corneal opacities were seen. There was a 1×1 cm. pigmented area over the right lateral forehead. Hair, nails, teeth and internal organs were clinically normal.

Neurologic examination was negative. Mental development was normal; the patient was to enter the 5th grade of a school for the handicapped.

Roentgenograms revealed changes similar to those in Cases I and II (see Section of Roentgenographic Features).

REVIEW OF THE LITERATURE

Apert, Liege and Denet,[1] in 1934, described a 12 year old girl with severe dwarfism, kyphoscoliosis and excessive bowing of the lower extremities. Roentgenograms

showed skeletal changes identical to those in our patients.

In a family observed by Rask[13] in 1963, the proband, a 15 year old girl, inherited from her mother the classical features of osteogenesis imperfecta with increased bone fragility, blue sclerae and hyperlaxity of the joints. Her father transmitted a bone dysplasia interpreted as Morquio's disease. The clinical and roentgenologic features of both father and daughter do not support that diagnosis. Morquio's disease in these 2 patients is ruled out by the fact that it is inherited as a recessive trait, whereas dominance was present in Rask's patients. They are 2 more examples of parastremmatic dwarfism.

This review is based on these 3 cases from the literature and on our 3 cases. The original roentgenograms of Rask's patients were available for roentgenologic evaluation from the Portland unit of The Shriner's Hospitals (Fig. 10, *A–D*).

CLINICAL FEATURES

The disease manifests itself in the first year of life. Some babies are described as "stiff." This feature may be present in the neonate. Usually in the sixth to twelfth month of life, skeletal deformities develop which are interpreted as rachitic. Walking frequently is delayed and the gait is abnormal from the beginning. Roentgenographic changes at this time tend to be interpreted as achondroplastic in nature: with the appearance of spinal deformities this diagnosis is apt to be changed to Morquio's disease. By the age of 10 the full clinical picture has developed.

The patients may be described as twisted dwarfs. They are unusually small with an ultimate height of between 90 cm. and 110 cm. Their spine is distorted by varying degrees of kyphoscoliosis. Their neck is short and the anteroposterior diameter of the thorax is increased. There are bizarre and frequently asymmetric deformities of the lower extremities with a severe genu valgum and bowing of the long bones in both the frontal and sagittal planes. In addition, the thighs and shanks are twisted around their long axis.

In contrast to this, the arms appear relatively long. The hands are short and stubby, but do not show the trident configuration seen in achondroplasia. The skull is relatively normal, but in some cases is deformed due to a relative softness of the cranial bones. There are multiple contractures of the major joints. In Rask's younger patient, hyperextensibility of some joints was observed but probably was due to the osteogenesis imperfecta which co-existed with the parastremmatic dwarfism. Hyperlaxity has not been a feature in other affected individuals. There are no characteristic extraskeletal deformities. Corneal opacities have not been found. The internal organs seem to be unaffected and the mental development is normal. There are amazingly few complaints. Except for Reilly bodies in bone marrow cells described by Rask, laboratory investigations so far have been unrevealing. The urinary excretion of acid mucopolysaccharides including keratosulfate was normal in Case 1.

Biopsy material from the physeal region of the proximal femur of our Case 1 showed findings virtually identical to those described by Rask.[13] Studies performed by Dr. Remagen, Basel, revealed the endochondral bone formation to be decreased and irregular. The number of osteoblasts and osteoclasts was diminished. There was complete lack of columnation of the cartilage cells. In many areas the zone of provisional calcification was walled off by a thin bone plate.

ROENTGENOGRAPHIC FEATURES

The bone density is generally decreased. In particular, the zones of endochondral bone formation are clear, widened and coarsely trabeculated. They contain areas of very irregular dense stippling and streaking. The bone structure in these areas can be described as "flocky" or "woolly." This flocky bone is most conspicuously seen along the borders of the lower vertebral bodies, the iliac crest, the ischial and pubic

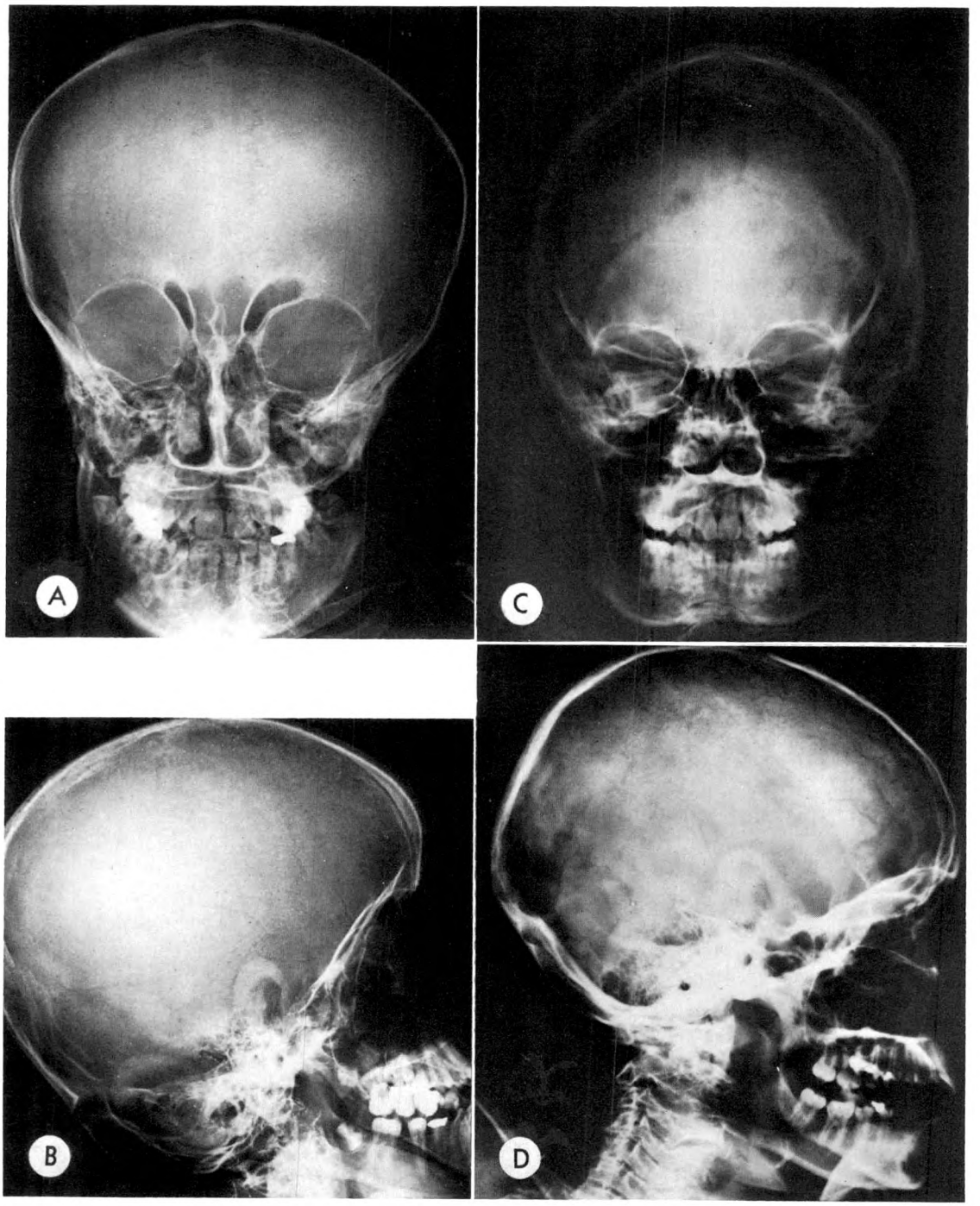


FIG. 3. (*A* and *B*) Case I. Thirteen years: brachycephaly, frontal and bitemporal bossing, steep anterior base, shallow posterior fossa. (*C* and *D*) Case III. Thirteen years; broad skull and mandible.

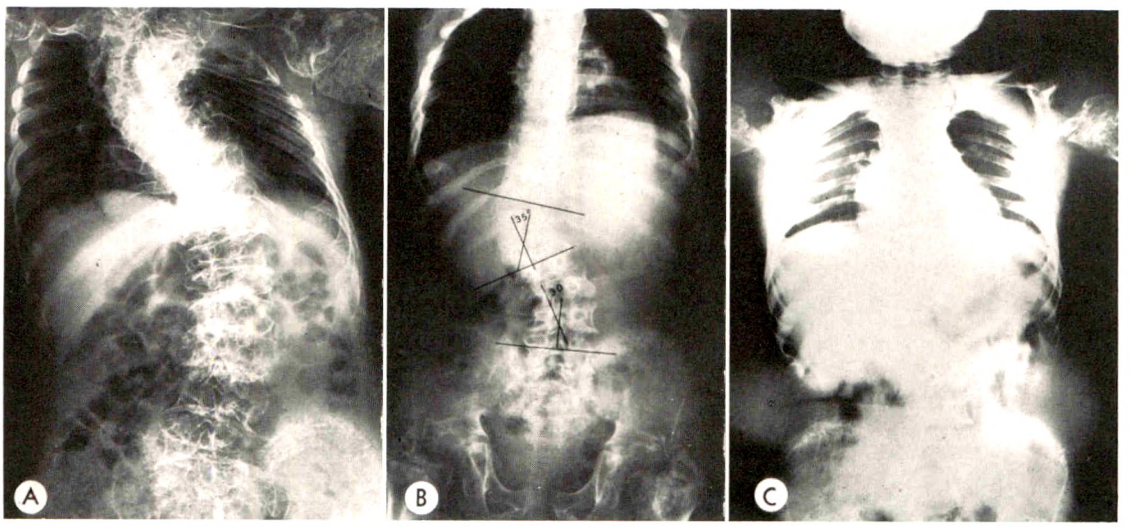


Fig. 4. (*A*) Case I. Thirteen years. (*B*) Case II. Eleven years. (*C*) Case III. Ten years. S-shaped scoliosis, decreased bone density with very irregular calcification of the vertebral bodies, iliac crests and proximal ends of the humeri.

bones and the epimetaphyseal segments of the long bones (Fig. 4–10). The bone density of the cranial bones is decreased. Deformities of the cranial vault secondary to the osteopenia, such as occipital flattening and temporal bossing, are present in some cases (Fig. 3, *A–D*). There is a kyphoscoliosis deformity of the spine with a moderate gibbus (Fig. 4, *A–C*; 5, *A* and *B*; and 10*C*). The vertebral bodies are flattened, rectangular in shape, with biconcave deformities in some cases. The homogeneous, ra-

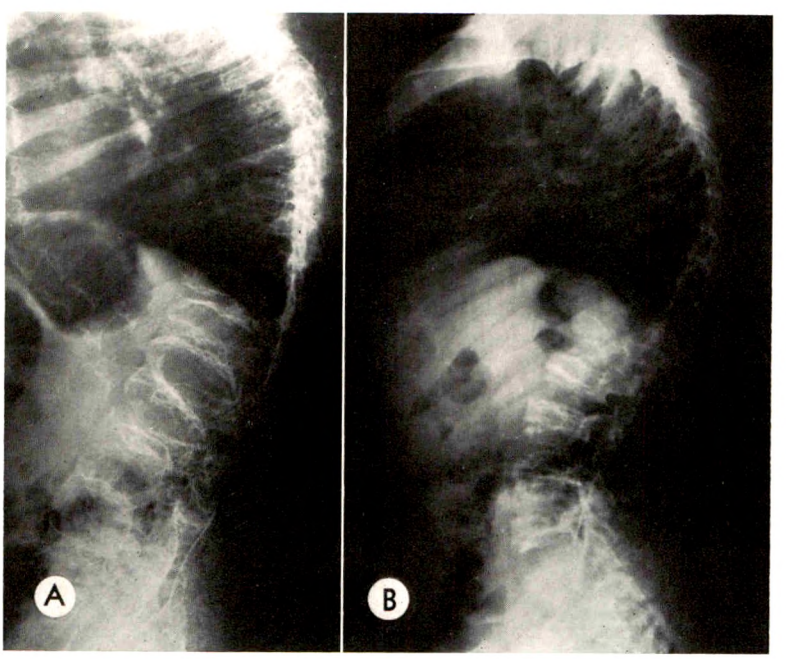


Fig. 5. (*A*) Case I. Thirteen years. (*B*) Case II. Eleven years. Increased thoracic kyphosis, severe flattening, decreased bone density and irregular calcification of vertebral bodies; wide intervertebral spaces.



FIG. 6. Case 1. Thirteen years. Decreased vertical diameter of the iliac wings, large ischial and pubic bones; strikingly abnormal bone structure with dyscalcific streaking and stippling in the iliac crests, ischial bones, and proximal femora. Epiphyseal and metaphyseal dysplasia of the proximal femora.

diolucent centers of the bodies are framed by a pencilled or very irregular, streaky and cystic cortex (Fig. 5, *A* and *B*; and 10*C*).

The pelvic bones are dysplastic (Fig. 6; and 10, *A* and *D*). The iliac wings are small and bordered by a lace of "flocky bone." The ischial and pubic bones are wide and their structure is irregular. The femoral neck is short and in the young contains broad areas of irregular flocky bone on both sides of the physeal line. The femoral head is small, distorted and frequently dislocated.

The diaphyses of the long tubular bones are thin and mildly bowed. The ends of the tubular bones show wide, clear metaphyses containing flocky bone as described above. There is acute bowing of the metaphyseal segments, especially in the knee region (Fig. 7, *A–C*). In addition, the metaphyseal segments may be rotated around their long axis. The epiphyses are severely deformed and radiolucent. Bowing is less pronounced in the upper extremities. The humerus, radius and ulna are short and their epimetaphyseal segments are distorted, containing streaks and flocks of calcified tissue in otherwise under-mineralized bone (Fig. 8, *A–D*; and 9*A*). The radial head is dislocated posteriorly in some cases, and the distal ulnar and radial growth plates have a V-shaped obliquity (Fig. 9*B*). The carpal and tarsal bones are irregular in outline and structure. The tubular bones of the hands and feet are similarly affected, being short with narrow shafts, wide, clear metaphyses, dysplastic epiphyses and irregular bone structure along the physeal lines (Fig. 9*B*). The scapulae and clavicles are mildly irregular. The vertebral and anterior ends of the ribs are flared and cupped (Fig. 4, *A–C*).

GENETICS

The disease was transmitted from the father to his daughter in the family described by Rask. The parents of the other cases were described as physically normal and only 1 child was affected. Parental consanguinity was not noted. The parental ages were 44 and 35 years at the time of birth of our Case 1. These data are consistent with a dominant mode of inheritance with new mutations as the cause for the occurrence of the disease in the isolated patients. Both autosomal and X-chromosomal dominance are possible. Five of the 6 described patients are female.

DIFFERENTIAL DIAGNOSIS

Structural bone changes similar to those seen in parastremmatic dwarfism are found in the Jansen type of metaphyseal dysostosis.[2,3,5,17] In Jansen's disease the areas of cystic dyscalcification are confined to the metaphyses. The epiphyses and vertebral bodies are normal.

In some rare cases of multiple epiphyseal dysplasia[4] and in disorders of thyroid function[14] the epiphyses may have a granular appearance. In these cases severe dwarfism and marked platyspondyly do not occur. The metaphyseal segments are normal and the bone density is not decreased.

In the cases of chondrodystrophia calcificans who survive beyond infancy dwarfing is not as severe as in parastremmatic dwarfism, the metaphyses are not involved, and bone density usually is normal. The

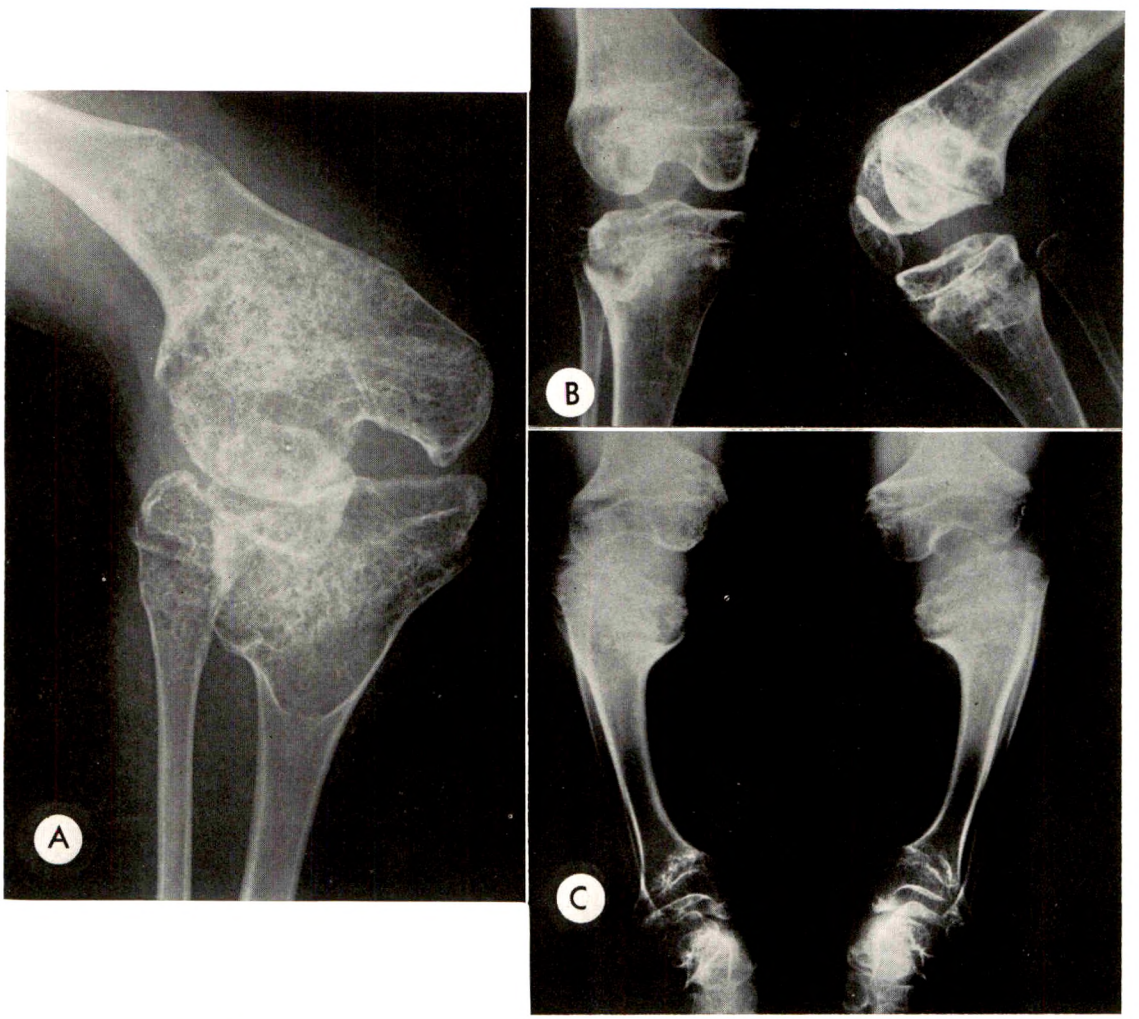


Fig. 7. (*A*) Case I. Thirteen years. (*B*) Case II. Thirteen years. (*C*) Case III. Ten years. Grossly irregular, "flocky" bone structure on both sides of the widened physeal lines; clear and wide metaphyses; epiphyseal dysplasia; genua valga; varus deformity of the tibia in *C*.

calcific stippling is rarely seen after the age of 2 years.

Morquio's disease is differentiated by characteristic roentgenographic features such as an age specific pattern of anterior hypoplasia in the flattened vertebral bodies in the thoracolumbar region, wide iliac flare and coxa valga.[7,10] The irregular flocky bone structure is not seen in Morquio's disease. Corneal opacities and increased urinary excretion of keratosulfate are present in Morquio's disease. The genetic mode of transmission is recessive in Morquio's disease and dominant in parastremmatic dwarfism.

Metatropic dwarfism[11] enters the differential diagnosis because of its progressive kyphoscoliosis with platyspondyly and its epiphyseal and metaphyseal irregularities. In metatropic dwarfism bone density is not as severely decreased, areas of flocky dyscalcification are not present and metaphyseal enlargement is more pronounced. In contrast to parastremmatic dwarfism it is inherited as an autosomal recessive trait.

Shortness of stature, kyphoscoliosis, shortened and deformed extremities are features of diastrophic dwarfism.[6,8] It is differentiated from parastremmatic dwarfism by its roentgenologic characteristics

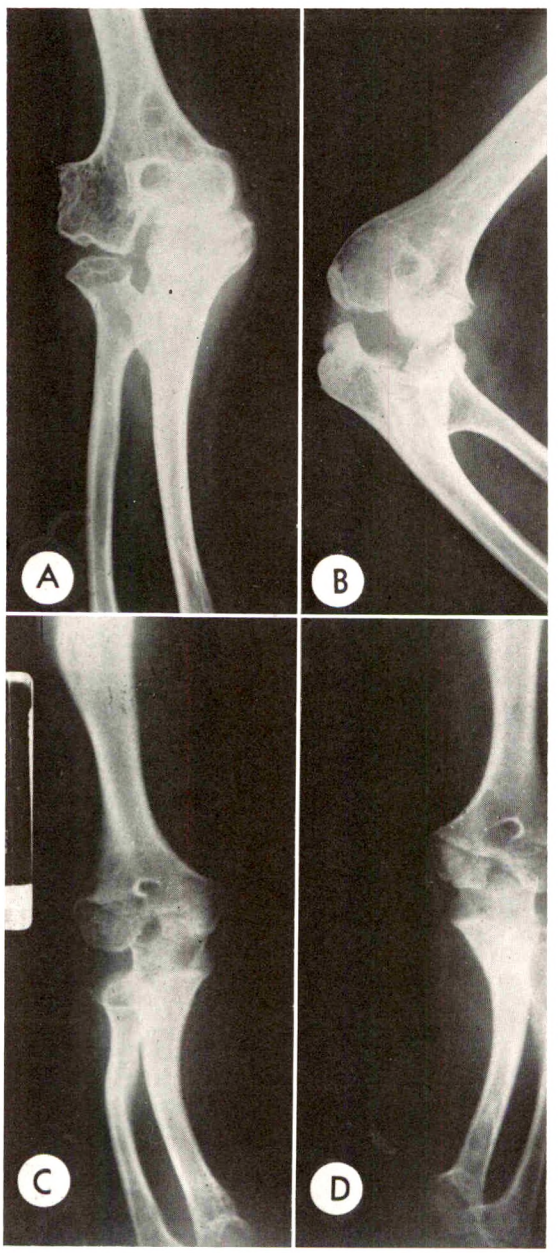


FIG. 8. (*A* and *B*) Case I. Thirteen years. (*C* and *D*) Case III. Ten years. Severe deformity of the distal humerus, proximal radius and ulna with widened, clear metaphyses, dysplastic epiphyses; bowing of the forearms in *C* and *D*.

such as severe shortening of the first metacarpal and first metatarsal bones, fork-like deformity of the distal humerus and relatively well preserved vertebral bodies. The clinical features of diastrophic dwarfism such as thickened external ears, cleft palate,

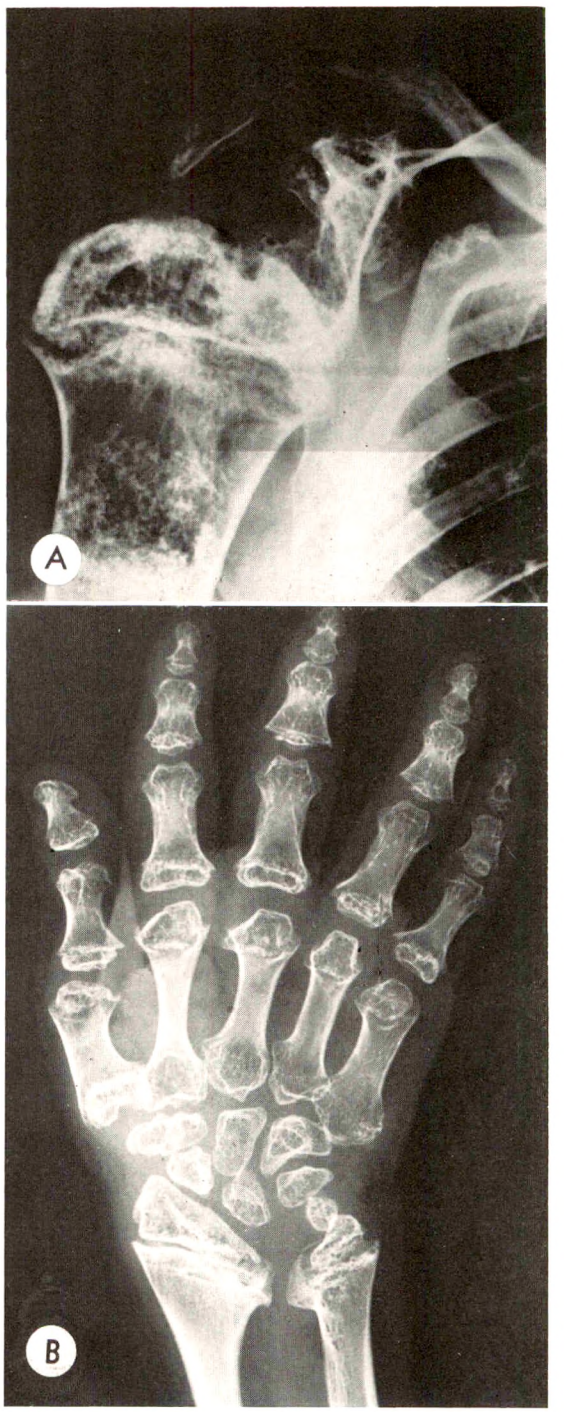


FIG. 9. Case I. Thirteen years. (*A*) Areas of irregular calcification of the scapula and of the proximal humerus. Flattened humeral epiphysis. (*B*) Decreased bone density; severe shortening of the metacarpal and phalangeal bones. Epiphyses, metaphyses and carpal bones are irregular in contour and structure. Obliquity of the distal radial and ulnar growth plates.

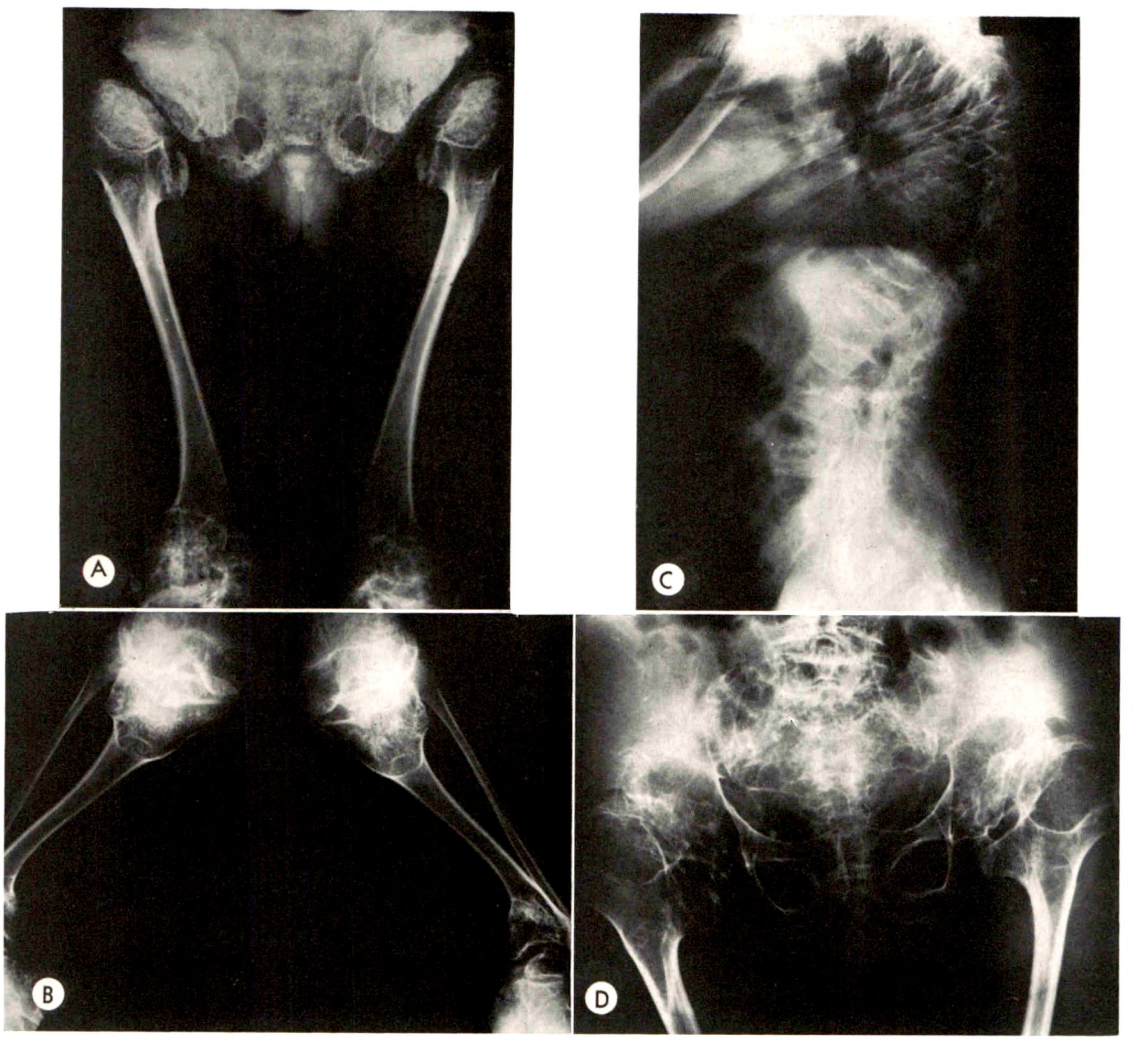


Fig. 10. Rask's adult patient. (*A* and *B*) In early adulthood, compare with Figures 6 and 7. (*C* and *D*) In early fifties, compare with Figures 5*A* and 6.

club feet, and hitchhiker's thumb will facilitate the differential diagnosis. This is an autosomal recessive condition.

SUMMARY

The term parastremmatic dwarfism is suggested for a bone dysplasia characterized by severe dwarfism, kyphoscoliosis, distortion and bowing of the extremities and contractures of the large joints.

Roentgenographically the disease is characterized by a combination of decreased bone density, bowing of the long bones, platyspondyly and striking irregularities of endochondral ossification with areas of calcific stippling and streaking in radiolucent epiphyses, metaphyses and apophyses.

The disease is inherited as a dominant trait and must be differentiated from other bone dysplasias with similar structural bone changes or kyphoscoliosis, such as the Jansen type of metaphyseal dysostosis, Morquio's disease, diastrophic dwarfism and metatropic dwarfism.

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FOCAL DERMAL HYPOPLASIA SYNDROME

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IN 1928 Jessner[22] described a 4 year old boy with unusual skin changes and syndactyly of the fingers. Other case reports, describing similar cutaneous changes with skeletal and other anomalies, appeared under a variety of names during the following decades. Goltz *et al.*[13] in 1962 and Gorlin *et al.*[14] in 1963 named this the Focal Dermal Hypoplasia (FDH) syndrome. They defined it as consisting of: (a) atrophy and linear hyperpigmentation of the skin; (b) localized deposits of superficial fat; (c) multiple papillomas of mucous membranes or periorificial skin; (d) anomalies of the extremities; and (e) anomalies of the nails.

No comprehensive evaluation of the roentgenologic findings has been reported in previous publications on the subject. Furthermore, no discussion of this ectodermal and mesodermal dysplasia has appeared in the radiologic literature. In this paper a new case is presented and the literature is completely reviewed. The clinical features are discussed and the roentgenologic diagnosis is described. In addition, available genetic data are analyzed.

REPORT OF A CASE

J.A.N., a female, was born prematurely on June 14, 1962. The mother had been gravida 2 and para 0, but no further information was available about the first pregnancy. The use of drugs during the second pregnancy was denied by the mother.

The infant's birth weight was 5 lb. 5 oz. Many congenital anomalies were obvious. There were multiple vesicles in the right groin region, where the skin was described as being transparent. Survey roentgenograms of the 1 day old infant were reported as showing a rudimentary right clavicle. They also demonstrated relative hypoplasia of the proximal humeral epiphysis and the right knee epiphyses.

The weeping skin vesicles began healing on the 11th day. At 16 days of age the infant weighed 4 lb. 5 oz. During the ensuing weeks she gained little weight, and suffered from chronic constipation because of anal stenosis. She was discharged on January 29, 1963 with a final diagnosis of "multiple congenital deformities."

The patient was admitted to Los Altos Hospital on December 10, 1968 for repair of extensive dental caries under general anesthesia. Physical examination revealed a poorly developed, mentally retarded child who weighed only 19 lb. 8 oz. The patient had a peculiar facies (Fig. 1). Scalp hair and eyebrows were sparse. There were red blotches, as well as telangiectasia, about the face and neck. Ocular hypertelorism and a broad nasal bridge were apparent. The forehead was in a continuous line with the nasal bridge, when viewed in profile. The left ala nasi was smaller than its counterpart and slightly notched. The left cheek was depressed. The lips were thin and the corners of the mouth drooped. Low-set ears were noted and the right helix was very thin. The tragi were elongated, as was the right antitragus. The right external auditory canal was moderately stenotic. Some hearing was present and vision seemed to be limited to light perception. Neither vision nor hearing were tested in detail. Horizontal nystagmus was present. The left cornea was opaque and there was microphthalmia on the right (Fig. 2). Pink, granular excrescences were present on the skin near the lateral canthus of the right eye and below the left nares. The neck was slightly webbed. Neither cardiac nor pulmonary abnormalities were detected by physical examination. Gross pectus excavatum and a ventral hernia, the result of an old omphalocele, were present (Fig. 3). Both the external genitalia and the anus were abnormally small. A rudimentary

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FIG. 1. Facies of patient at 6 years of age. Right shoulder is deformed because of clavicular hypoplasia.

tail was present (Fig. 4). Areas of skin atrophy and hyperpigmentation were distributed over the trunk and appendages, especially on the right side. These changes were most pronounced over the right iliac crest region, right groin and upper right thigh. In these same regions there were soft, verruciform, yellow and pinkish-yellow plaques (Fig. 5). On the extremities the latter lesions had a "baggy" appearance and were smaller collections than on the trunk. The right upper and lower extremities were shorter than their counterparts. There were multiple congenital defects of the hands and feet. The nails were dystrophic and some showed spoon deformity. Routine laboratory tests revealed a hemoglobin of 11.6 gm. per cent, packed cell volume of 38 per cent, and a white blood cell count of 12,800. The differential count was 73 per cent segmented cells, 18 per cent lymphocytes, 4 per cent monocytes and 5 per cent eosinophils. The fasting blood sugar was 78 mg. per cent and the blood urea nitrogen was 14.3 mg. per cent. No permit for skin biopsy could be obtained from the parent.

Roentgenographic survey of the skeleton was performed. It revealed diffuse osteoporosis and skeletal immaturity. The skull was brachycephalic with a steep clivus. Asymmetry of the craniofacial skeleton was demonstrated (Fig. 6). Tooth crowns were abnormally shaped (Fig. 7). Chest roentgenograms revealed no gross abnormality of the heart or lungs, but many skeletal abnormalities (Fig. 8). An intravenous urogram was suggestive of fused kidneys (Fig. 9). The skeletal structures of the lumbar region and pelvis were poorly developed. The lumbar vertebral bodies were relatively tall. There was a transitional, lumbosacral vertebra. The tail was composed of unusually long and large coccygeal segments (Fig. 10). Bilateral coxa valga was demonstrated. The long bones had a gracile appearance. The bones of the right thigh, leg, arm and forearm were slightly hypoplastic. There were asymmetric malformations of the knee, ankle, shoulder and elbow



FIG. 2. Microphthalmia of right eye. Note pink, skin excrescences at lateral canthus.

joints. Skeletal immaturity of the elbows was greater on the right, and the right radial head sloped in a posterolateral direction. Skeletal maturity was estimated to be that of an average 4 year old girl, on the basis of hand roentgenograms. There was, however, a significant difference between the two sides. Syndactyly and brachydactyly of the hands were evident. The severest changes were present in the right hand (Fig. 11, *A* and *B*). Roentgenograms of the feet demonstrated brachydactyly and oligodactyly (Fig. 12, *A* and *B*).

REVIEW OF LITERATURE

CLINICAL AND LABORATORY FINDINGS

General. Except for one of Jessner's original cases,[22] all subsequent patients reported to manifest the FDH syndrome have been females. All patients have been Caucasian, except for 2 Negroes.[24,31] Prematurity is common.[13,16,32,34,38] Slow growth may occur and the patients are sometimes small.[7,11,14,17,26,41] In addition to physical retardation, mental retardation is frequent.[12,13,14,33,40]

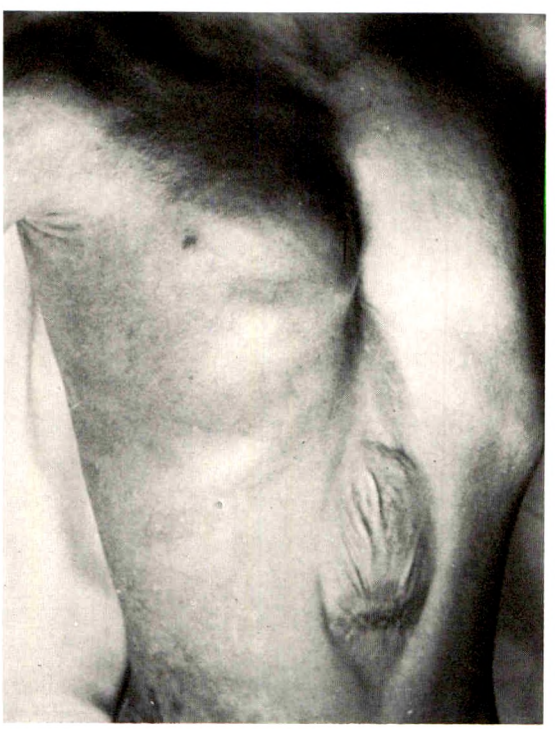


FIG. 3. Pectus excavatum. Ventral hernia represents an old omphalocele.



FIG. 4. Rudimentary tail. Note localized skin atrophy on posterior aspect of right thigh.

Skin and Skin Appendages. Skin lesions are universal. Typically, they are asymmetric. The patients usually present at birth with scar-like abnormalities: streaky hyperpigmentation; atrophy and telangiectasia.[14] Telangiectasia is especially common about the head and neck. Occasionally, "blisters" are present at birth[7,13] and evolve into the scar-like lesions. Fatty collections in the skin are also common. They have been variously described as nodules, papules, plaques and "baggy" collections. Their color varies from tan to yellow or pinkish-yellow. The iliac crest, groin and posterior aspect of the thigh are frequent sites. Unlike the scar-like changes, the fatty lesions are unique to the FDH syndrome.[13]

Arborescent papillomas of the oral mucosa and/or lips,[1,3–7,9,12,14,17,21–23,25,26,28,30,31,33–37,39] perianal region,[1,7,9,17,20,22,34,39,42] and genital mucosa[7,21,34,40] also occur. Single reports have described conjunctival papilloma,[36] papilloma of the external auditory meatus[40] and periungual "fibroma."[7]



FIG. 5. Gross appearance of Focal Dermal Hypoplasia skin lesions over right iliac crest, right groin and upper right thigh. These are soft, yellow and pinkish-yellow plaques mixed with hyperpigmentation and skin atrophy.

Hypotrichosis[2–4,6,7,10,14,16,17,20,23,26,27,33,34,36,40,42] and dystrophic nails[1–11,13,14,16,17,20,22,24,26,27,29,34,36,39,40,42] are very common. Spoon deformity of the nails also occurs.[6] Other findings include photosensitivity,[2,6,26,34,35] hypohydrosis,[6,10,34] hyperhydrosis,[4] palmar hyperkeratosis,[42] skin hyperelasticity[33] and acrocyanosis.[4]

Eyes, Ears, Nose and Mouth. Chorioretinal coloboma,[2,3,11,14,23,29,33,37,38,42] iris coloboma[10,11,14,23,32,33,37,38,40,42] and "eye coloboma"[12,41] are the most frequent ocular findings. Other eye anomalies include strabismus,[8,11,12–14,27,38] nystagmus,[13,14,23,38] obstructed tear ducts,[3,7,41] microphthalmia,[14,17,23,40,42] unilateral anophthalmia,[23,24] heterochromia iridis,[33,37] blue sclerae,[7] cataract[40,42] and subluxation of the lens.[40]

Reported anomalies of the ears include poorly modeled ears,[1,7,30,37,39] hypoplastic ear cartilages,[7,27,42] "satyr" ears,[6,27] preauricular tags,[7,22] deafness,[17,39] low-set ears[14] and a finger-like antitragus.[7]

Atresia of a nostril,[12,14] asymmetry of the nasal cartilages,[42] notching of an ala nasi,[12] a broad nasal bridge[27] and a flat nose[7] are reported nasal abnormalities.

Aside from the dental lesions, subsequently discussed under roentgenologic findings, soft tissue abnormalities of the mouth also occur. Microglossia,[2,24] cleft tongue,[13,14] double frenum[14] and perioral rhagades with leukokeratosis of the buccal mucosa[42] are examples.

Neck. A long, thin neck has been reported.[14]

Chest. A systolic murmur, thought to be due to aortic stenosis, was described in 1 of the patients reported by Goltz *et al.*[13] One

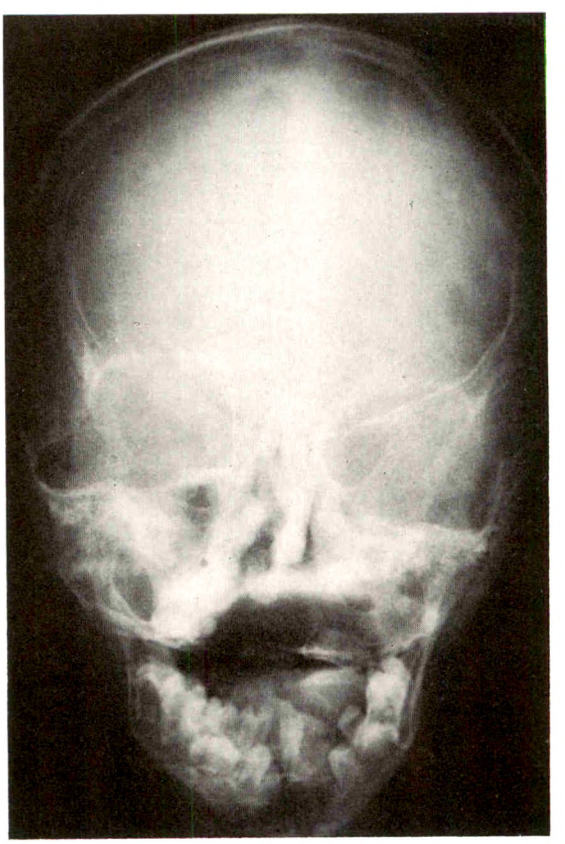


FIG. 6. Anteroposterior skull roentgenogram. Left petrous ridge is relatively low and left maxillary sinus is hypoplastic. Note presence of dental dysplasia.

of their other patients was later reported to have a systolic murmur.[14] The electrocardiogram of the second patient was normal, and no specific diagnosis was made. Other significant abnormalities of the heart have not been found in previous reports of the FDH syndrome. Small breasts have been described,[10,26,40] as well as pectus excavatum.[16]

Abdomen. Umbilical hernia,[13,14,27] inguinal hernia[27] and omphalocele[17] occur. Diastasis recti has been described by Naegeli,[31] and a "defect in the abdominal musculature" by Daly.[7]

Rectum and Genitalia. Hypoplasia is probably the most frequent abnormality of the genital system.[1,10,25,37,39,40] Nonpalpable ovaries have been recorded.[1]

Laboratory. Routine blood and urine tests show no abnormalities diagnostic of the FDH syndrome. Chromosomal studies have been reported in several cases, but are unremarkable.[14,17,20,24,40] To date, skin biopsy is the only diagnostic laboratory test. In essence, biopsy of a fatty lesion demonstrates normal adipose cells in the corium. Less specific changes are found in the scar-like areas. It is not within the scope of this paper to discuss the histopathologic changes in detail. The subject has been well covered by other authors.[13,18]

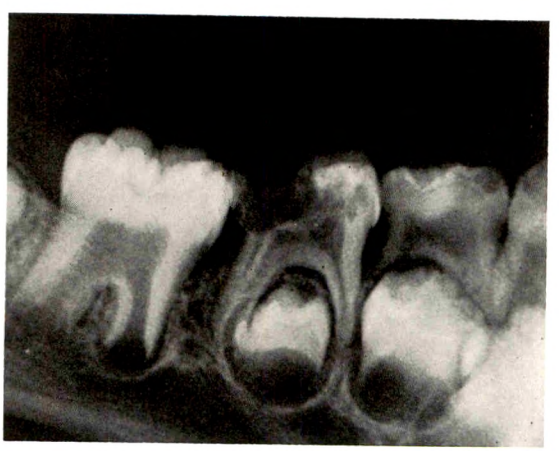


Fig. 7. Dental roentgenogram. Advanced caries is demonstrated, but the enamel is of normal thickness and normally calcified. Other roentgenograms (not shown) reveal that other teeth have crenated crown forms and the anterior teeth tend to be conical. Both deciduous and permanent teeth are similarly affected.

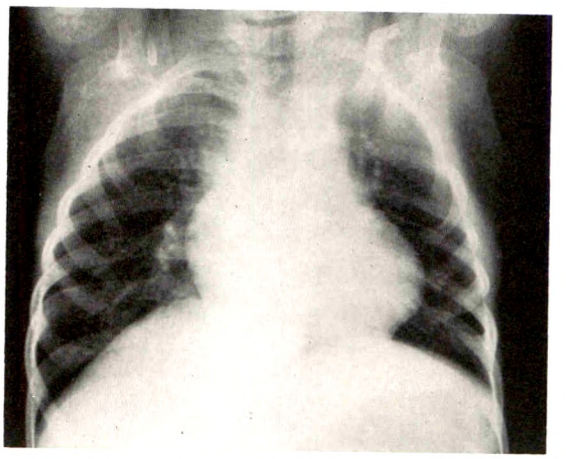


Fig. 8. Anteroposterior supine chest roentgenogram. The ribs have an abnormal pitch and some show "ribbon" deformity. Only eleven pairs are present. Thoracic scoliosis and spina bifida occulta of C_7 are also demonstrated. Note that right clavicle is almost aplastic, except for its lateral end.

Roentgenologic findings

General. Evaluation of the roentgenologic signs is difficult. Few case reports have included roentgenologic findings.[2,6,7,14,17,27,34,41]

Skeleton.

Craniofacial. Microcephaly is not uncommon.[12–14,27,39] Dyscephaly,[23] steep clivus[28] and asymmetric hypoplasia of the craniofacial skeleton[23] also occur.

Congenital and acquired dental diseases are common accompaniments of the FDH syndrome. The most frequent finding is hypodontia or oligodontia.[7,10,11,16,23,27,36,39] Other features include microdontia,[6,12,14,16,20,25] deformed teeth,[12,14,23] retarded eruption,[16,29,39] malposition of teeth,[34] enamel fragility[3,4,7,20] and "bad teeth."[14,20,26,27,29,42]

Spinal. Scoliosis is common.[7,12,16,17,36,39–41] Errors of segmentation occur: spina bifida occulta,[17,27,39,42] transitional vertebra[37] and "failure of segmentation."[17] A rudimentary tail has been reported by Martin-Scott.[29]

Pelvic. Hypoplastic pelvic bones have been recorded on 2 occasions[7,36] and hip

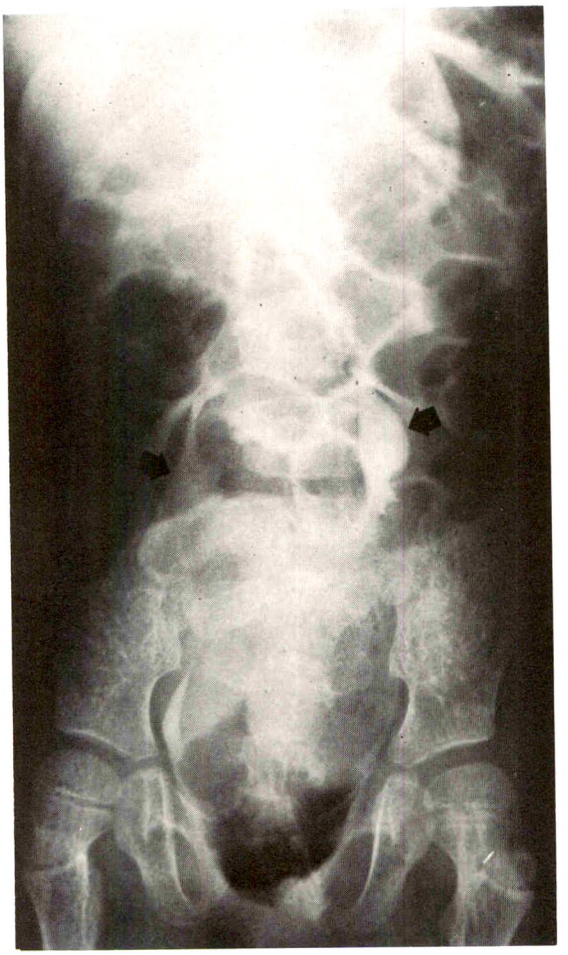


FIG. 9. Ten minute study from intravenous urography. Arrows indicate pyelocalyceal systems of ectopic and malrotated kidneys. They are probably fused and may have a horseshoe or disk configuration. Note curvilinear shadow of ventral hernia above kidneys.

dislocation once.[24] Hailey *et al.*[16] have reported cystic changes in the ilium, thought to be the result of low-grade osteomyelitis.

Thoracic. Most of the abnormalities involving the thoracic skeleton are errors of segmentation. Hypoplastic ribs[7] and bifurcated ribs[29,39] are examples. Clavicular aplasia and hypoplasia occur, especially on the right.[7,12,27,36,41]

Appendicular. Anomalies of size are common. The extremities, or parts thereof, may be shortened.[1,6,7,12,14,17,23,25,35,36,39,41] Brachydactyly can result from shortened phalanges, metacarpals or metatarsals.[2,5,17,20,39,40] Wodiansky[42] has reported an enlarged capitate. Clinodactyly is one of the abnormal shapes that occurs frequently.[1,6,11,23,36,39,41] Unilateral calcaneus valgus[23] and unilateral talipes[20] also occur.

Abnormalities of number, like the abnormalities of size and shape, tend to be asymmetric. They are especially common in the hands and feet. Examples include polydactyly,[13,14,28,31,40] oligodactyly and adactyly,[6–8,10–12,14,16,20,23,24,27,29,31,34–36,40,42] and syndactyly.[1–4,6,7,9–11,13,14,17,20,22–24,26–31,33,34,36,37,39,40–42] Bilateral syndactyly, especially between the third and fourth or second and third rays, appears to be the most common of these findings. Skeletal maturity of one hand, including the number of carpal ossification centers, may be different from the other.[7] Rook *et al.*[35] have described hypoplasia of the carpus, and aplasia of the fibula has been reported by Lanzieri.[23]

Viscera. Roentgenologic abnormalities of the heart and lungs have not been reported in any significant number of FDH syndrome patients. Only 2 reports have included urographic findings.[2,14]

DISCUSSION

Focal Dermal Hypoplasia syndrome is a



FIG. 10. Anteroposterior pelvic roentgenogram. The iliac wings are hypoplastic. The rudimentary tail is composed of enlarged coccygeal segments and curves toward the left. Note presence of osteoporosis and bilateral coxa valga.

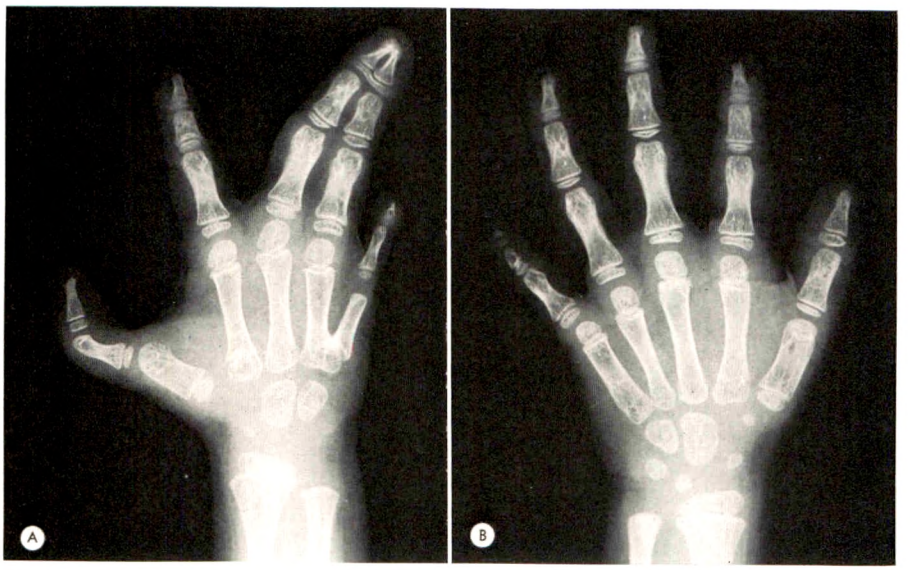


Fig. 11. Posteroanterior roentgenograms of the hands. (*A*) Right hand. Complex pattern of skeletal immaturity, syndactyly and brachydactyly with metacarpal shortening. The most pronounced changes involve the little finger. Note presence of proximally-placed thumb, as well as collar-button epiphysis in index finger at base of the middle phalanx. (*B*) Left hand. Except for skeletal immaturity (which is more pronounced on the right), abnormal changes are limited to brachyphalangism of little finger.

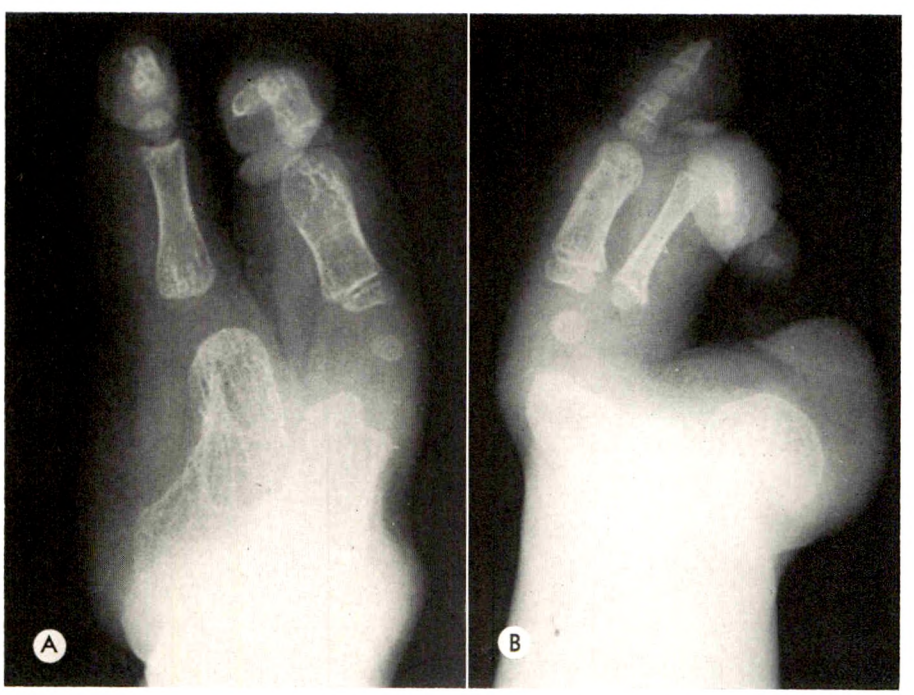


Fig. 12. Dorsoplantar roentgenograms of the feet. Brachydactyly and oligodactyly. The phalanges and metatarsals are hypoplastic. (*A*) Right foot. There is complete separation of the two rays. These probably represent the first and the third or fourth rays. The tarsus is underdeveloped. The talus and calcaneus are malformed. (*B*) Left foot. The metatarsals are bridged by soft tissue, unlike those on the right. These probably represent the first and second toes. The tarsal bones are similar to those on the right side. Note the presence of soft tissue bud arising from heel, probably representing an ectopic and hypoplastic ray.

rare, congenital dysplasia of systems derived from ectoderm and mesoderm. A review of the literature shows that there have been almost as many names applied to this syndrome as there have been case reports of it. Admittedly, Focal Dermal Hypoplasia syndrome does not describe it completely. But, as aptly pointed out by Daly, ". . . it is difficult to devise a designation which would begin to describe the many clinical facets and still be of practical length and equally acceptable to the many disciplines of medicine which encounter such patients."[7] One could not reasonably quarrel with other suggestions: ectodermal and mesodermal dysplasia with osseous involvement,[6] congenital ectodermal dysplasia and mesodermal dysplasia,[11] combined mesoectodermal dysplasia[17] or focal dermatophalangeal dysplasia.[40]

CLINICAL

With rare exceptions, the patients are *Caucasian females*. A history of prematurity is frequent and some physical and/or mental retardation is not uncommon. Lesions of the skin and its appendages are universal and diagnostic features of this syndrome. The most characteristic lesions are *superficial fatty collections in the skin*. Originally thought to be subcutaneous fat that herniated through areas of focal dermal hypoplasia, more recent histopathologic studies by Howell[18] have indicated that these fatty collections may be linear hamartomas, *i.e.*, nevoid neoplasms. Focal areas of skin atrophy, linear hyperpigmentation and telangiectasia of the skin constitute a second type of lesion. In some patients these *scar-like changes* are preceded by "blisters," present at birth. We wonder if similar vesicles are the intrauterine precursors of the scar-like lesions present at birth. *Dystrophic nails* are almost a constant feature. *Papillomas* of the periorificial skin and oral mucosa are common, as is hypotrichosis.

Ocular lesions, especially *colobomas*, are frequent. Our patient manifested a corneal opacity and ocular hypertelorism—findings not previously described in the FDH syndrome. Other clinical abnormalities about the head, such as *malformed ear and nasal cartilages*, are less common than the ocular lesions. Stenosis of an external auditory canal, elongation of a tragus and webbing of the neck were present in our patient. Like the new ocular findings, these signs have not been described in previous reports of this syndrome.

On the basis of available clinical evidence, it does not appear that a significant number of cardiac or pulmonary abnormalities occur in the FDH syndrome. Except for some *hernias of the abdominal wall*, clinical examination of the abdomen is usually unremarkable. Pelvic examination sometimes reveals hypoplastic genitalia. Our patient demonstrated another new finding, anal stenosis.

The occurrence of a *rudimentary tail* in the FDH syndrome has been described only once before the present case report. Clinically, it appears to be similar to that found in metatropic dwarfism.[38]

The clinical differential diagnosis, particularly for the skin lesions, is comprehensively covered by Holden and Akers.[17]

ROENTGENOLOGIC

Roentgenologic abnormalities are virtually limited to the skeletal system. The most common changes are *asymmetric anomalies of size, shape and number*. Typically, these involve the appendicular skeleton and teeth. Syndactyly between the second and third or third and fourth rays of the appendages, and partial anodontia or malformed teeth, are prime examples. The patient herein reported also demonstrated a collar-button epiphysis in the hand and a proximally placed thumb. These findings are also new to this syndrome. Random hypoplasia of the long bones is common. The osteoporotic, gracile appearance of our patient's long bones and minor malformations of her joints were thought to be due to disuse during the developmental period. Similar changes have been described by Holden and Akers.[17] Coxa valga

and the tall vertebral bodies demonstrated by our patient were also ascribed to non-ambulation.

The most common roentgenologic abnormalities of the thorax are errors of rib segmentation and clavicular underdevelopment. Only eleven rib pairs were present in our patient. Some of the ribs had the "twisted ribbon" appearance seen in neurofibromatosis. Previous reports of the FDH syndrome have not reported these abnormalities. *Clavicular hypoplasia and aplasia* are found in only a small proportion of these patients. However, because such abnormalities occur so rarely outside of this syndrome and cleidocranial dysostosis, they are findings worthy of note. For some reason, as yet unknown, such clavicular anomalies predominate on the right.

Similarly, roentgenologic abnormalities of the spinal column are usually errors of segmentation. Accompanying *scoliosis* is common. A rudimentary tail is rare, but like the clavicular anomalies, this abnormality is noteworthy.

With the exception of those roentgenologic changes secondary to chronic crippling, the remainder are congenital anomalies that can occur in the absence of the FDH syndrome. Their incidence in other conditions can cause diagnostic confusion. Some examples have already been mentioned; *e.g.*, neurofibromatosis, metatropic dwarfism and cleidocranial dysostosis. Others include incontinentia pigmenti, chondroectodermal dysplasia, oculoauriculovertebral dysplasia, Hallermann-Streiff syndrome, Moebius syndrome, Rothmund-Thomson syndrome, trisomy 13–15 and trisomy 17–18. The roentgenologic signs of each have been well reviewed in the literature.[15,18,19]

GENETICS

Among the cases from which we can gather family pedigree information (Fig. 13, *A* and *B*), there is little evidence of a genetic basis for this syndrome. All cases described thus far, except for two,[13,14] have been isolated examples. Only in those two

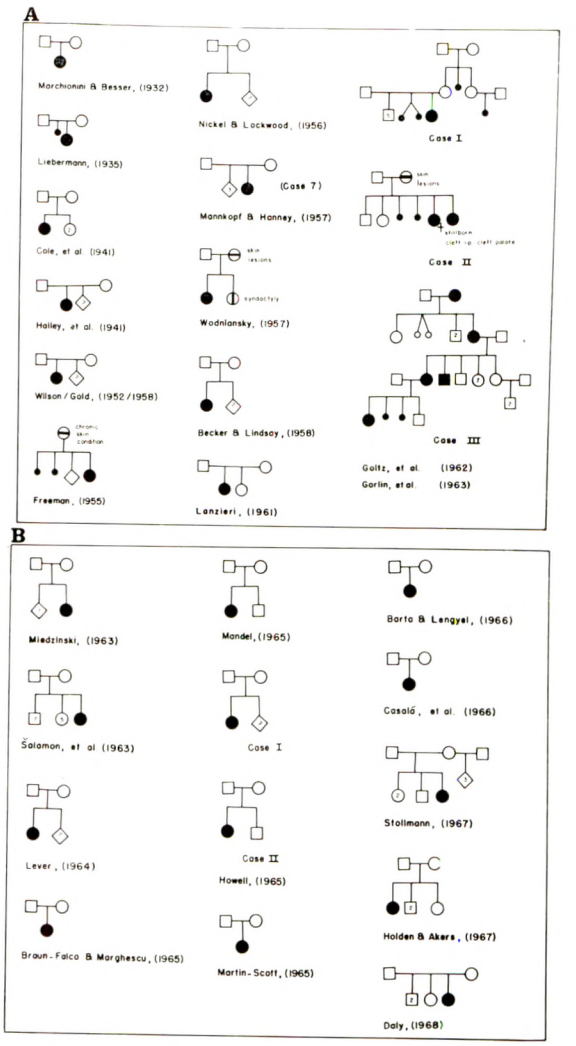


FIG. 13. (*A* and *B*) Genetic pattern of Focal Dermal Hypoplasia syndrome cases described in the literature.

exceptions have the pedigrees demonstrated transmission of the condition through the mother, to the succeeding generation. But, with few exceptions,[13,14,26] the probands have been unmarried. Nearly all of the probands have been children at the time of report. Thus, very few of the probands have had the opportunity to reproduce, and our genetic evaluation is necessarily based on statistically inadequate data.

It is possible that this condition is inherited as an X-linked dominant trait, lethal in the male[13,29] and producing markedly reduced fertility in the female, but this

is only a hypothesis. It is of some interest that an analogous pattern may be seen in incontinentia pigmenti.

CONCLUSIONS AND SUMMARY

1. Focal Dermal Hypoplasia (FDH), a congenital dysplasia of ectodermal and mesodermal structures, has protean manifestations.

2. The patients are almost exclusively Caucasian females.

3. Clinically, this condition can be identified by the typical skin lesions. These are often accompanied by ocular anomalies, such as colobomas, and malformed ear or nasal cartilages.

4. The only definitive test for this condition, to date, is skin biopsy.

5. This syndrome should be suspected roentgenologically when there are asymmetric anomalies of size, shape and number involving the axial or appendicular skeleton. The combination of syndactyly and anomalous dentition is typical. While rare, a rudimentary tail and a hypoplastic or aplastic clavicle may also point to the diagnosis.

6. On the basis of limited genetic data, it is postulated that the FDH syndrome may be inherited as an X-linked dominant trait.

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CONGENITAL HYPOPLASIA ALONG THE RADIAL RAY*

A REPORT OF TWO CASES

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ACCORDING to Gegenbaur[3] the development of the hand occurs partially from an ulnar ray and partially from a radial ray. The radial, or first ray of the forearm includes the radius, the radiale, the first carpal bone, the first metacarpal bone, and the 2 phalanges of the thumb. Also, according to embryologic theory, interference with a proximal and more primitive element will most likely be manifest as absence or hypoplasia of its distally associated elements.

The first report of a case of congenital hypoplasia along the radial ray was, according to Kato,[5] in 1733. Kato[5] and others[2,7] considered Gegenbaur's archipterygeal theory to be the most likely explanation for the apparently separate and distinct variants of this defect. The reported cases have varied from complete absence of the radius and its distally embryologically associated elements to mere hypoplasia of these structures.

Davison[2] has suggested a classification according to the degree of hypoplasia of the carpal navicular (scaphoid). Apparently this bone arises, in part, from the radiale bone.[4] There had been no reports on isolated familial and congenital thumb deformities until Bilbrey[1] described a study of 13 members of one family. He suggested that this defect is transmitted by a dominant gene with incomplete penetrance.

The 2 patients presented here are mother and son and both show such a condition unassociated with any other abnormality.

REPORT OF CASES

CASE 1. This patient, a clerk aged 25 years, came to the outpatient department of the University Hospital because of pain in the left wrist and elbow. On examination he was noted to have unusually small and abducted thumbs. Roentgenologic examination of the left elbow revealed no abnormality. Roentgenograms of the left hand showed a short and deformed first metacarpal, somewhat hypoplastic thumb phalanges (Fig. 1*A*), a hypoplastic radial styloid process, and a slightly hypoplastic navicular (Fig. 1*B*). Roentgenograms of the right hand showed fusion of the greater multangular (trapezium) with the first metacarpal (Fig. 1*C*; most obvious when comparison is made with Fig. 4, *B* and *D*), hypoplasia and deformity of the navicular and hypoplasia of the radial styloid process and of the thumb phalanges (Fig. 1*D*). The first metacarpal was seen to be irregularly formed and thinner than the other normal metacarpals, and an indentation was visible at the site of fusion of the greater multangular and the first metacarpal (Fig. 1*C*). There was subluxation of the first metacarpophalangeal joint and abduction of the thumb.

The patient is the younger of 2 children. His father has no known skeletal defects, but his mother has bilateral congenital hypoplasia of the thenar muscles (Case II, see below). His mother's mother also had congenital hypoplasia of both thumbs, but she is now deceased and no further information is available. There is no other history of any skeletal defects in the family.

The patient himself has had no serious illnesses. The records of his anomaly go back to the age of 9 months when he was noted to have "bilateral thumb abduction without much tension." The first roentgenograms were taken when he was 2 years old (Fig. 2, *A* and *B*) and showed hypoplasia of both first metacarpals and slight subluxation at both first metacarpophalangeal joints. Since then he has been followed, and early arthrodesis was considered as he had "bilateral hypoplastic first metacarpals

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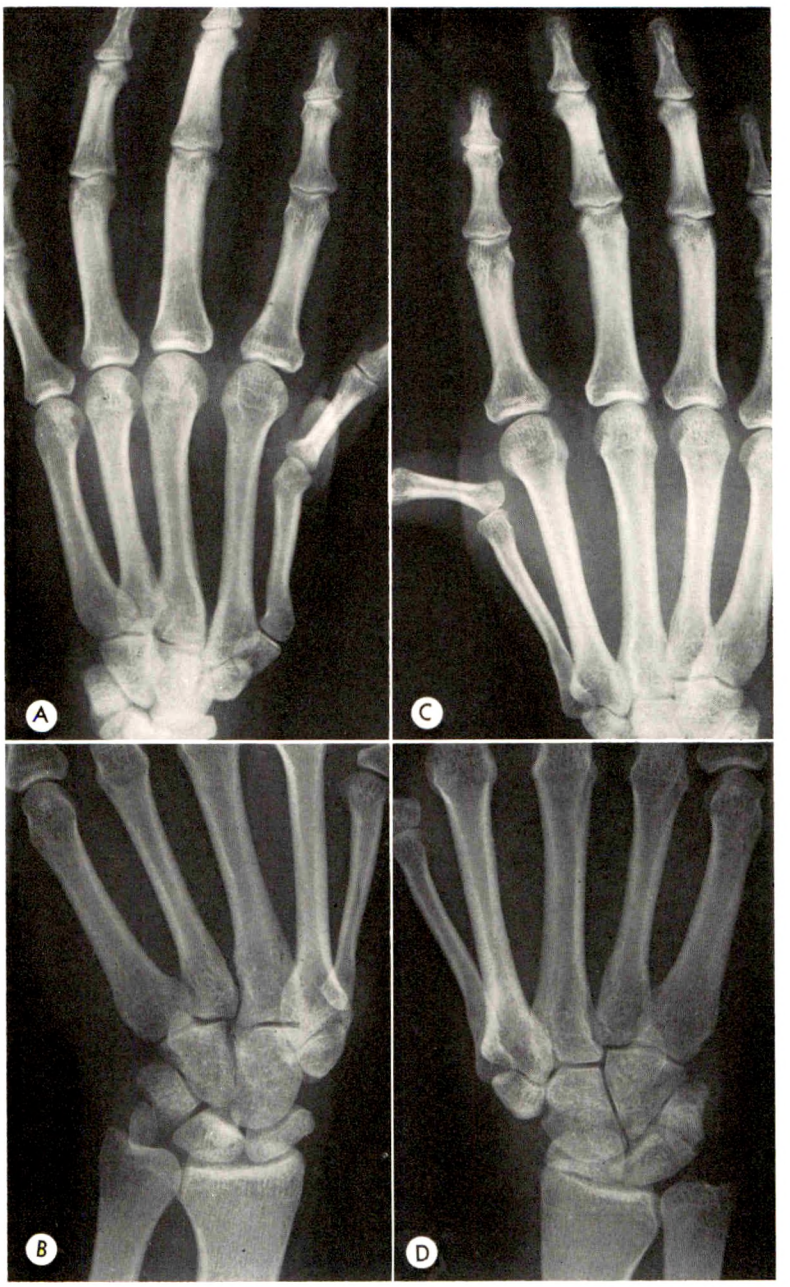


Fig. 1. Case 1. (*A*) Left hand. Deformed first metacarpal; hypoplastic phalanges. (*B*) Left hand. Hypoplastic styloid process; hypoplastic navicular bone. (*C*) Right hand. Greater multangular bone fused with first metacarpal; hypoplastic phalanges; subluxation in first metacarpophalangeal joint. (*D*) Right hand. Hypoplastic styloid process; hypoplastic navicular bone.

without any active movement of his thumbs." At the age of 5 years, roentgenograms (Fig. 3, *A* and *B*) showed both first metacarpals to be hypoplastic and deformed with absence of the proximal epiphysis of the distal phalanx of the right thumb. It was noted that he had "in his left thumb some degree of flexion and extension in the metacarpophalangeal joint, whereas in his right thumb movement was possible only at the carpometacarpal joint." It was hoped that his thumbs would eventually increase sufficiently in size to benefit from surgery.

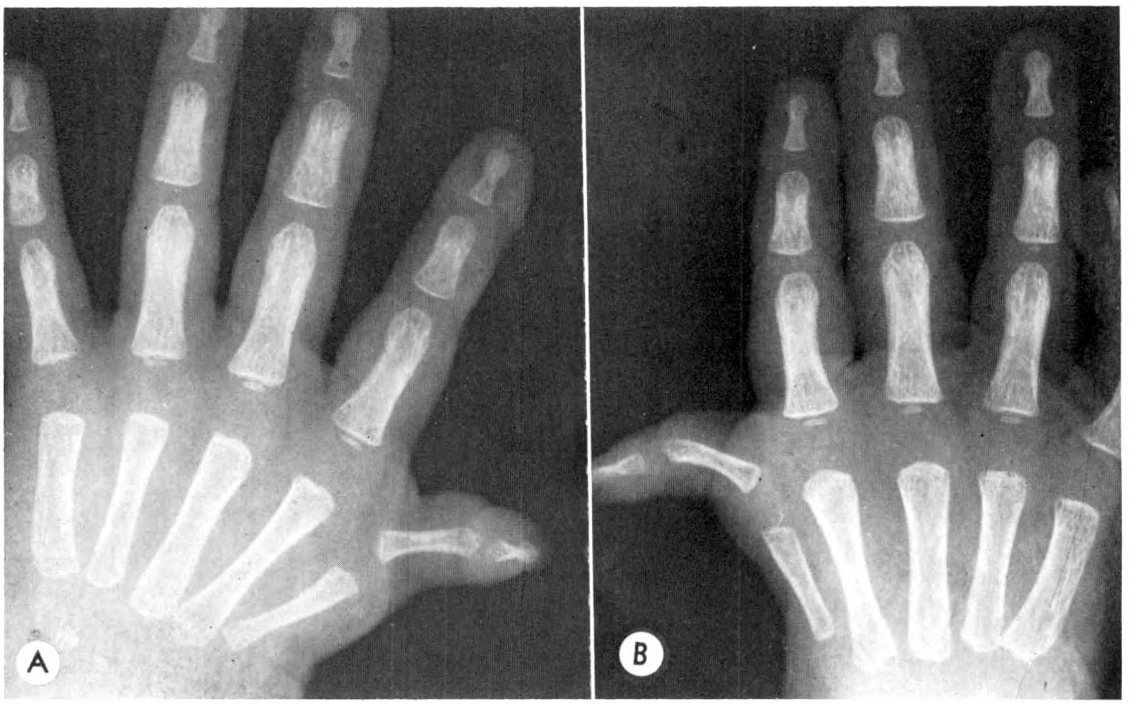


FIG. 2. Case 1. (*A* and *B*) At 2 years of age. Bilateral hypoplastic first metacarpal; slight subluxation in first metacarpophalangeal joints.

At 13 years of age the patient still had hypoplastic and abducted thumbs associated with poorly developed thenar muscles and first metacarpals. Roentgenograms at that time (Fig. 4, *A* and *B*; and *C* and *D*) showed hypoplasia of the navicular bones and radial styloid processes, a small and cylindrically malformed right greater multangular, and hypoplasia of both first metacarpals and of the phalanges of both thumbs. Surgery was then undertaken on the left hand and 2 years later on the right hand. The procedure was basically a transposition of the extensor indicis and the palmar part of the flexor sublimis tendons from the index finger to the rudimentary extensor and flexor pollicis longus tendons, respectively, at appropriate sites. The right greater multangular bone later became fused with the first metacarpal (compare Fig. 4, *B* and *D*; and Fig. 1*C*). This fusion must have been spontaneous; the operation was confined to the tendons without any surgery to the bones of the hand or wrist, and there were no postoperative complications such as inflammation.

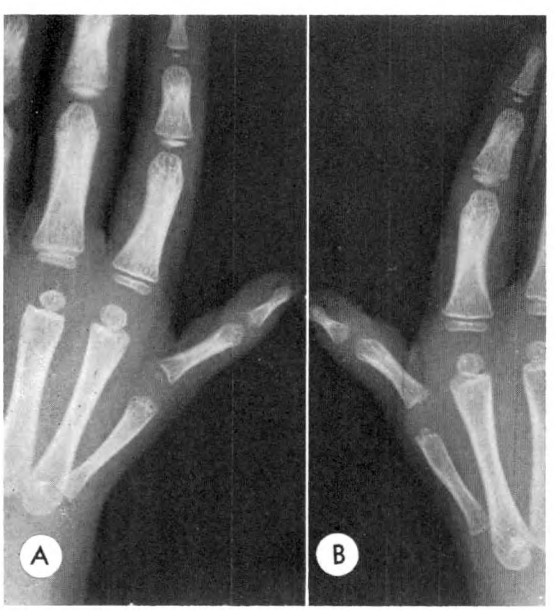


FIG. 3. Case 1. (*A* and *B*) At 5 years of age. Bilaterally deformed, hypoplastic first metacarpals; absent proximal epiphysis of distal phalanx of right thumb.

CASE II. This patient is the 46 year old mother of the first patient. She also has bilateral congenital hypoplasia of the thenar muscles, and roentgenographic examination of her hands (Fig. 5, *A* and *B*) showed hypoplasia of both radial styloid processes, a short right first me-

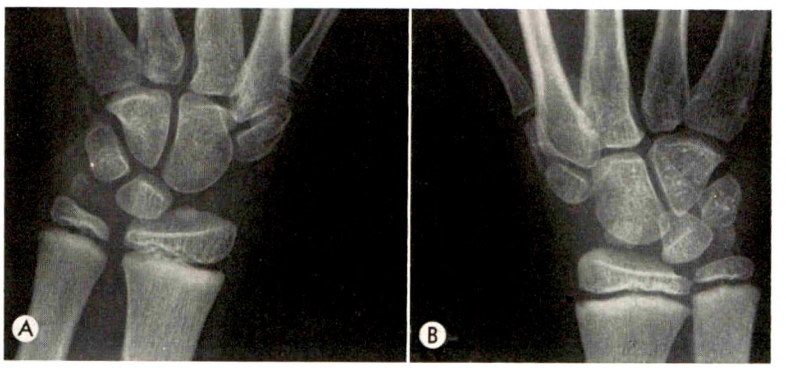


Fig. 4. Case 1. (*A* and *B*) At 13 years of age. Bilaterally hypoplastic navicular bones and styloid processes.

tacarpal and a slender left first metacarpal. Roentgenogram of the left wrist (Fig. 5*C*) revealed a dysplastic bipartite navicular with a distal fragmented component, having a small distal articulating surface, and a small proximal component.

DISCUSSION

Case 1 exhibits a defect which is consistent with Type 2 of Davison's[2] classification, *i.e.*, absence of the radial styloid process, deficient thenar musculature, hypoplastic or absent navicular and greater multangular bones, and hypoplasia of the thumb.

We believe that this case gives support to Bilbrey's[1] hypothesis that the defect is transmitted by a dominant gene, as we have evidence (roentgenographic and anamnestic) for 3 consecutive generations. The penetrance of the gene appears here to be complete in contrast to the incomplete penetrance suggested by Bilbrey.[1] To this date no similar cases have been reported in the Swedish medical literature.

The fusion of the first metacarpal with the greater multangular seen in our Case 1 (compare Fig. 1*C* with Fig. 4, *B* and *D*) has not been reported previously. However, it has been noted that there is a general tendency for fusion to occur in hypoplastic joints.

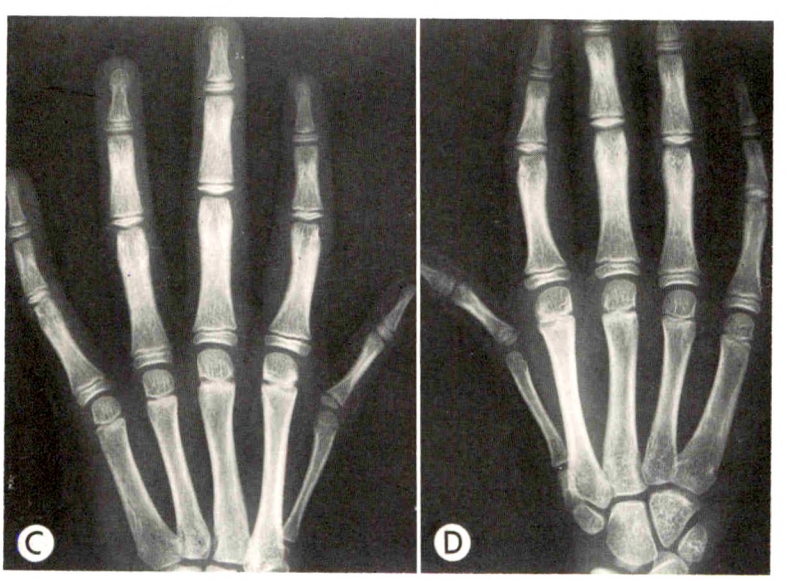


Fig. 4. (*C* and *D*) Bilateral hypoplastic first metacarpals and thumb phalanges; malformed right greater multangular bone.



FIG. 5. Case II. (*A* and *B*) At 46 years of age. Bilateral hypoplastic styloid processes; shorter right first metacarpal; thinner left first metacarpal.



The functional improvement following surgery has been satisfactory to the extent that the patient was able, 1 year after the final operation, to become a clerk—an occupation which requires at least average writing ability. Four years after the final operation he was, furthermore, able to serve in the Swedish army without significant handicap.

SUMMARY

Two patients with congenital hypoplasia along the radial ray are reported (mother and son).

In one of the cases there were repeated roentgenologic examinations covering a pe-

FIG. 5. (*C*) Left wrist. Dysplastic, bipartite navicular bone.

riod of 23 years. Comparison is made with previously reported cases.

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DISTAL RADIAL GROWTH PLATE REMNANT SIMULATING FRACTURE*

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INDIVIDUAL variations in development are not only intriguing roentgenologic curiosities but not infrequently may be interpreted as abnormal with resulting overtreatment or mismanagement. A recently recognized normal variation in growth and development of the distal radial epiphysis may be misinterpreted as a fracture.

REPORT OF A CASE

An 18 year old male was examined because of pain in his wrist persisting 6 days following an injury. Roentgenograms demonstrated a faint radiolucent line in the distal radius, adjacent to the volar and ulnar edge of the joint surface (Fig. 1, *A*, *B* and *C*). The poorly marginated defect could only be visualized on the 30° to 45° semisupination oblique views. The lucency was tentatively diagnosed as a hairline fracture. Two weeks later the line was unchanged, and the patient was asymptomatic.

DISCUSSION

The probability that the lucency represented a remnant of the growth plate stimulated a review of 120 recent wrist examinations in adolescents 13 to 19 years of age. The examinations were obtained for a variety of reasons, but all had been interpreted as normal. Two cases showed the distal radial growth plate to lie close to the joint on the semisupination view, similar to the presented case. Figure 2 is a roentgenogram of one of the patients with a painful wrist of 1 month's duration and no history of trauma. The growth plate is just beginning to close in this 17 year old male, and there is no difficulty in recognizing that the fine lucencies at the arrow are part of the plate. The second patient was a 16 year old girl, whose roentgenograms are illustrated in Figure 3, *A–D*. She had recently been in an automobile accident and recovered uneventfully without casting. The growth plate is partially obliterated. On the semipronation view the growth plate appears to touch the joint cartilage—a projection phenomenon seen frequently in this age group (Fig. 3*B*). The semisupination and the lateral views show the proximity of the plate to the joint on the ulnar and volar edges (Fig. 3, *C* and *D*). In this case also, the defect is readily recognized as part of the plate which is still largely visible.

The usual shape of the distal radial epiphysis is demonstrated in Figure 4, *A* and *B*. The illustrations of Hall[2] and the roentgenograms of Greulich and Pyle[1] show this type of epiphysis where the growth plate lies 3 mm. to 5 mm. from the joint surface. Usually the medial aspect of the plate obliterates early so that it is not a cause of confusion. The relative persistence of the plate on the lateral aspect is frequently seen and recognized as normal. When the position of the plate medially is unusual, and when the medial plate persists unusually long, the combination of features may cause the erroneous diagnosis of fracture.

SUMMARY

A normal variation in the development of distal radial epiphysis which may be misinterpreted as a fracture is demonstrated. In some individuals the plate is close to the joint surface, and when the plate becomes partially obliterated may simulate a fracture.

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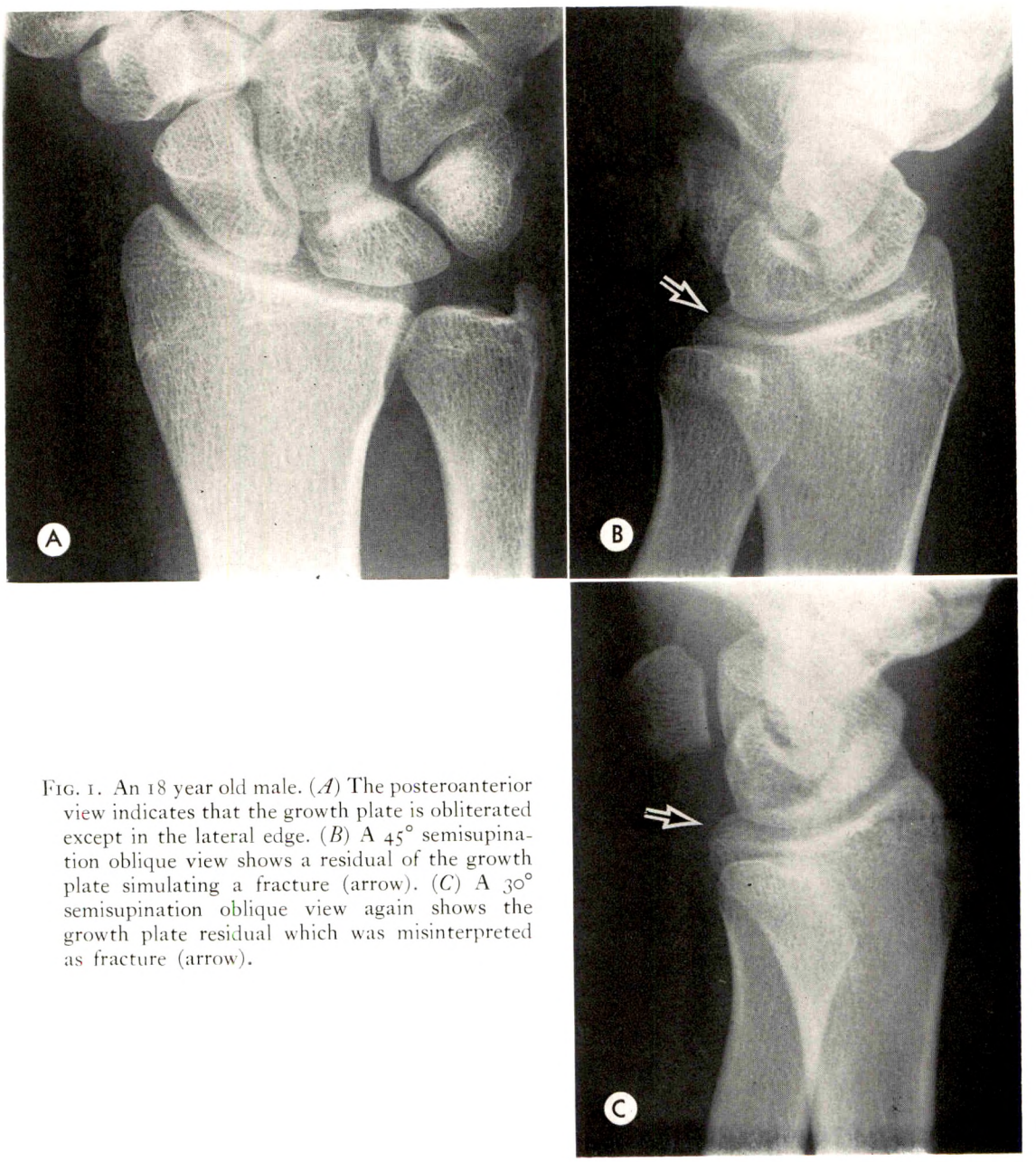
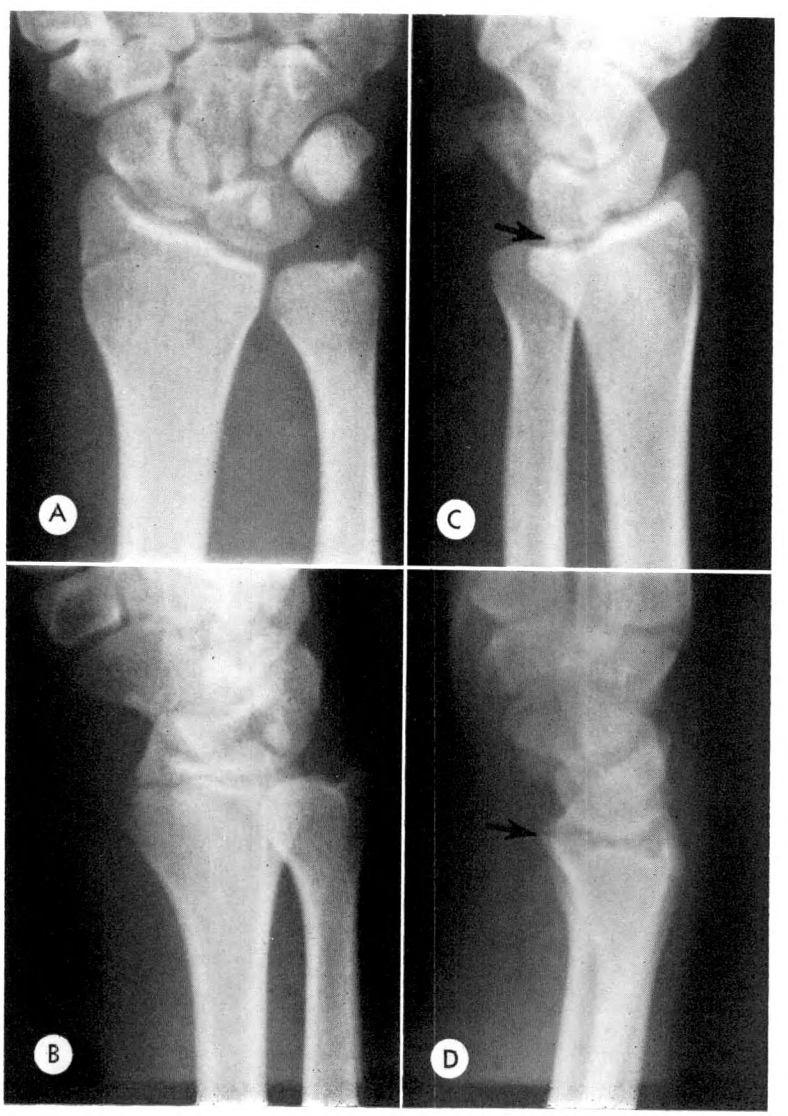


Fig. 1. An 18 year old male. (*A*) The posteroanterior view indicates that the growth plate is obliterated except in the lateral edge. (*B*) A 45° semisupination oblique view shows a residual of the growth plate simulating a fracture (arrow). (*C*) A 30° semisupination oblique view again shows the growth plate residual which was misinterpreted as fracture (arrow).



FIG. 2. A 17 year old male. Several delicate radiolucent lines indicate the growth plate residual adjacent to the joint surface (arrow). The growth plate is just beginning to close, and the lines offer no diagnostic dilemma.



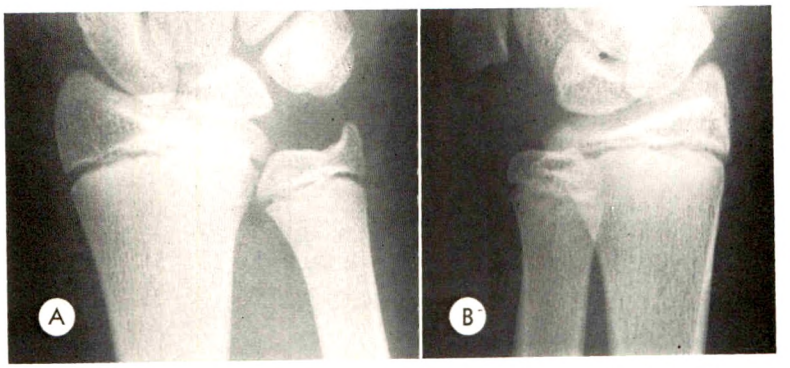


Fig. 4. A 13 year old male. (*A*) The posteroanterior view shows the usual relationship of the growth plate to the wrist joint. (*B*) The 45° semisupination oblique view shows the usual separation of the plate from the joint. Persistence of the plate medially in this location causes little confusion.

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Fig. 3. A 16 year old female. The growth plate is beginning to close. (*A*) Posteroanterior view shows the plate in the lateral aspect. (*B*) A 45° semipronation view shows the plate apparently crossing the joint surface, an effect due to projection. (*C*) A 45° semisupination oblique view showing the plate immediately adjacent to the joint at the medial and volar edge (arrow). (*D*) Lateral view of the same feature (arrow).

LYMPHANGIOMATOSIS OF BONE DEMONSTRATED BY LYMPHANGIOGRAPHY*

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MULTIPLE angiomatosis of bone is an infrequently occurring disease entity. In 1964, a review of the literature revealed only 11 reported cases.[9] At least 5 additional cases have since been reported.[7,11,12] The presently reported case is added to the literature because the diagnosis was established by lymphangiography. This has not been previously reported.

REPORT OF A CASE

J. R., a 13 year old Caucasian male, was admitted to Childrens Hospital of Los Angeles October 21, 1969, for evaluation of swelling of the right lower extremity of 2 months' duration. During the year preceding admission, spotty pigmentation of this limb was noted.

This patient had been hospitalized at the Children's Hospital of Pittsburgh at the age of 3 years because of right thigh pain. Evaluation revealed leukocytosis with shift to the left, fever, and tenderness about the distal thigh. Roentgenograms of the entire right lower extremity demonstrated a solitary, lytic, rather large lesion in the metaphysis of the distal femur. No other abnormalities were noted. The diagnosis of osteomyelitis was made and confirmed by surgical curettement. Staphylococci were cultured from the purulent material obtained.

This boy recovered without sequelae, and had been asymptomatic through the years until the onset of his current symptoms.

Positive physical examination findings, at the present hospitalization, were limited to the right lower extremity. This extremity was slightly larger proximal to the ankle as compared to its companion limb, but there was no disparity in length. Several areas of hyperpigmentation in the skin were noted, associated with mild (1+), firm, pitting pretibial edema. No tenderness or neurologic alteration was found. The clinical impression was lymphedema with secondary skin change.

Routine laboratory studies were normal. Serum calcium (9.6 mg./100 ml.) and alkaline phosphatase (4.9 Bodansky units) were also within normal limits.

Roentgenograms of the right lower extremity revealed multiple small cortical radiolucent defects confined to the metaphyseal and adjacent diaphyseal regions of the proximal tibia and both ends of the femur (Fig. 1, *A* and *B*). A few of the larger defects were within the medullary cavity of the proximal tibia. These lucent areas were sharply marginated and many had fine sclerotic borders. Bone modeling was not affected. The thickness of the cortex in uninvolved areas was normal.

A complete skeletal survey revealed no other bone abnormality. Venography of the right lower extremity was normal.

Bilateral lymphangiography was accomplished. Films were exposed at the completion of contrast material injection and 24 and 48 hours later. Widespread abnormalities of the lymphatic system were demonstrated. These changes were characterized by dilated lymphatics with delayed drainage and a paucity of lymph nodes in the involved regions (Fig. 2, *A* and *B*). The lymph channels in the right lower extremity were dilated. A large plexus of abnormal lymphatics occupied the right inguinal region with collateral drainage to the sacral lymph nodes and along the iliac crest to lower lumbar channels. The lymph vessels of the iliac and paraaortic regions were also dilated. No lymph nodes were noted in the right inguinal, iliac, or paraaortic areas. Retrograde filling of several intercostal lymphatics occurred bilaterally (Fig. 3). The lymphatic structures in the left lower extremity appeared normal.

There was delayed drainage of contrast material from the right lower extremity. Considerable contrast medium remained in dilated ab-

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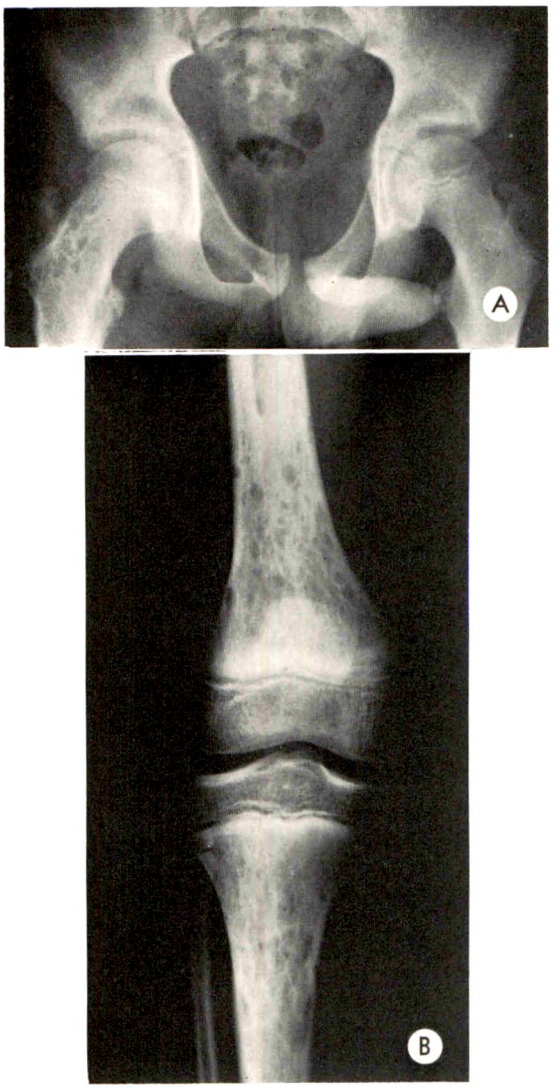


Fig. 1. Anteroposterior roentgenograms of (*A*) the lower pelvis and hips and (*B*) right knee at 13 years of age. There are multiple radiolucent defects in the metaphyses and adjacent diaphyses. The smaller lesions are confined to the cortex, while some of the larger lesions also involve the medullary portion of these bones. Fine sclerotic margins outline many of the defects.

normal channels and small lymph nodes at 48 hours, at which time contrast material was demonstrated for the first time within one of the cortical lesions in the distal femur (Fig. 4, *A* and *B*). This location was confirmed with tomograms.

This patient was discharged following the completion of the roentgenographic studies.

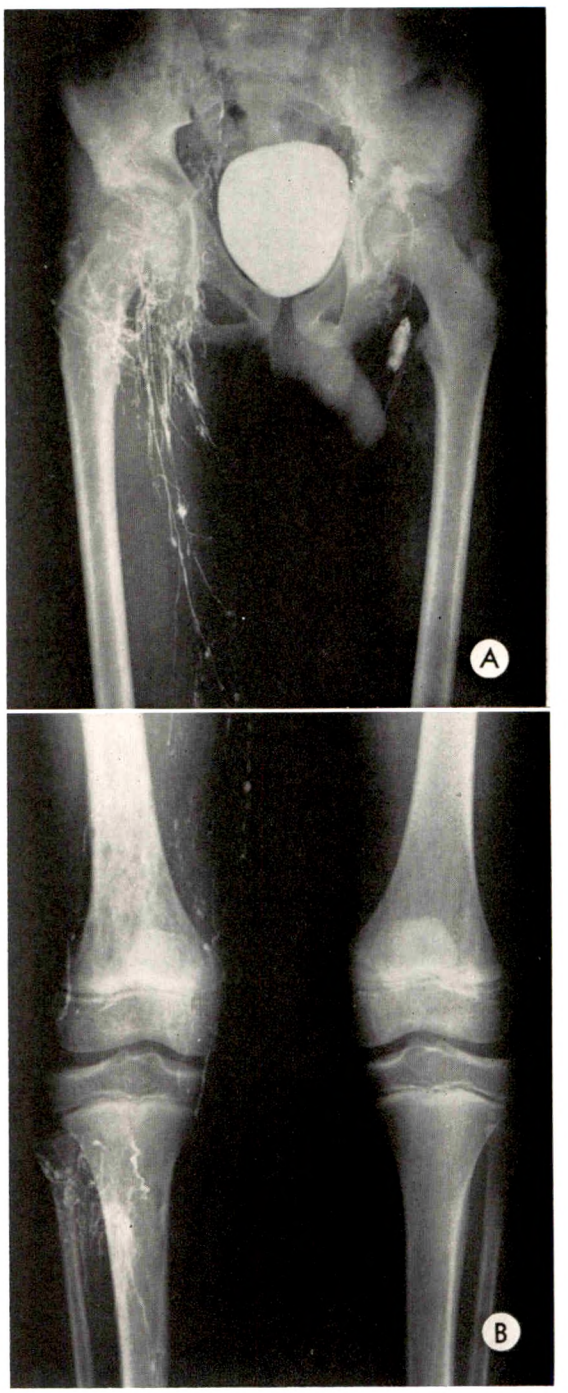


Fig. 2. (*A* and *B*) Bilateral lymphangiograms at 24 hours. Dilated lymphatic channels with delayed drainage are seen in the right lower extremity. A large plexus of abnormal lymphatics is noted in the right inguinal region. There are no lymph nodes demonstrated in the right inguinal or iliac regions. There is no abnormality on the left.

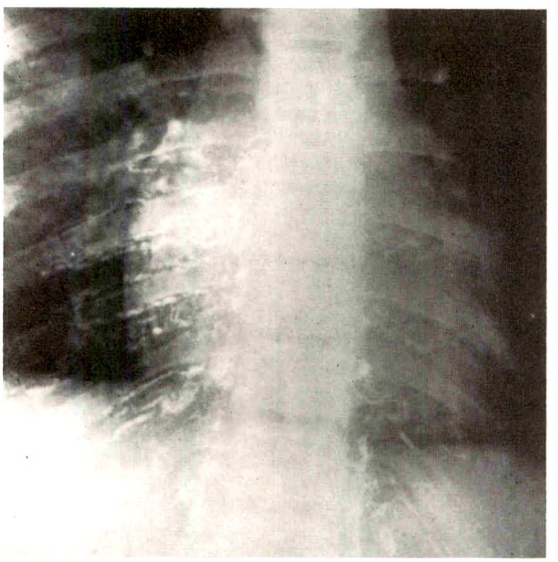


FIG. 3. An anteroposterior view of the thoracic region during lymphangiography reveals abnormal retrograde filling of several intercostal lymphatics.

The definitive diagnosis rendered by lymphangiography precluded the need for biopsy.

DISCUSSION

The pathogenesis of angiomatous bone disease is poorly understood. Various etiologies have been suggested and reviewed.[1] A congenital malformation is the most likely cause. Inflammation, neoplasm, and lymphatic obstruction have also been suggested as the basic cause.

The radiolucent areas in bone apparently result from pressure atrophy from ectatic endothelial-lined channels. These lesions may originate from lymphatic tissue (lymphangiomatosis), blood vascular tissue (hemangiomatosis), or both. Microscopic differentiation of these similarly endothelial-lined cystic lesions is difficult and often impossible. Small biopsy specimens frequently fail to demonstrate any definite abnormality. These diagnostic difficulties have been noted by several authors.[3,5,9,11,12] Because of the problem in definitive diagnosis, an inclusive term, cystic angiomatosis has been suggested.[6,9]

The age at which these lesions are roentgenographically detectable, or their duration before the disease becomes clinically manifest, is unknown. Most reported cases were in the 10 to 15 year age group, although a few were seen in early childhood. No previously reported case has had completely normal skeletal roentgenograms before angiomatous bone changes occurred. Additional bone lesions may develop, however, during the course of the disease. In one case,[8] only 4 ribs were involved at 1 year of age. Three years later, most of the long and flat bones were involved.

The course and prognosis of this disease vary. Patients with only bone involvement are generally asymptomatic, but pathologic fractures have been a frequent complication. Otherwise, their outlook seems good. Bone lesions have usually been slowly progressive. Long-term follow-up of these patients has not been reported, and the eventual result remains speculative. One case report extended over an 11 year period with minimal progression of the bone lesions.[4]

Five of the 11 cases reviewed by Moseley and Starobin[9] had multiple organ systems involved in the angiomatous disease. When this occurs, it is usually fatal with chylous effusions being the prominent factor.

ROENTGEN APPEARANCE

The bone lesions of angiomatosis are invariably radiolucent, and usually finely etched by a thin, sclerotic margin. A portion of a lesion may be quite ill-defined, however.

The lesions affect both the cortex and the spongiosa of the medullary cavity, and are characteristically widespread in distribution. The medullary lesions may be quite large with mild expansion of the bone and thinning of the cortex.

The skull and flat bones, as well as the long bones, are usually involved. Defects have been noted less frequently in the spine and scapula. The small bones of the extremities have been spared.

Demonstration of the type of endothelial-lined tissue, causing the bone changes with the use of contrast media, has not previously been reported. In one case,[10]

opaque medium was injected directly into a skull lesion and communication with adjacent areas in a multilocular cystic region was demonstrated. Lymphangiography has been performed in 3 reported cases. None showed loculation of contrast medium in the bone lesions. Two of these patients[7,10] had an associated soft tissue lymphangioma in the inguinal region. The third case[11] had bone lesions in both lower extremities and bilateral chylous pleural effusions. Lymphangiography demonstrated abnormal retrograde filling of several intercostal lymphatic channels on one side. No lymphangiographic abnormality was noted below this. We are aware of one unreported case[2] of lymphangioma of a lumbar vertebra which had contrast medium from a lymphangiography loculated within the bone lesion at 24 hours.

The present case report suggests that delayed filming may be of value in demonstrating contrast medium within bone lesions when abnormal lymphatics with delayed drainage are present in the affected limb.

DIFFERENTIAL DIAGNOSIS

The roentgenograms of this case present the diagnostic problem of evaluating polyostotic lucencies in childhood. Several diseases must be considered when confronted with this situation. Histiocytosis X, fibrous dysplasia, metastases, Gaucher's disease, congenital fibromatosis, neurofibromatosis, and enchondromatosis should be considered in the differential diagnosis. Careful analysis of the routine roentgenograms should lead one to consider the diagnosis of angiomatous disease.

The relative sparing of the medullary cavity in preference to the cortex would eliminate those diseases which are primarily marrow replacement processes, either local or generalized, such as fibrous dysplasia and Gaucher's disease.

Histiocytosis X and metastatic disease are frequent causes of multiple radiolucent bone lesions. These can be dismissed in the presence of sharp margination and finely sclerotic borders.

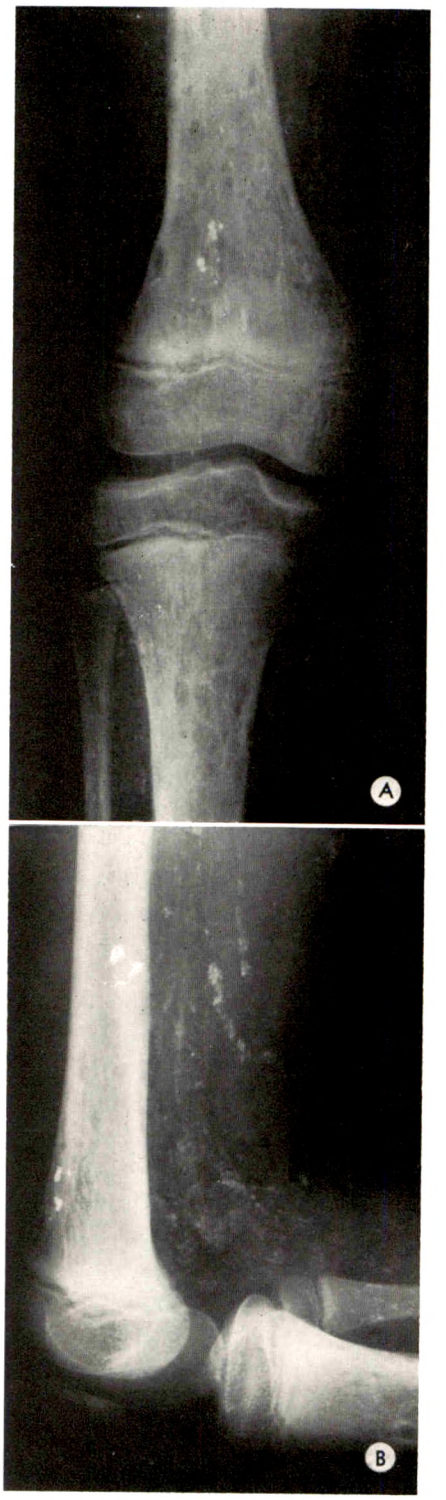


Fig. 4. Delayed lymphangiograms at 48 hours. (*A*) Anteroposterior and (*B*) lateral views show contrast material loculated within one of the bony lesions in the distal femur.

Congenital fibromatosis is a disease of infancy. If the patient survives, the bone lesions apparently resolve by an early age. The long bone lesions are metaphyseal in location. They appear to arise in both the medullary cavity and cortex. The lytic defects are large.

Neurofibromatosis rarely causes localized lytic bone changes. A few cases have been reported with multiple cystic bone lesions which were large and seemed to arise within the medullary cavity.

Enchondromatosis often involves only one lower extremity. The radiolucencies are usually elongated. Round lucencies, arranged in columns, may occur. There is often a failure of modeling of the metaphyseal region. These defects arise from the epiphyseal plate without preferential involvement of the cortex.

SUMMARY

A case of lymphangiomatosis of bone, demonstrated by lymphangiography, is reported. This uncommon disease should be considered in children with polyostotic lytic lesions.

The diagnosis can be confirmed with lymphangiography if contrast material is demonstrated within the bone lesions. In the presence of delayed drainage of the contrast medium from the lymphatics, delayed filming may be necessary. This confirmatory demonstration precludes the need for biopsy.

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EARLY DIAGNOSIS OF CONGENITAL DYSPLASIA OF THE HIP; NEW ROENTGENOLOGIC SIGNS*

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CONGENITAL dysplasia of the hip (CDH) is very frequent in the area attended by the Vimercate General Hospital. Every year more than 1,500 infants of about 6 months of age are brought as outpatients to the Department of Radiology for check-up of the conditions of their hips, so that almost all children born in our district are subjected to a roentgenographic examination of the pelvis. A high percentage of them show clear signs of CDH, including femoral dislocation, but many cases present some difficulty in the early diagnosis of CDH. Our main diagnostic problem is to screen normal from borderline cases, to be able to treat them and follow them, thus avoiding later complications such as premature arthrosis.

Numerous methods are reported[1,4] for the diagnosis of CDH, based on changes in the relations between the acetabulum and the upper end of the femur, on the obliquity of the acetabular roof, or on the delayed and irregular ossification of the nucleus of the femoral head. Some classical works of the older literature[7] describe roentgenologic findings of CDH even in early stages, but recent studies on the changes of initial and slight degrees of CDH are rare.[1,5,6] Our method[6] is to consider mainly the little alterations in the upper end of the acetabular roof, since it is here that the very early signs of dysplasia are located and, in mild cases, only in this location.

We emphasize that femoral dislocation, even though slight, is too severe a sign of dysplasia to be considered as an early sign; in fact, many of our borderline or initial cases do not show dislocation at all.

This affirmation may seem to be in contradiction with the work of some authors[2,4] who consider dysplasia secondary to dislocation. A possible explanation for this is that our roentgenographic material concerns a sort of mass screening examination and not a study of cases affected by serious pathology exclusively.

OSSEOUS SIGNS

The main osseous changes we encountered are limited areas of sclerosis in the upper end of the acetabular roof. In very initial cases the only pathologic image is a small triangular area of increased density as shown in Figure 1*A*. A shallow groove appears in other cases (Fig. 1*B*). In still more advanced cases a more or less deep fossa is observable in the outer part of the acetabular roof (Fig. 1*C*), as described by Doberti and Manhood.[5]

During recovery from dysplasia, the mild findings tend to disappear; in some cases a bony spur develops (Fig. 2).

NONOSSEOUS SIGNS

Deriving all the possible information from a standard roentgenogram of the pelvis, we have observed an image that has not yet been reported in the relevant literature we consulted. It is an arched image of slight radiopacity which delineates from the surrounding soft tissues as a curve cephalad and convex laterally, starting from the upper edge of the acetabular roof and reaching the femur at the greater trochanter (Fig. 3). The finding is well demonstrated in correctly exposed roentgenograms but may be invisible on too contrasted films. Using a fairly high kilovoltage so as to reduce the radiation dose absorbed by the small patients, we avoid strong contrasts, thus visualizing soft tissues.

In our opinion, the "arcuated" image

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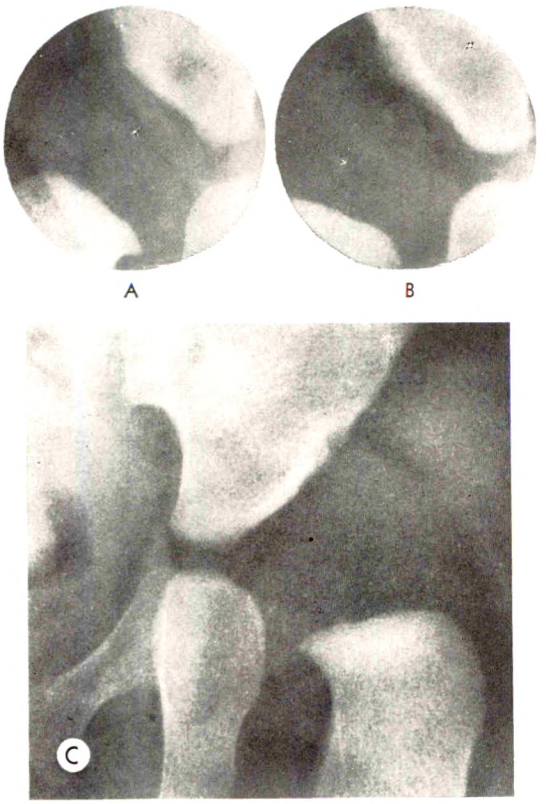


Fig. 1. (*A*) Small triangular area of sclerosis at the upper end of the acetabular roof. (*B*) Shallow groove. (*C*) Fossa at the outer third of the acetabular roof.

represents the head of the femur encircled by the articular capsule and, in normal cases, it forms an ideal arch with the outline of the acetabular roof and the acetabular part of the pubis (Fig. 4). The arcuated line becomes more evident as the



Fig. 2. Left hip with dysplasia in the process of recovery. Note the bony spur at the upper end of the acetabular roof.



Fig. 3. Right hip normal. Left hip shows extremely slight sclerosis of the acetabular roof. The "arcuated line" is visible on both sides with a normal appearance.

child grows older; enclosed in it appears the cephalic nucleus of the femur. It provides information on the shape and position of the femoral head before the appearance of the nucleus. Figures 1 to 3 show this image; printing of nonosseous images is not easy and original roentgenograms are more demonstrative than reproductions.

In case of CDH, the ideal arch arcuated line—acetabular profile may be interrupted and the arcuated line may be in the above-described acetabular groove (Fig. 5). In case of femoral dislocation, it becomes enlarged and deformed, losing its sharp circular contour (Fig. 6). Furthermore, in advanced cases of luxation with serious delay in the appearance of the cephalic nucleus, it may define the shape and position of the still cartilaginous head.

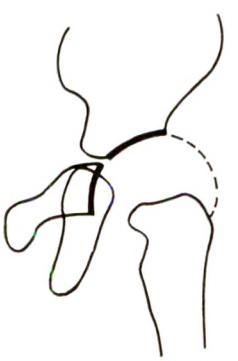


Fig. 4. Schematic drawing of a normal arcuated line (dotted). This line makes an ideal arch with the acetabular roof and the acetabular part of the pubis (heavy lines).



Fig. 5. Dysplasia on the right. The arcuated line is visible in the acetabular groove, interrupting the ideal arch "arcuated line"–acetabular profile.



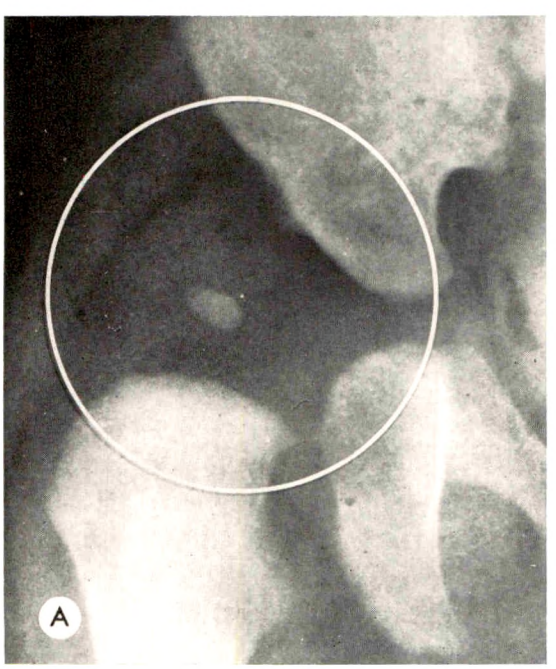
Fig. 7. Slightly lateralized femora, small acetabula, normal arcuated lines. Subsequent controls show delay in the ossification of nuclei in normal position.



Fig. 6. Dislocation on the left. Deformation and obscuration of the arcuated line, which is normal and well visible on the opposite side.

The arcuated line may give information about prognosis; as shown in the case in Figure 7, the femora are slightly lateralized and the acetabula small, but the arcuated lines are normal. Subsequent controls show delay in the ossification of nuclei in normal position.

In some cases of acetabular dysplasia we observed a pathologic nonosseous image, not yet described. It is a radiolucent triangular area limited caudad by the arcuated line, medially by the dysplastic



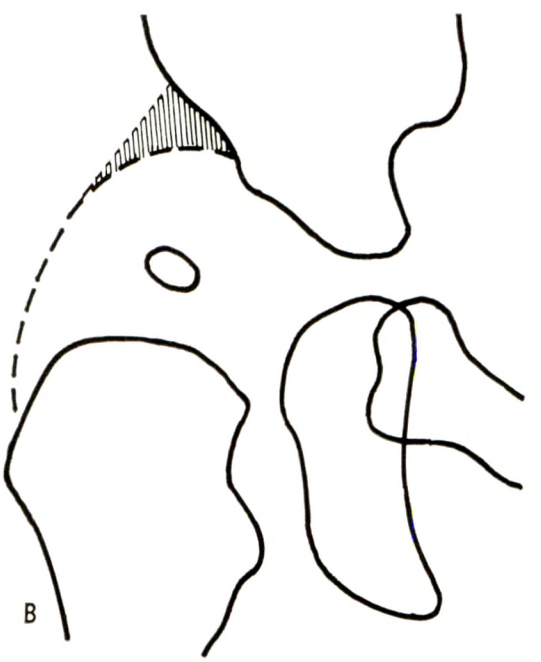


Fig. 8. (*A*) This figure shows the arcuated line and the triangular area of radiolucency in dysplasia, explained by (*B*) the schema. Note the fossa at the outer third of the acetabular roof.

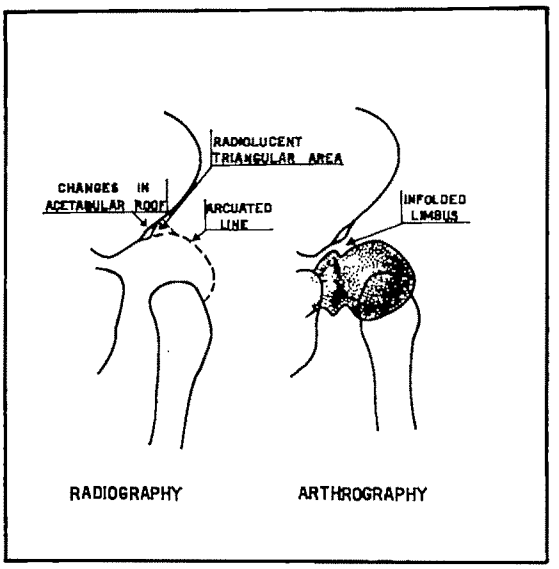


FIG. 9. Schematic drawing of the above described roentgenograms compared to an opaque arthrogram in a case of a dysplastic hip with a slight degree of dislocation.

acetabulum and laterally by the soft tissues of higher radiopacity due to muscular masses (Fig. 8, *A* and *B*). This area is not visible in normal cases and we ascribe it partially to the fibrocartilaginous limbus, enlarged and infolded, as is observed in the arthrographic examination of dysplastic hips.[3] The signs we have described are given in the schema of Figure 9 and are compared with those of an opaque arthrogram, which explains suggestively our assertions.

CONCLUSIONS AND SUMMARY

In studying a population strongly affected by congenital dysplasia of the hip and examining almost all the children of about 6 months of age born in this area, we observed a noteworthy number of borderline and initial cases of dysplasia.

Our findings lead us to the conclusion that initial roentgenologic signs of dysplasia begin at the upper end of the acetabular roof. These signs represent dysmorphic changes, such as sclerosis, a shallow groove, or a fossa.

Moreover, we would like to call attention to the importance of the nonosseous images for the study of the position and shape of the cartilaginous head of the femur and the pathologic changes of the fibrocartilaginous limbus.

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HEALING IN CONGENITAL OSSEOUS SYPHILIS*

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UNFORTUNATELY syphilis has not been eradicated and is still a problem in our modern society.[3] According to epidemiologists the incidence of syphilis is increasing and it is reported that instances of penicillin resistant lues are being encountered. Despite the justifiable fears and apprehensions of these epidemiologists, syphilis, at least with roentgenologic manifestations, is a relatively rare phenomenon. Furthermore, the ease and efficacy of antiluetic therapy has virtually eliminated congenital syphilis. Nevertheless, babies are born with syphilis and although it is an academic exercise to use roentgenography as a diagnostic procedure, serial studies can be invaluable in determining the effectiveness of therapy.

The objective of this study is to detail the characteristic features of healing in penicillin treated congenital osseous syphilis, as this has never been adequately described or demonstrated.

CLINICAL MATERIAL

Of the 20 cases of congenital osseous syphilis in this series, 12 had no roentgenograms subsequent to therapy. The roentgenograms served to amplify and complement the demonstrations of pathology in the treated cases. Three of the infants were followed at intervals of 2–4 weeks, until healing was advanced. In 5 others roentgenograms were obtained only once following therapy.

There was a total of more than 40 involved metaphyses which were clearly demonstrated and closely followed. Many of the lesions were not only bilateral and symmetric, but the metaphyses involved in any patient were generally in the same stage of evolution. The principal variations from this pattern were seen where pathologic fractures distorted or callus formation obscured the underlying detail. Aside from the skull all luetic lesions were confined to the tubular bones.

The initial metaphyseal lesion, prior to therapy, consisted of a poorly defined mottled radiolucent band usually contiguous with the zone of provisional calcification, breaching some or all of the cortex as well as the periosteal new bone when present (Fig. 1; and 2).[1,2,9] Bilateral symmetric involvement of the medial aspect of the proximal tibial metaphyses, Wimberger's sign, was clearly seen in 4 cases. Fractures with displacement or impaction through the involved metaphyses were frequent (Fig. 3; and 10).

Following the institution of therapy there was an orderly progression of healing and all the involved metaphyses responded similarly; at no time was there indication of an acceleration of the disease process.[4] Within 2 weeks there was unequivocal evidence of healing and in 3 weeks healing was well advanced (Fig. 3). This was mani-

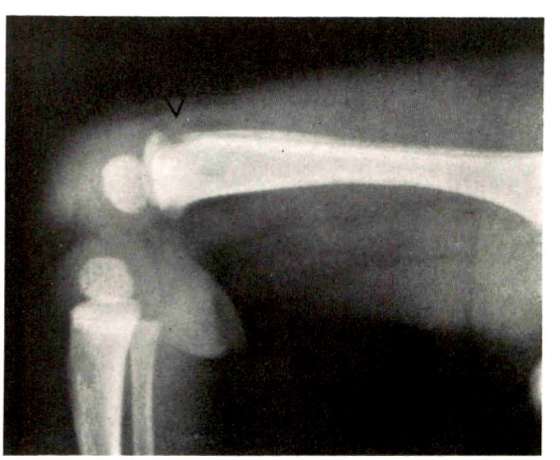


FIG. 1. July 19, 1963. Prior to treatment there is a lesion of the distal femoral metaphysis with destruction of the adjacent periosteal new bone.

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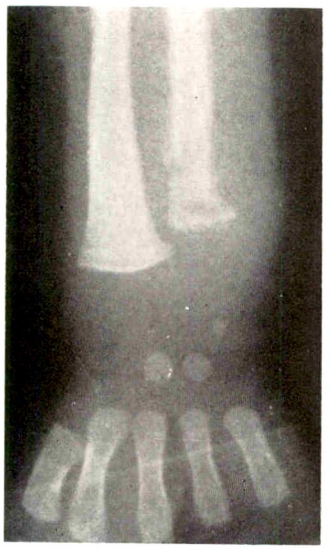


FIG. 2. July 19, 1963. The initial ulnar metaphyseal lesion shows a fracture with minimal displacement.



FIG. 4. August 26, 1963. The bone ends are united by bridging callus.

fested by absorption of detritus, deposition of bone in the lesional tissues and smoothing of the contours of the metaphyses. When impaction or displacement was present, the opposing bone ends united before realignment by modeling occurred (Fig. 4).

In approximately 2 months healing was complete or almost so (Fig. 5). The configuration and the trabecular pattern of the metaphyses were normal except where there had been impaction or fracture with displacement. The metaphyses in these cases showed residual deformity and a smudged trabecular pattern distinct from that in the diaphyses. Healing was complete thereafter and in roentgenograms obtained 5 months after therapy anatomic



FIG. 3. August 8, 1963. A considerable amount of detritus has been absorbed and there is now impaction.

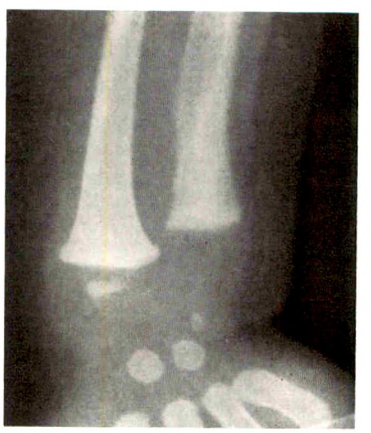


FIG. 5. September 23, 1963. Healing of the ulna is now complete.



Fig. 6. July 19, 1963. There is a fracture through an area of destruction in the proximal metaphysis of the humerus.



Fig. 8. September 23, 1963. The callus is being incorporated into the bone and modeling is well advanced.

realignment of bone was present except in 1 case (Fig. 12, *A* and *B*).

In 2 cases exuberant callus surrounded the proximal metaphyses of the humeri or femora. In each of the 5 areas of involvement callus formation appeared to be consequent upon fracture. In 1 case the initial roentgenogram showed only the destructive metaphyseal lesion and a faint fracture without callus formation (Fig. 6). Within 10 days following the institution of therapy, abundant spongy callus appeared. Subsequently there was a slight increase in the extent of the callus but there was a pronounced increase in its density (Fig. 7). Within 1 month the callus solidified and united with the cortex. Within 2 months there was considerable modeling and in 1 year the configuration was normal (Fig. 8; and 9). In the case showing exuberant callus of both proximal femoral metaphyses as well as lesions in the distal femora, follow-



Fig. 7. August 5, 1963. Heavy callus sheaths the lesion in the proximal metaphysis of the humerus.

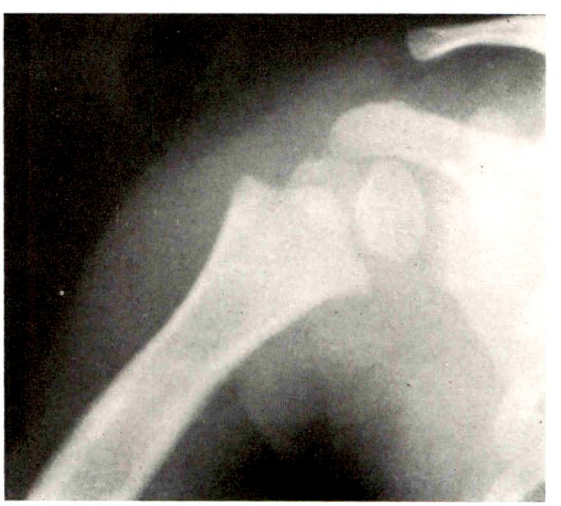


Fig. 9. July 7, 1964. There is now complete healing of the humerus.

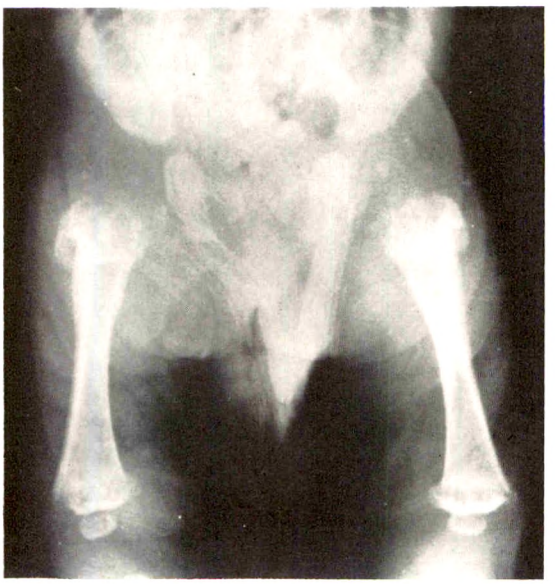


FIG. 10. February 26, 1968. There are impaction fractures through the proximal femoral metaphyseal lesions and destructive lesions in the distal metaphyses prior to treatment.

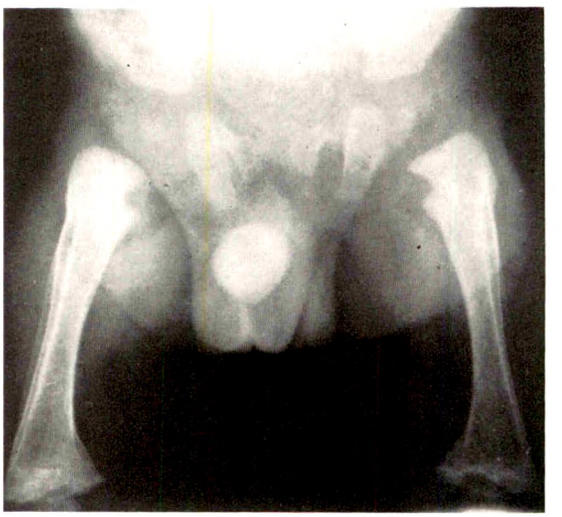


FIG. 11. April 16, 1968. There is advanced healing in all areas of involvement but deformity of the femur is now obvious. Note the extensive inert periosteal new bone formation.

up examinations at 7 weeks showed advanced healing. There was, however, an indeterminate type of deformity present and there was periosteal new bone proliferation over the lateral aspect of the femoral shafts (Fig. 10; and 11). At 5 months the contours of the femora were unchanged but osseous healing was complete. Two years following therapy there was moderately severe anterolateral bowing of both femora (Fig. 12, *A* and *B*). These femora were the most severely involved in the entire series. There was never clinical

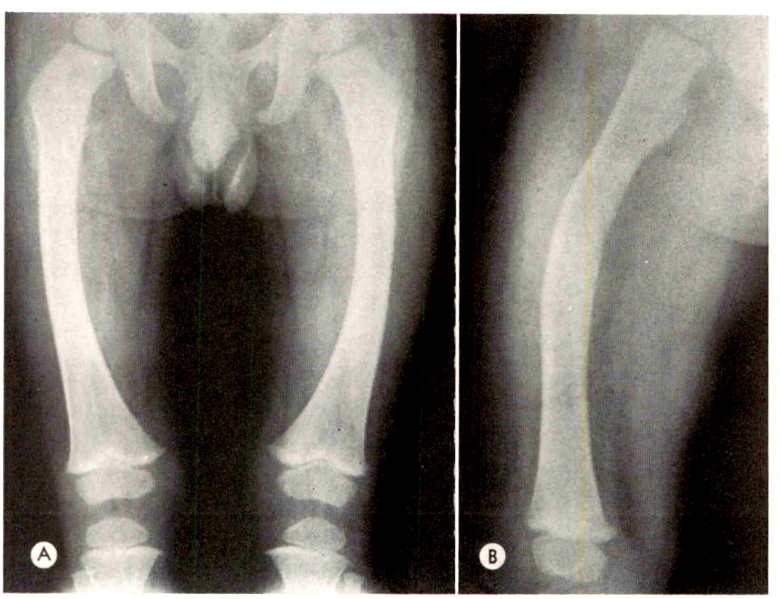


FIG. 12. February 3, 1970. (*A* and *B*) The metaphyseal lesions have healed completely but there is anterolateral bowing of the femora.

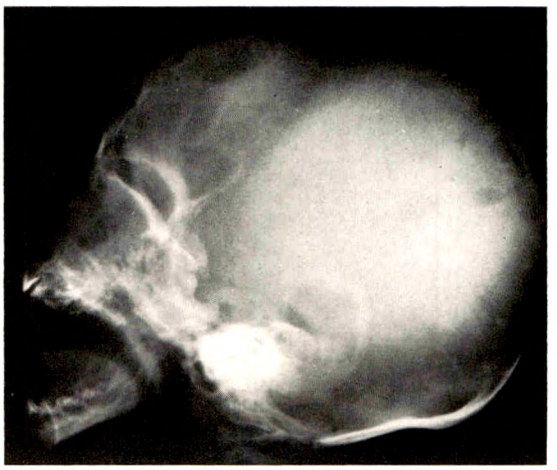


Fig. 13. May 4, 1962. Multiple oval lesions are scattered in the calvarium but are concentrated and confluent over the glabella.

or roentgen evidence of rickets or other systemic abnormality.

Calvarial lesions were seen in only 1 case. These consisted of numerous indistinctly marginated, rounded radiolucent defects averaging 1–1.5 cm. in diameter (Fig. 13). In the frontal area, particularly in the glabella, the lesions became confluent. Eighteen days following therapy only hazy remnants of the defects remained (Fig. 14).

The most unusual presentation was in the sigmoid notch of the ulna. This localization of the disease has not been mentioned in the literature but it occurred in 6 cases

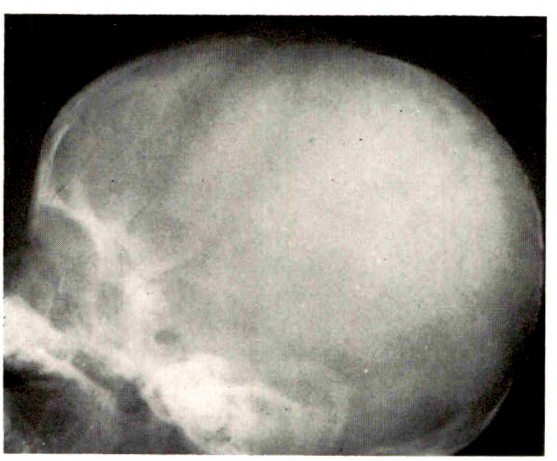


Fig. 14. May 19, 1962. There is almost complete resolution of the lesions leaving only a hazy suggestion of the initial process.



Fig. 15. February 19, 1968. There is a gross erosive process in the sigmoid notch of the ulna with additional lesions in the radius and humerus.

and is so characteristic as to be virtually pathognomonic of syphilis. The articular surface was grossly eroded and at times there was also ragged disorganization of the adjacent metaphyses of the radius and humerus (Fig. 15). Within 3 weeks healing was well advanced, in 2 months it was almost complete, and in 5 months healing was complete (Fig. 16; and 17).

Periosteal new bone formation was never a prominent feature in this series of cases and the hypertrophic cloaking, "periostitis of Pehú," was never seen. Small strips of



Fig. 16. March 11, 1968. Twenty days after the institution of therapy there is early but definite healing.



FIG. 17. April 16, 1968. The sigmoid notch and the radius are normal but there is still residual irregularity of the distal humerus.

periosteal new bone tended to occur shaftward of metaphyseal lesions and increased in extent following therapy. During the healing phase, a thin layer or multiple thin layers of new bone occurred over many diaphyses which were otherwise roentgenographically normal. Within 5 months even the most extensive laminations were incorporated invisibly into the cortex. Lastly, exuberant callus, "periostitis callosa of Schneider," was seen over some fractures, particularly in the shoulder and hip, and resembled a club. Within 2 months the new bone had consolidated and coalesced with the adjacent bone and within 1 year healing was complete without residual deformity.

DISCUSSION

Regardless of the duration or virulence of the maternal disease, luetic infection of the fetus does not occur before the fifth month of intrauterine life and it may not occur until the day of delivery.[10] There is a lag period of approximately 6 weeks between the initial infection and the appearance of changes around the osteochondral junction, regardless of the stage of fetal development.[8] When maternal infection occurs late in pregnancy or is untreated, lesions may not appear in the infant until 6 to 8 weeks after birth.[8]

The roentgenologic patterns of congenital osseous syphilis are well known and when distinct are probably even more diagnostic than the Wassermann test.[6] McLean[10] and others[2,6,7] have described the roentgenographic features of congenital lues and correlated them with postmortem findings. McLean[10] found lesions in every bone but the greatest number were in the long bones.

The earliest roentgen evidence of lues is change in the architecture of the zone of provisional calcification. It may become thicker and more dense or it may disappear entirely.[11] On magnification with a hand lens, tiny prongs or teeth may be seen extending into the epiphyseal cartilage giving a serrated or saw-toothed pattern. The most frequent lesion is an irregular band of mottled radiolucency in the metaphysis, but neither this alone or in combination with the defects in the zone of provisional calcification is conclusive proof of a luetic infection.[5]

Lesions in the diaphyses are unusual. They appear as mottled moth-eaten areas of radiolucency associated with irregularities of the endosteal and periosteal surfaces of the cortex.

The pathognomonic evidence of luetic infection is the polyostotic metaphyseal lesions within the first 6 months of life. The involved metaphyses are irregularly outlined areas of mottled radiolucency frequently associated with fractures showing displacement or impaction. The areas of involvement tend to be symmetrically distributed and in the same stage of evolution. An erosive process in the sigmoid notch of the ulna, particularly if bilateral, is indicative of a luetic infection.

Following the institution of antibiotic therapy there is an immediate improvement in the clinical status, and in 10–14 days early but unequivocal evidence of osseous healing may be seen.[7] In the metaphyses there appears to be simultaneous absorp-

tion of detritus and deposition of new bone. Callus may be present prior to therapy but increases in extent subsequently. A localized sheath forms around the lesion, but within 4 weeks modeling both internally and externally produces approximately normal contours. Complete healing occurs thereafter.

The gross erosive process in the sigmoid notch of the ulna is unique in that it is the only articular surface involved in the entire series. Healing here keeps pace with healing elsewhere. In 2 weeks the subchondral area fills in with new bone and there is a decrease in the irregularity of the notch. In 4 weeks healing is almost complete.

Periosteal new bone is seen only in small amounts initially but increases in extent progressively for approximately 2 months, but even in extensive cases there is incorporation into the shaft of the bone virtually without a trace in 3 months.

The explanation for the deformity of the femora in 1 case is unknown. This patient who was first seen at the age of 7 weeks had the most severe and the most extensive degree of involvement in the entire series. There was symmetric fragmentation of the proximal and distal femoral metaphyses bilaterally with medial displacement of the distal femoral fragments as well as impaction deformities at the proximal metaphyses. Intrinsic healing of bone progressed normally but on examination at the age of 2 years there was prominent anterolateral bowing of the femora while the metaphyses and epiphyses appeared to be normal. No other bone showed abnormality. A careful survey was made to eliminate other systemic and hereditary disease but none were found.

SUMMARY

There is a striking uniformity and rapidity in the rate and pattern of healing in penicillin treated congenital osseous syphilis.

Within 2 weeks healing is evident and within 2 months it is almost complete.

In 1 patient, contrary to accepted concepts, there was residual deformity consisting of moderately severe anteromedial bowing of the femora 2 years after treatment. It is probable but not certain that the deformity is consequent upon the severity of bone involvement.

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FRACTURES OF THE PETROUS AND TYMPANIC PARTS OF THE TEMPORAL BONE IN CHILDREN: A TOMOGRAPHIC STUDY OF 35 CASES*

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FRACTURES of the petrous and tympanic parts of the temporal bone are uncommon in children. During the past 18 months, 3,000 children have presented at the Hospital for Sick Children in Toronto with a history of an injury to the cranial vault. A fracture of the petrous and tympanic parts of the temporal bone was identified with the aid of hypocycloidal tomography in 35 of these children—an incidence of 1.2 per cent.

In previous studies of 4,465 children with head injuries at this Center,[1] 312 or 7 per cent presented with one or more signs of the triad of cerebrospinal fluid (C.S.F.): otorrhea; lower motor neuron seventh nerve palsy; or severe bleeding from the middle ear. The incidence of each sign in the 4,465 children was: C.S.F. otorrhea, 0.6 per cent; lower motor neuron seventh nerve palsy, 1 per cent; and severe bleeding of the middle ear, 5 per cent. A fracture of the petrous part of the temporal bone (petrous bone) was demonstrated in 10 per cent of the triad group, or in 0.8 per cent of all 4,465 head injuries. The over-all incidence of the roentgenographic demonstration of fractures of the petrous bone in the previous and present studies together (7,465 children) is 1 per cent.

The accurate demonstration of the pattern of fractures of the petrous bone is important in those cases with persistent otorrhea or facial nerve palsy or both if surgical intervention is contemplated. The localization of the entrance of a fracture into one or other intracranial fossa will determine the operative approach. Demonstration of the site of involvement of the facial canal will aid the surgeon in the decompression of the facial nerve.

This report will detail the roentgenographic characteristics of fractures of the petrous bone and tympanic bone. The tympanic bone forms the anterior wall and floor and part of the posterior wall of the external auditory canal, and is attached to the rest of the temporal bone at the tympanomastoid fissure and the petrotympanic fissure.

CLINICAL MATERIAL

Fractures of the petrous bone were demonstrated by tomography in 26 boys and 9 girls, between the ages of 11 months and 14 years with an average age of 8 years. All had a history of a head injury and one or more of C.S.F. otorrhea, facial nerve palsy, severe middle ear bleeding or conductive hearing loss (Table 1). C.S.F. otorrhea (65 per cent) and a facial nerve palsy (33 per cent) were common. Middle ear bleeding was judged to be severe only if it was marked and persistent. No fracture of the petrous bone was demonstrated by tomography in an additional 2 children with

TABLE I

PRESENTING SYMPTOMS

Otorrhea—22
Seventh nerve palsy—11
Severe middle ear bleeding—9
Conductive hearing loss—6

* Presented at the Annual Meeting of the American Society of Neuroradiology, Cleveland, Ohio, April 17–18, 1969, and the Annual Meeting of the Canadian Association of Radiologists, Montreal, March 17–20, 1970.

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C.S.F. otorrhea and in 4 others presenting with seventh nerve palsy. The signs resolved spontaneously in these last 6 children.

A history or clinical evidence of trauma to the jaw was specifically excluded in all children in this series.

TECHNIQUE AND RESULTS

Hypocycloidal tomography at intervals of 1 mm. was performed on every child. Lateral tomography was performed on all the children, anteroposterior tomography in 33 and basal tomography in 12. It was found necessary to perform these sections at intervals of 1 mm. in the lateral, but at 2 mm. in the anteroposterior and basal projections as the complete identification of fractures in the lateral projection was more difficult. The entire petrous bone from the external auditory canal to the foramen lacerum areas was visualized in all cases.

Conventional roentgenograms demonstrated associated fractures of the vault in 29 of the 35 children (83 per cent), whereas a fracture of the petrous bone was seen in these roentgenograms in only 6 children (17 per cent).

An associated tympanic bone fracture was demonstrated in 23 children (66 per cent) and the ossicles were disrupted in 3. Fractures passed into the middle fossa of the skull alone in 21 cases, into the posterior fossa alone in 9 and into both in 5.

SPECIFIC PATTERNS OF PETROUS FRACTURES

The precise patterns of petrous fractures have been studied at operation, post mortem, or by stresscoat skull experiments by Hough and Stuart,[2] McHugh,[4] McGovern[3] and Proctor *et al.*[5] There has been surprisingly little accurate roentgenographic confirmation of these patterns in spite of the advent of the polytome.

The classification of petrous fractures as determined by the above studies, as longitudinal fractures, transverse fractures and combined fractures was critically evaluated in our group of 35 children. This evaluation was derived from the appearance of the fractures on serial tomographic cuts and, in part, from visual confirmation when surgical intervention was undertaken.

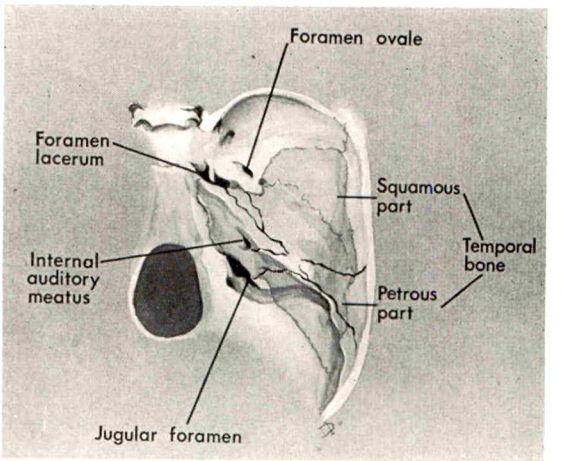


FIG. 1. Diagrammatic representation of the superficial pattern of the longitudinal petrous bone fractures.

1. LONGITUDINAL FRACTURES

Lateral tomography is the procedure of choice for demonstrating longitudinal fractures, as these are not visible in the anteroposterior projection. They made up 86 per cent of petrous bone fractures and passed from the temporal squama or from the posterior parietal bone into the petrous bone along its longitudinal axis. Two types, of longitudinal fractures occurred: one type, associated with a fracture of the anterior parietotemporal region, extended along the *anterior* aspect of the petrous bone through the tegmen tympani to the region of the labyrinth capsule (Fig. 1); the other, associated with a fracture of the posterior parietal bone, involved the superior mastoid complex and the *posterior* and superior portion of the petrous bone. This type converged also on the labyrinth complex.

Roentgenograms showed the vestibule never involved and the cochlea seldom involved, although the longitudinal fractures did cross the semicircular canals.

From this confluence at the labyrinth, the fractures proceeded either along the

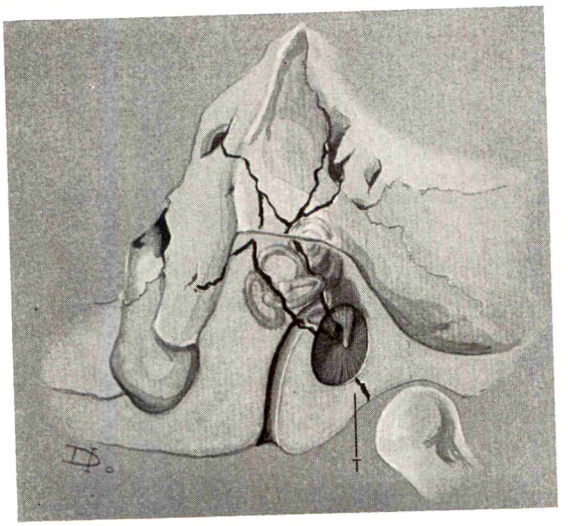


Fig. 2. A cross section diagram of the usual course of longitudinal petrous bone fractures. Note fractures of the tympanic bone (T).

roof of the eustachian tube or to one of the nearby foramina lacera, jugular foramina or internal auditory meati, with no particular preference. Extension into the latter foramina involved the posterior fossa. In 2 children, a posterior parietal fracture line appeared to enter the posterior fossa directly at the sinus plate of the petrous bone.

When the relation of the fracture line to the facial canal could be demonstrated, anterior longitudinal fractures involved the horizontal portion of the canal at or just posterior to the anterior genu, and posterior fractures involved the vertical portion of the canal or the posterior genu (Fig. 2).

The frequently associated fracture of the tympanic bone (T in Fig. 2) indicated the site of involvement of the facial canal by the extrapolation of the force vector line presumed to have produced the fractures of the petrous and tympanic bones. Both the anterior and posterior longitudinal fractures appeared to pass from the petrous bone across the middle ear cavity (Fig. 2). Similar studies confirmed these findings in adults.[6]

Illustrative examples of these groups of fractures are:

(a) The *anterior longitudinal fractures* involved the tegmen tympani with and without an extension across the external canal and middle ear into the tympanic bone (Fig. 3; and 4). Note the anteriorly placed head of the mandible in the roentgenograms enabling proper orientation. A fracture of the tympanic bone was present without a visible fracture of the petrous bone (Fig. 5). This child had a seventh nerve palsy. Diastasis of the petrotympanic fissure in children may be confused with a fracture (Fig. 6). Another child had an anterior longitudinal fracture extending down into the floor of the middle ear, entering the jugular fossa (Fig. 7*A*); tomographic sections showed the progression of this fracture into the roof and floor of the internal auditory canal (Fig. 7*B*). Additional

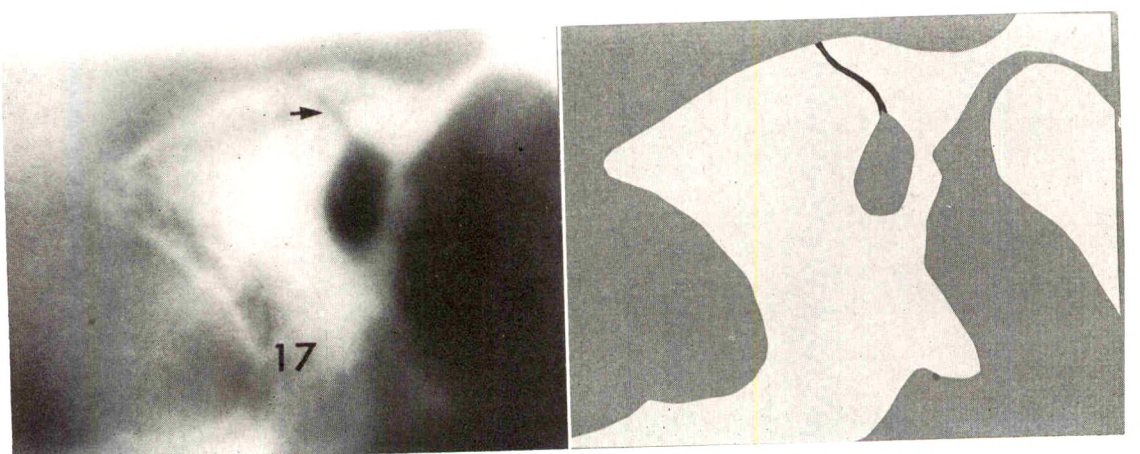


Fig. 3. Anterior longitudinal fracture only, through roof of petrous bone.

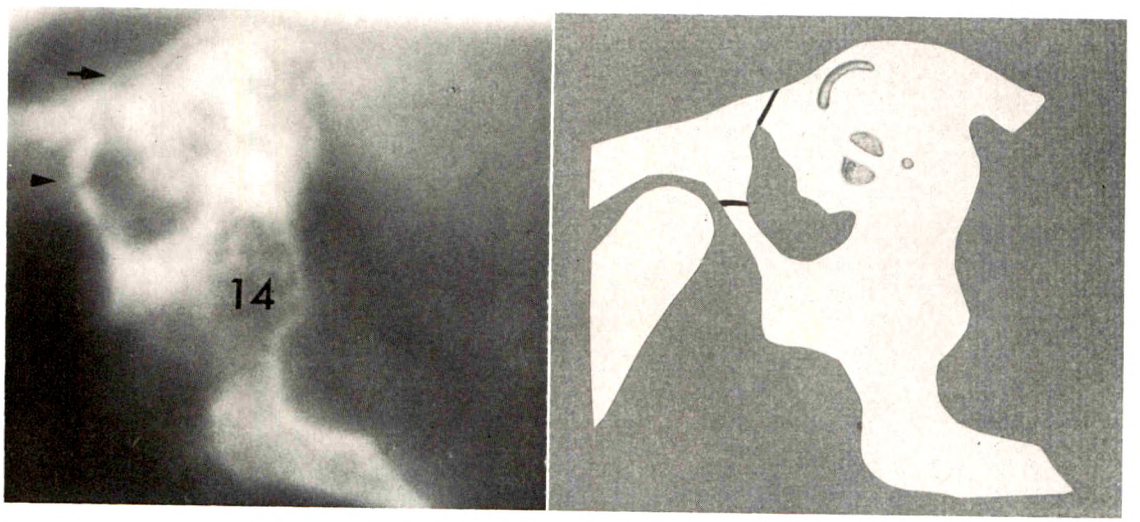


Fig. 4. Anterior fracture (arrow) with an associated fracture of tympanic bone (arrowhead).

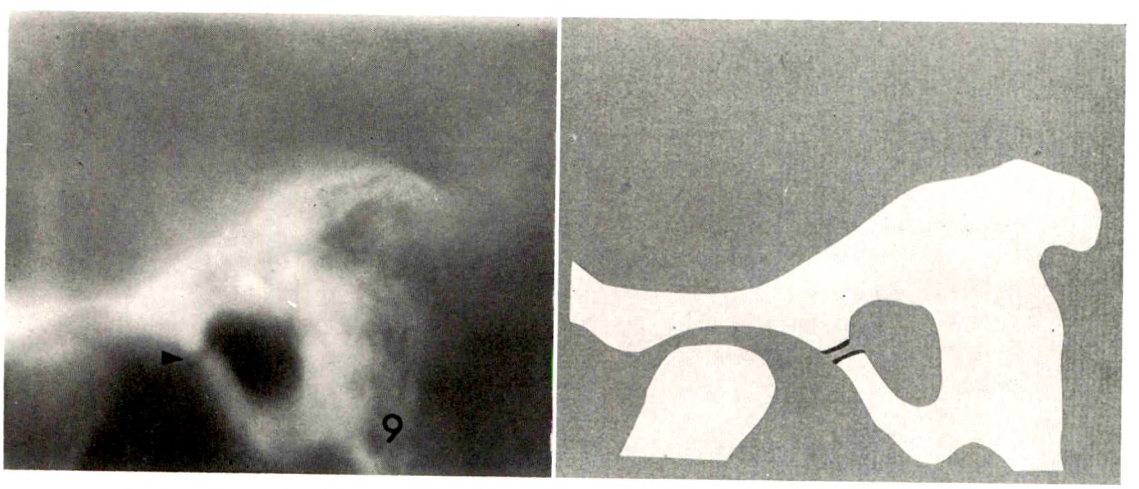


Fig. 5. A fracture of the tympanic bone only.

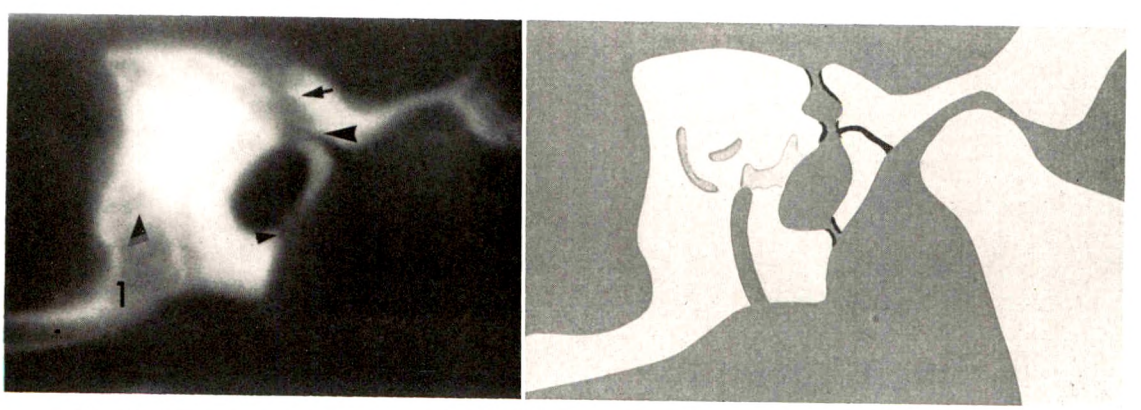


Fig. 6. An anterior fracture (arrow) with a fracture of the tympanic bone (small arrowhead) and an associated diastasis of the petrotympanic suture (large arrowhead).

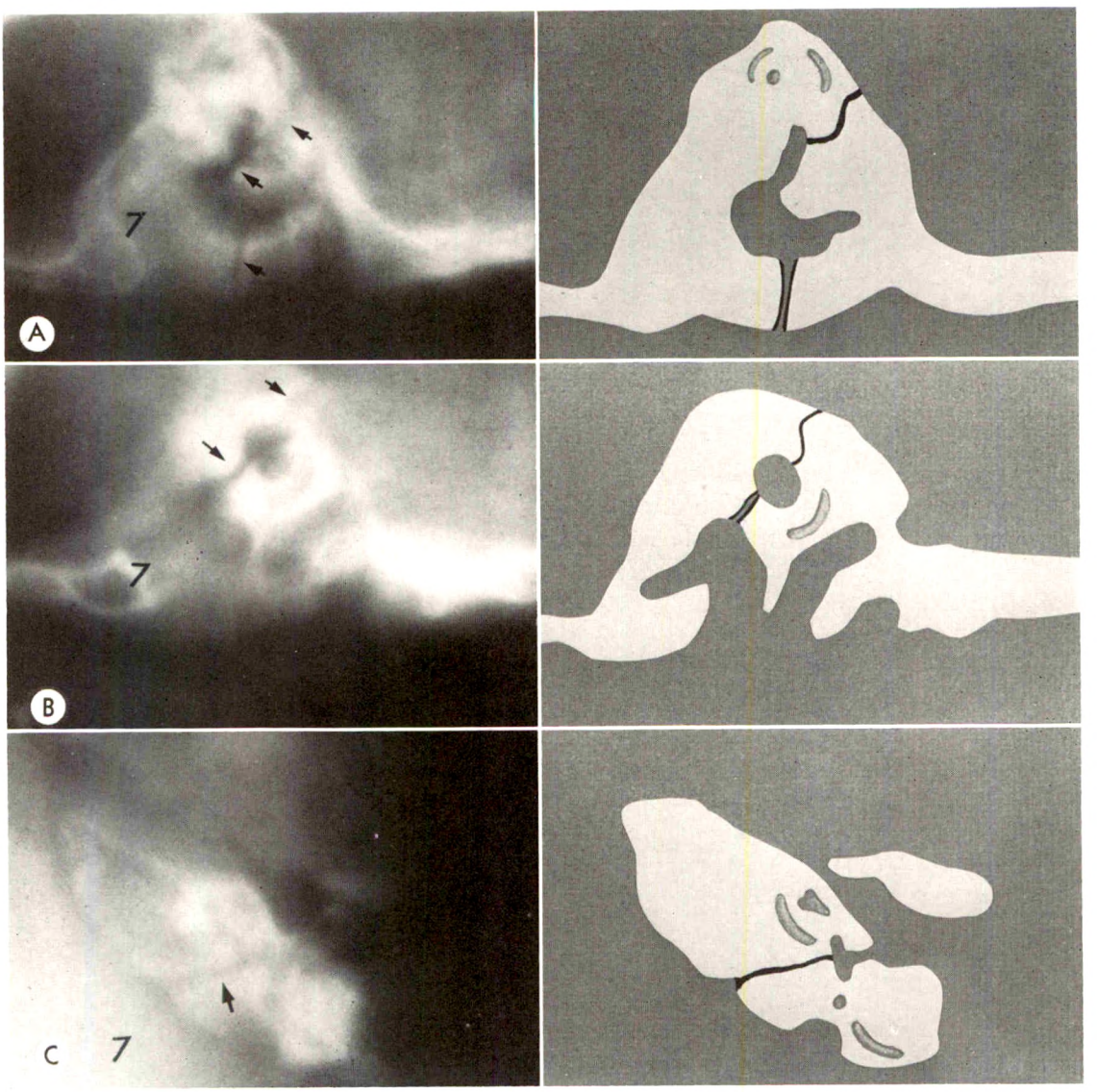


FIG. 7. (*A*) A fracture extending across the middle ear cavity to its floor and into the jugular fossa. (*B*) Involvement of the roof and floor of the internal auditory canal by the extension of this longitudinal fracture. (*C*) A basal tomogram demonstrating fracture involving the entire length of internal auditory canal.

axial tomography demonstrated this fracture in the entire length of the roof of the internal auditory canal (Fig. 7*C*). Similarly, tomographic sections taken in another child show an anterior fracture and a tympanic fracture (Fig. 8*A*) and more medially both an anterior and posterior fracture (Fig. 8*B*). Both the anterior and posterior limbs of the fracture passed across the external auditory canal and middle ear to the tympanic fracture. Finally, the fracture involved the bony septum between the anteriorly placed cochlea (c) and the more posterior internal auditory canal (i) (Fig. 8*C*).

(b) A *posterior longitudinal fracture* involved both the posterior superior portion of the petrous bone and the sinus plate inferiorly and posteriorly (Fig. 9, *A* and *B*). Again note the confluence of both limbs of the fracture across the middle ear and external auditory meatus at the tympanic fracture.

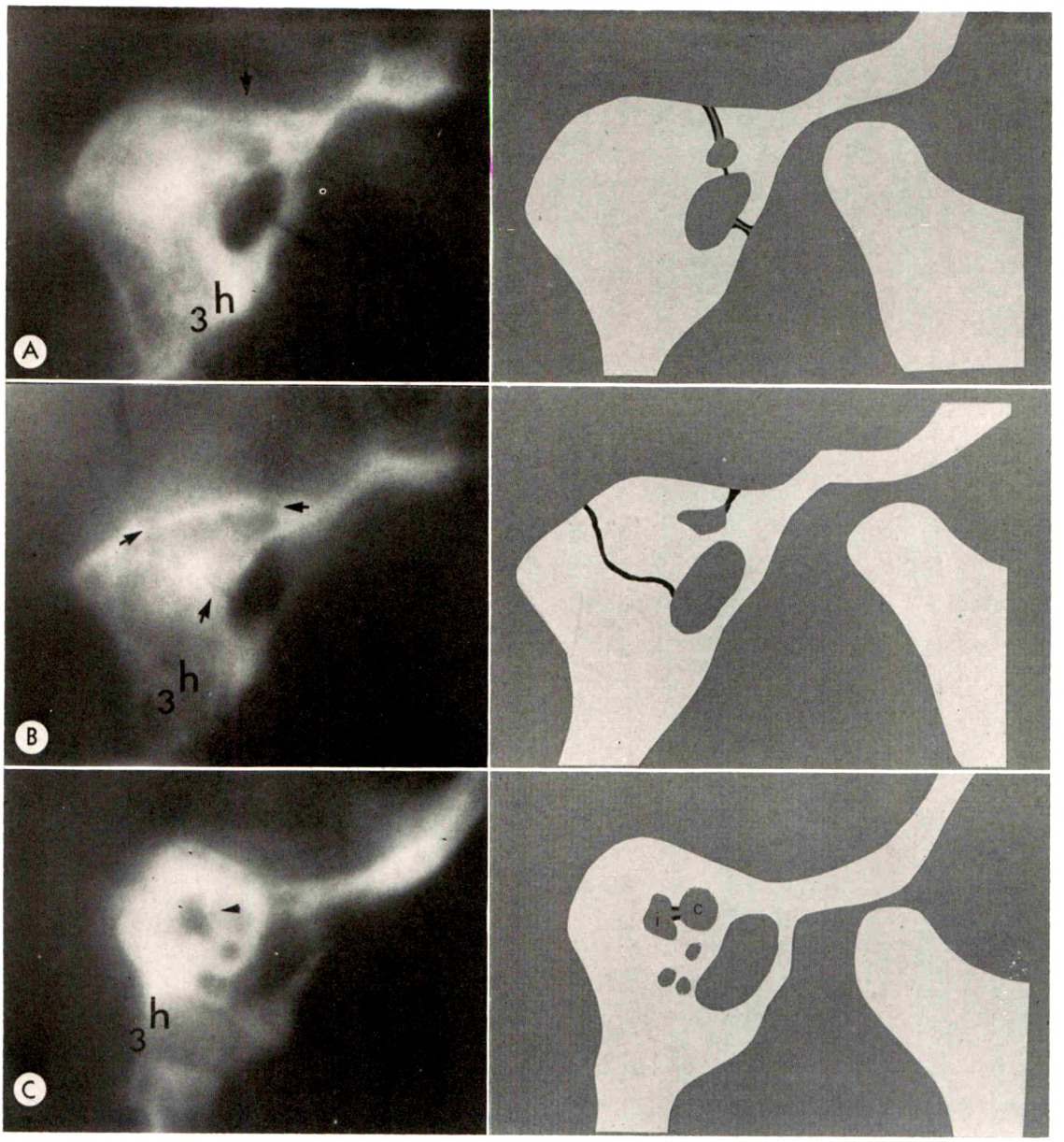


Fig. 8. (*A*) An anterior fracture and a tympanic fracture present laterally. (*B*) A posterior fracture seen more medially. Note that its direction appears to converge with the anterior fracture at the tympanic fracture in *A*. (*C*) This fracture also involved the septum between the cochlea (c) and the internal auditory meatus (i).

2. TRANSVERSE FRACTURES

Transverse fractures alone were uncommon (8.5 per cent) and involved either the roof and floor of the middle ear cavity (Fig. 10), or the labyrinthine complex entering the vestibule (Fig. 11). These fractures could be demonstrated in the anteroposterior but not in the lateral projection. Transverse fractures were associated with occipital fractures. One child presented with a C.S.F. otorrhea, 1 with a seventh nerve palsy and 1 with both. None had a tympanic fracture.

3. COMBINED OR COMMINUTED FRACTURES

Two children had a longitudinal and a

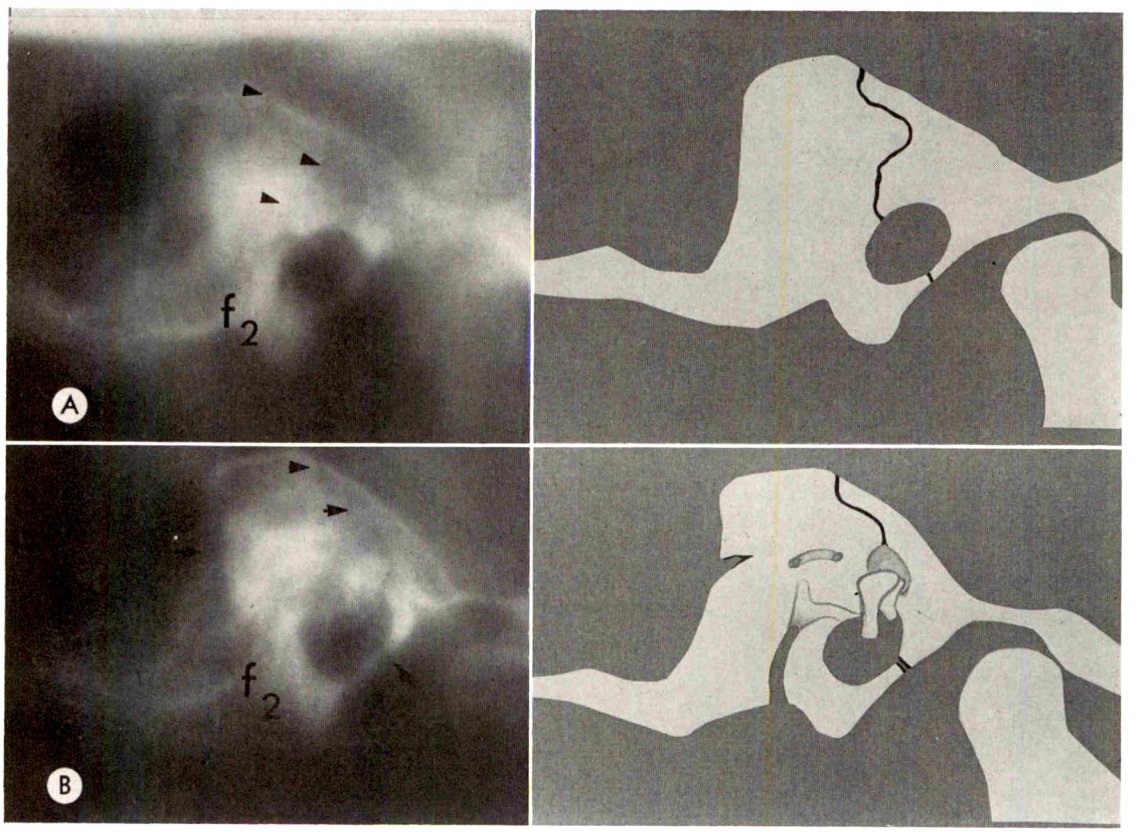


FIG. 9. (*A*) A posterior longitudinal fracture and a tympanic fracture. (*B*) An additional fracture involving the sinus plate. Note their direction toward the tympanic fracture.

transverse fracture producing a shattering effect with fragmentation of the petrous bone (Fig. 12). Both had C.S.F. otorrhea, a facial nerve palsy and a posterior parietal fracture.

4. OSSICULAR DISRUPTION

The malleus, incus, *or both* may be disrupted and displaced by a fracture of the petrous bone. In 1 child both were disrupted (Fig. 13) and in 2 others the position of the incus only was disturbed.

OPERATIVE CONFIRMATION

Thirteen children underwent an operation, 4 for a decompression of the seventh

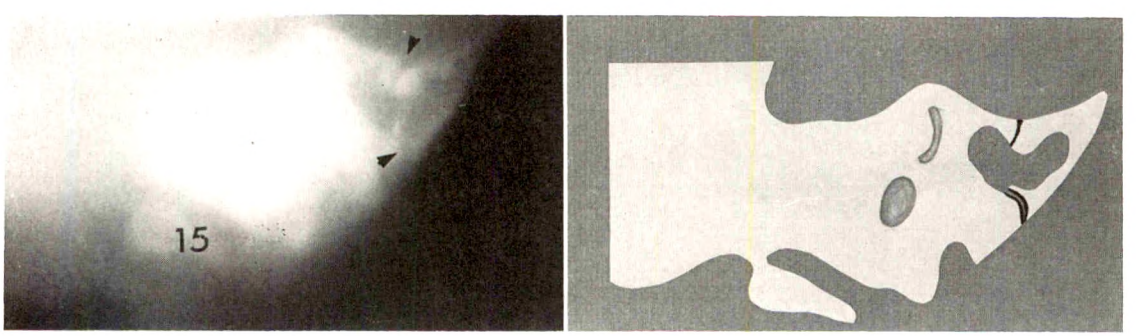


FIG. 10. A transverse fracture involving the tegmen tympani and floor of the middle ear in an 11 month old child.

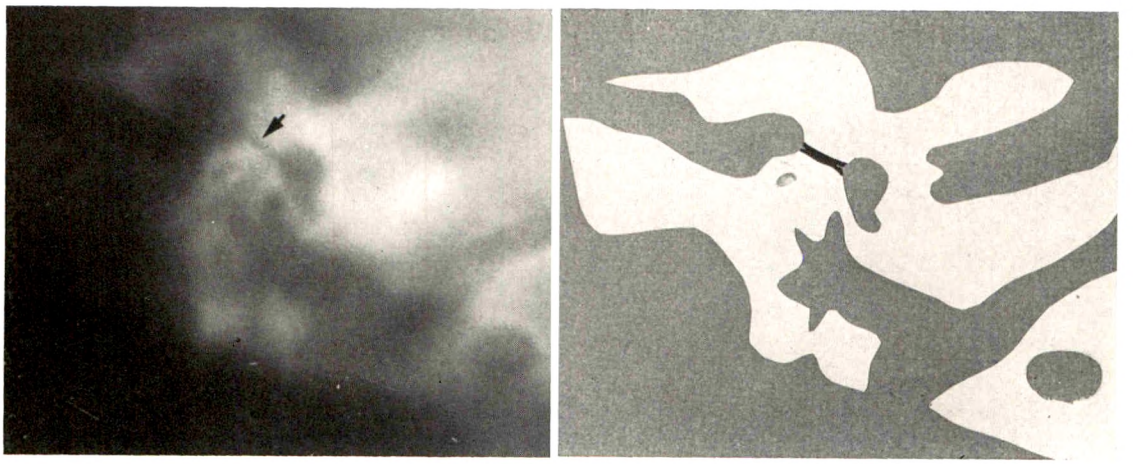


Fig. 11. A transverse fracture entering the vestibule from the antrum.

nerve canal and 9 for repair of a persistent C.S.F. otorrhea. The line of the fracture as demonstrated by tomography was confirmed in every case. In those children in whom the exact site of involvement of the seventh nerve canal was not seen on the tomogram, the suggested site obtained by extrapolation of the force vector from the fracture of the petrous bone to that of the tympanic bone was confirmed.

DISCUSSION

In this study, the tomographic characteristics of petrous bone fractures in children are similar to those found at operation by Hough and Stuart,[2] and those produced by stresscoat studies of the cadaver skull as performed by Proctor, Gurdjian and Webster.[5] The fractures are longitudinal, transverse or a combination of the two but the following additional roentgenographic features occurring in children were discovered:

i. Longitudinal fractures involved either the anterior or posterior portions of the roof of the petrous bone and, in a minority of cases, both in the same child. These fractures converged in the region of the bony labyrinthine capsule and then extended to one or another of the neighbouring foramina (jugular, internal auditory meatus, foramen lacerum) or along the roof of the eustachian tube. Proctor *et al.* described longitudinal fractures as if they "course through the roof of the middle ear and then along the anterior edge of the pyramid" only, and from their own stresscoat studies found that pure longitudinal fractures are uncommon. We found, however, that pos-

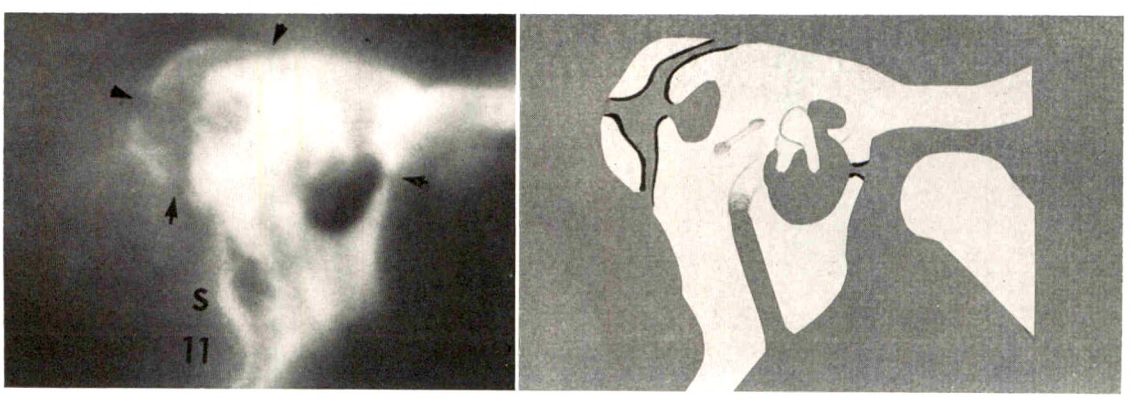


Fig. 12. A combined and comminuted fracture with a fracture of the tympanic bone.

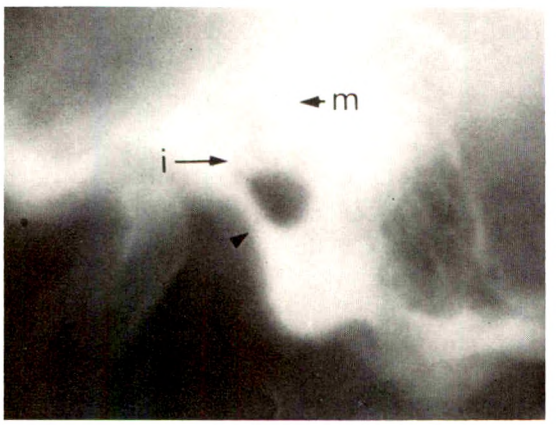


FIG. 13. A fracture of the tympanic bone and dislocation of both the incus (i) anteriorly and the malleus (m) superiorly.

teriorly placed longitudinal fractures and pure fractures were common.

ii. Extensions of a fracture line across the external auditory meatus and middle ear to involve the tympanic bone were common in longitudinal fractures (66 per cent). A tympanic fracture was not significant by itself. It is worth repeating that diastasis of the petrotympanic fissure superiorly should not be confused with a tympanic fracture (Fig. 6).

iii. Combination fractures (longitudinal and transverse), although in the minority, shattered the bone in every instance leaving large fragments of bone lying free of the posterior superior and posterior inferior portions of the petrous bone (Fig. 12) and producing a C.S.F. otorrhea.

iv. Fractures involving the cochlea were difficult to detect roentgenographically. Anterior longitudinal fractures appeared to pass between the bony capsules of the cochlea and labyrinth (Fig. 1; and 2).

Aggressive tomographic investigation of the petrous bone in children with a head injury is indicated in those with a subsequent persistent C.S.F. otorrhea, facial nerve palsy or severe middle ear bleeding. If operative repair of a persistent C.S.F. otorrhea is undertaken, the fracture can be easily identified extradurally, and a characteristic arachnoid adhesion to the dural tear can be seen intradurally. In a number of children, this adhesion was not present in the region of the confirmed fracture, and another more inaccessible site of the C.S.F. leak was presumed, *e.g.*, via the internal auditory meatus, the petrous apex or close to a large dural venous sinus. Involvement of the facial canal by the fracture is readily confirmed, particularly with the aid of an operating microscope.

The high incidence of C.S.F. otorrhea and facial nerve palsies associated with fractures of the petrous bone is distorted, as these are the very signs which prompted tomographic investigation. The incidence of 0.6 per cent and 1 per cent, respectively, in children with head injuries is more realistic. Many fractures of the petrous bone undoubtedly remain undetected by conventional skull and temporal bone roentgenograms. Even with the tomographic evidence of a fracture of the petrous bone in the children of this series, only in 17 per cent was the fracture demonstrated by these conventional roentgenograms.

SUMMARY

The fractures of the petrous bone in children are: longitudinal, transverse, or combined, relative to the axis of the bone. Hypocycloidal tomography demonstrated that, in addition to these accepted patterns, longitudinal fractures can involve the anterior and the posterior portions of the petrous bone; an associated fracture of the tympanic part of the temporal bone occurs in two-thirds of all petrous fractures; and combined fractures result in distractive fragmentation of petrous bone. An associated ossicular disruption is uncommon.

Standard roentgenographic studies of the temporal bone are inadequate to demonstrate these complex fractures. Aggressive tomographic studies of the petrous bone are indicated if one or more of a triad of presenting signs of C.S.F. otorrhea, low motor neuron seventh nerve palsy, or severe bleeding from the middle ear are present.

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CHOROID PLEXUS PAPILLOMAS*

OBSERVATIONS ON RADIOGRAPHIC DIAGNOSIS

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CHOROID plexus papillomas are rare tumors of the central nervous system occurring with a relative incidence of about 0.5 per cent of intracranial neoplasms in adults[1,6,10,28] and 3.0 to 5.0 per cent of brain tumors in children.[12,15,18] Although not common, these tumors are usually benign and are often accessible to surgical extirpation. It is therefore of paramount importance that their presence be recognized early and appropriate therapy be initiated promptly. The clinical manifestations of these neoplasms are protean with the vast majority of cases, especially in children, presenting as hydrocephalus. The ultimate diagnosis therefore invariably rests upon the neuroradiographic examination.

We have recently collected 234 cases of choroid plexus papillomas from the literature and have added 11 unpublished cases of our own. Of these 245 cases, 105 (43 per cent) occurred in one of the lateral ventricles, 26 cases (10 per cent) originated in the third ventricle, 95 cases (39 per cent) in the fourth ventricle, and 17 cases (9 per cent) were found in the cerebellopontine angle (Fig. 1 and 2). In rare instances (3.7 per cent), multiple papillomas presumably arose virtually simultaneously in more than one site (Fig. 2). The radiographic data in the 10 cases of which we have personal knowledge have been reviewed in detail and we have supplemented this survey with an analysis of the findings of the 234 cases of choroid plexus papillomas previously published in the literature.

This report is concerned with the radiographic diagnosis of choroid plexus papillomas and includes our observations on salient procedures that should be performed in these patients as well as pitfalls that must be avoided.

PLAIN RADIOGRAPHY OF THE SKULL

Since the majority of choroid plexus papillomas arising in the lateral ventricles occur within the first 10 years (Fig. 3) and are invariably associated with hydrocephalus, suture separation, craniofacial disproportion, increased digital markings and the other roentgenographic changes of raised intracranial pressure are common. These indirect findings of increased intracranial pressure provide no specific proof

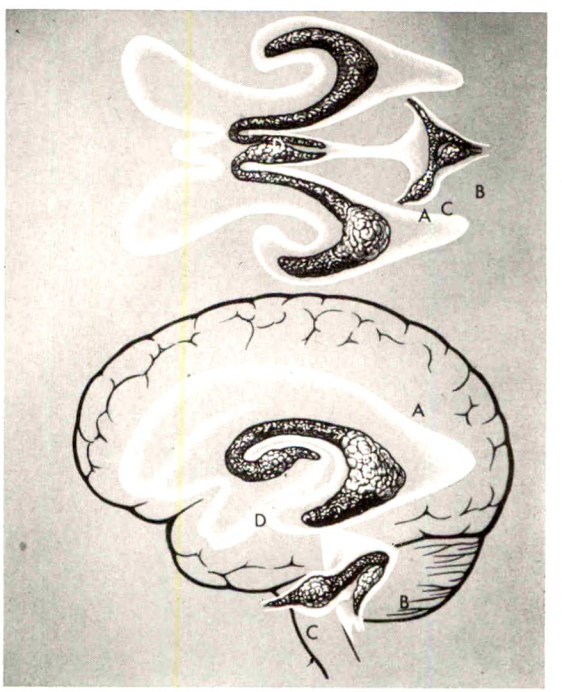
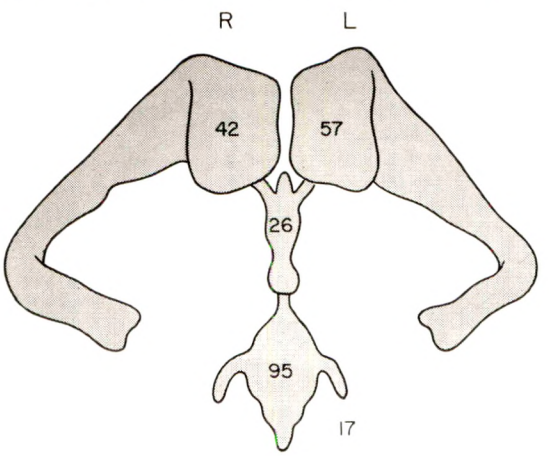


FIG. 1. Choroid plexus papillomas—characteristic locations. A = lateral ventricle; B = fourth ventricle; C = angle; and D = third ventricle.

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as to the nature or location of the underlying pathology.

In 4.1 per cent of cases in our collected series, calcification was present in sufficient quantity within a choroid plexus papilloma to be recognized radiologically. It is unusual for the normal choroid plexus to be calcified within the first decade of life,[4] and this is the age group in which 70 per cent of lateral ventricle papillomas occurred. When calcification is in the region of the choroid plexus in a child below the age of 10 years, and if unilateral, it represents an important diagnostic point. Calcification, when present in a choroid plexus papilloma, usually presents in a stippled configuration and is obviously located within the ventricular system (Fig. 4, *A–C*). Since many of the papillomas, especially within the lateral ventricle are pedunculated, the calcification may shift position within the ventricle relative to changing positions of the head. When demonstrable, this is an important diagnostic finding. Of course other tumors of childhood also occasionally display calcification (*e.g.*, craniopharyngioma, ependymoma, pinealoma) and these would obviously have to be considered in the differential diagnosis.



MULTIPLE PAPILLOMAS

BOTH LATERAL VENT.	6
RIGHT LAT. V + IV V	1
LAT. V + III V	1
III V + IV V	1

Fig. 2. Location of choroid plexus papillomas (245 cases).

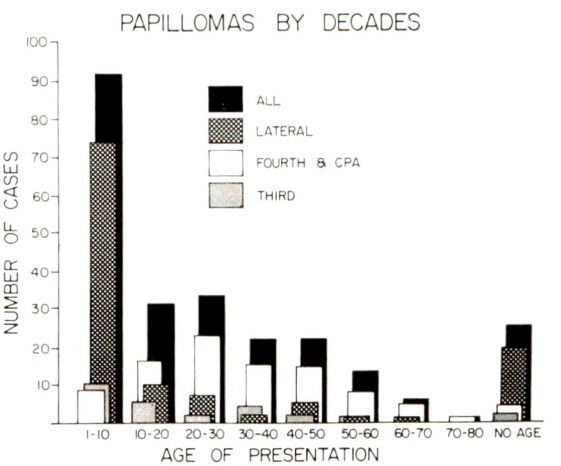


Fig. 3. Location of choroid plexus papillomas (245 cases) in various decades of life.

In adults calcification is not uncommonly seen within the normal choroid plexus.[9] It is extremely uncommon, however, for a lateral ventricle papilloma to occur after the third decade of life (Fig. 3), and the diagnostic probability of a choroid plexus papilloma within the lateral ventricle in a patient with calcification in this region after the age of 30 years would be less likely. Papillomas in other locations, however, occur not uncommonly in the adult age group (Fig. 3), and the finding of calcification in the third and fourth ventricles as well as in the cerebellopontine angle should raise one's index of suspicion of the presence of this neoplasm, especially if the calcification is accompanied by signs of hydrocephalus and increased intracranial pressure.

PNEUMOGRAPHY

Pneumography is the most valuable single radiologic procedure in the evaluation of these tumors. Pneumography was responsible for the definitive diagnosis in 10 of the 11 patients in our series. In the other patient, the pneumogram outlined the tumor and this was confirmed by angiographic studies as well.

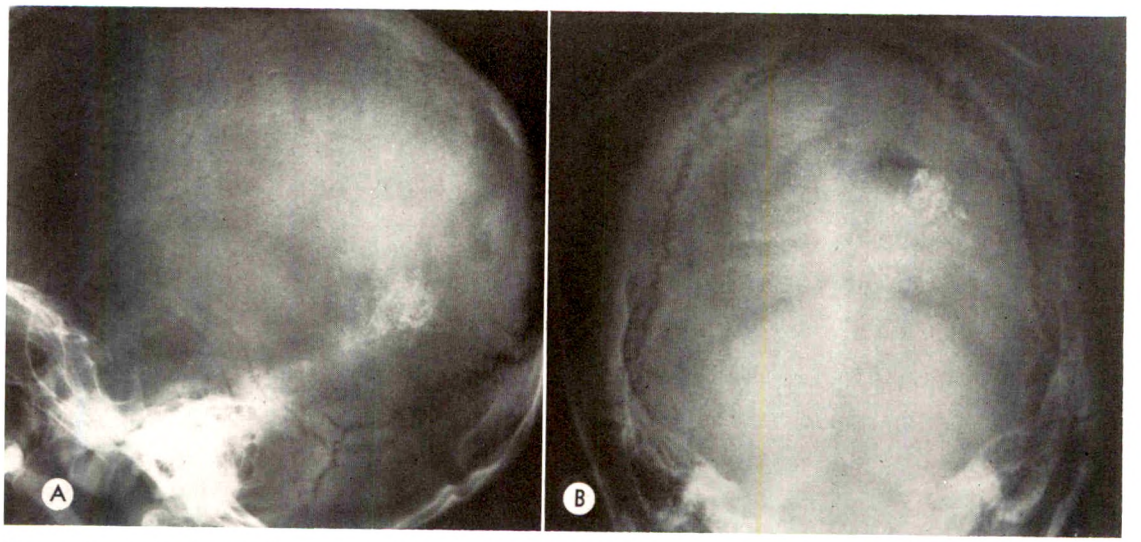


FIG. 4. Patient C.S. (*A*) Lateral skull roentgenogram showing "speckled" calcification in region of trigone of the left lateral ventricle. (*B*) Posteroanterior half axial view showing dense, irregular calcification in region of trigone of the left lateral ventricle. There is slight separation of the coronal sutures.

Whether the air for the pneumographic study should be introduced via the lumbar route or injected directly into the cerebral ventricle must be determined in each individual case. In infants with split sutures and open fontanelles direct ventricular puncture is invariably the safer technique and usually provides the more rewarding radiographic study. Occasionally it may be necessary to introduce air via both lumbar and ventricular routes. Whatever technique is selected, it is of paramount importance that air be introduced in whatever amounts necessary to outline the entire ventricular system. The so-called "bubble" study should be avoided since choroid plexus papillomas have a specific gravity higher than that of cerebrospinal fluid and hence may lie submerged within the cerebrospinal fluid filled ventricle and thus escape detection.

Certain characteristics of the pneumographic examination should alert the radiologist to the possibility of a choroid plexus papilloma. Since it is thought that these tumors are responsible for the production of excess cerebrospinal fluid,[13,20] the lateral ventricle harboring the tumor is usually larger than its opposite member (Fig. 4*C*). Moreover the segment of the ventricle containing the tumor usually is the most dilated portion of the ventricular system. Often the tumor may insinuate itself into the surrounding ependyma and thereby create an interface between tumor and brain. Excess secretion of cerebrospinal fluid by the tumor thereby encased may produce the radiographic appearance of a porencephalic cyst capping a tumor, a portion of which also projects into the ventricular system.[8]

In patients with choroid plexus papillomas the ventricular system caudad to the tumor usually partakes in the ventricular dilatation so that cases having only partial obstruction of the lateral or third ventricles by an intraventricular mass will often show a disproportionate degree of enlargement of the more caudally situated fourth ventricle and aqueduct (Fig. 4*D*). It has been suggested that excess secretion of cerebrospinal fluid by the papilloma is responsible for this ventricular dilatation below the site of the tumor,[7,13,20] but other factors may also be incriminated.[2,14,21,24,26]

Papillomas of the choroid plexus are of-

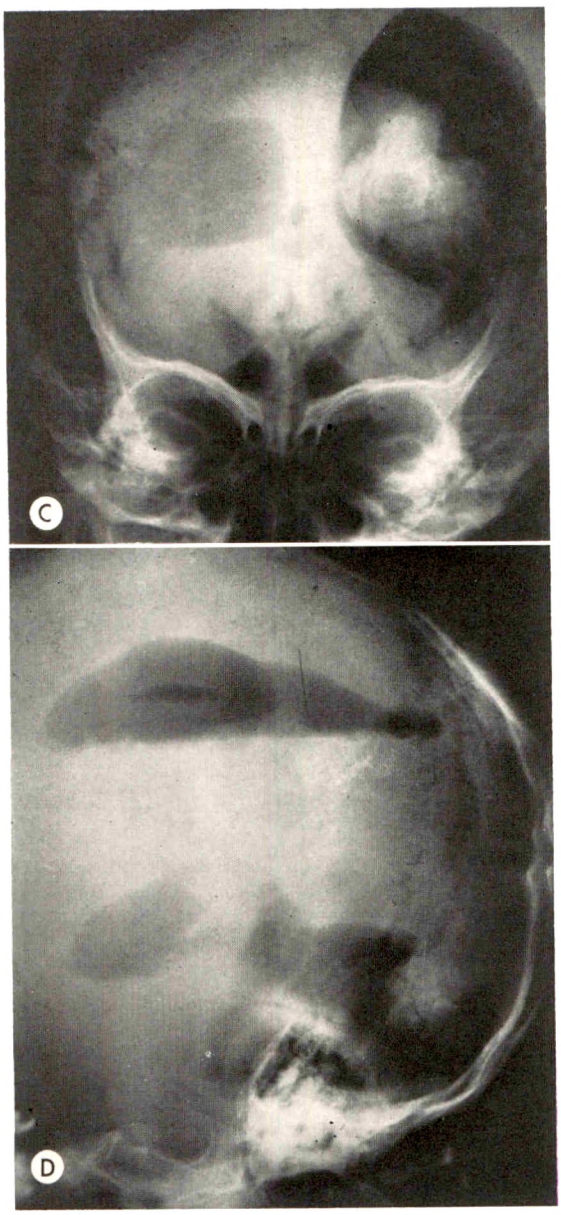


Fig. 4. (*C*) Posteroanterior brow-down pneumoencephalogram. The entire ventricular system is dilated, especially the trigone and temporal horn of the left lateral ventricle containing the partially calcified choroid plexus papilloma. (*D*) Erect lateral pneumoencephalogram. There is a large papilloma of the left lateral ventricle which is "hidden" at this juncture, within the fluid-filled ventricle. Note the dilated third ventricle, aqueduct, fourth ventricle and cisterna magna. Without additional air in the left lateral ventricle, and without calcification, this patient might have been diagnosed as "congenital communicating hydrocephalus."

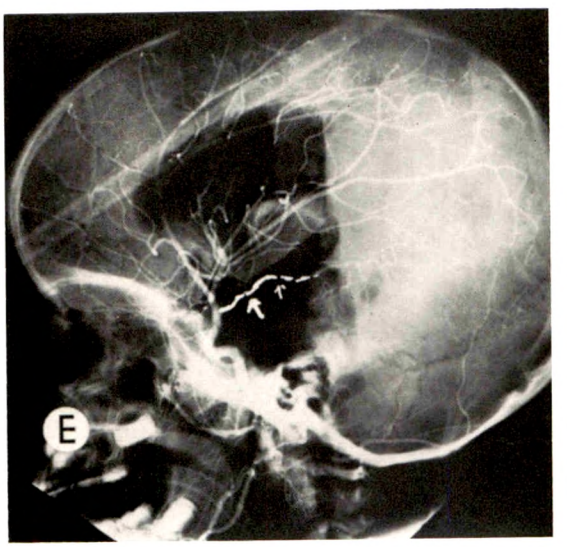


Fig. 4. (*E*) Left lateral carotid arteriogram—arterial phase. Residual air remains in the ventricles from the previous pneumogram. There is severe ventricular dilatation, as evidenced by "bowing" of anterior and middle cerebral arteries, and stretching of the carotid siphon. Note especially the hypertrophied anterior choroidal artery (white arrows) which supplies a tumor mass, outlined by a vascular "stain" in the region of the trigone of the left lateral ventricle (anterior choroidal artery retouched).

ten lobulated and air introduced during pneumography may enter the clefts between tumor lobules producing a speckled appearance in the roentgenogram (Fig. 5).



Fig. 5. Patient J.F. Brow-down lateral ventriculogram. There is a large lobulated mass which almost entirely fills the dilated fourth ventricle. The air-filled "clefts" between the tumor nodules produce a speckled appearance.

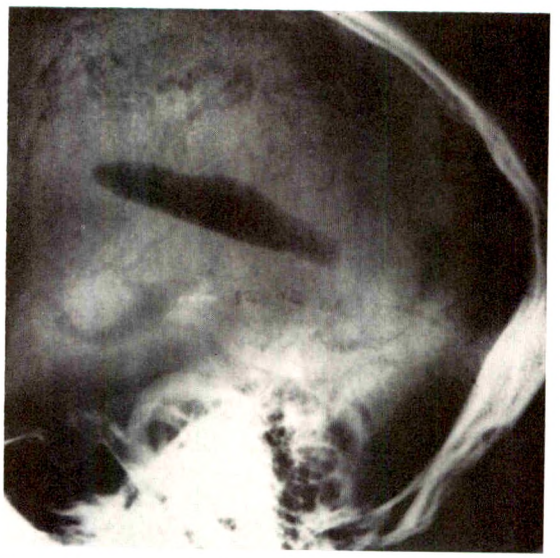


FIG. 6. Patient T.K. Lateral pneumoencephalogram. There is a smooth mass within the third ventricle just posterior to the foramina of Munro producing partial obstruction of the lateral and anterior third ventricles. Choroid plexus papilloma.

This finding is not, however, absolutely characteristic of papillomas which occasionally display a smooth surface on pneumography (Fig. 6).

Papillomas are usually pedunculated arising from a vascular stalk attached to the normal choroid plexus. The stalk when demonstrable, may be found in fairly predictable positions. Papillomas arising from the lateral ventricle are usually attached at their inferior aspect in the region of the trigone. Third ventricle papillomas are attached superiorly to the roof of the ventricle (Fig. 6) and those originating in the fourth ventricle have their stalk located posteriorly and inferiorly in continuity with the roof of the fourth ventricle near the obex (Fig. 7, *A* and *B*). This point of attachment, when demonstrable, will often serve as an important distinguishing characteristic between a choroid plexus papilloma and other neoplasms which are also found within the ventricular system (*e.g.*, ependymoma). Often choroid plexus papillomas develop secondary adhesions to the surrounding ventricular wall and their actual primary point of attachment may not be obvious.

The lateral ventricle papillomas are usually quite large when first recognized. This may not necessarily be characteristic of a papilloma *per se*, but may be indicative of any tumor growing within the ventricle (*e.g.*, ependymoma, meningioma). What, however, is characteristic of a choroid

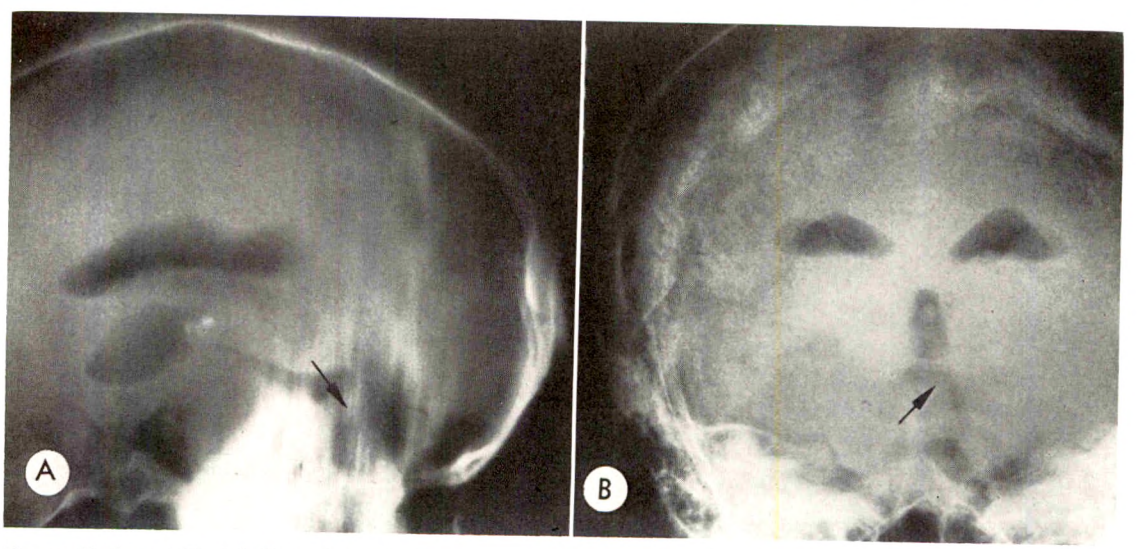


FIG. 7. Patient A.P. (*A*) Erect lateral pneumoencephalogram. There is a lobulated mass attached to the roof of the fourth ventricle (black arrow), producing partial obstruction of cerebrospinal fluid. (*B*) Erect posteroanterior pneumoencephalogram. Note the lobulated mass which partially fills and partially displaces the fourth ventricle to the left (black arrow). There is dilatation of the third ventricle and aqueduct.

plexus papilloma, but unfortunately is not often recognized radiographically, is the movement of the tumor within the ventricle about its pedunculated stalk. It should be emphasized that the entire ventricular system must be outlined before one can definitely exclude the presence of a tumor arising from the walls of the ventricles. Usually the diagnosis will immediately become obvious, if sufficient quantities of air can be introduced and manipulated into all portions of the ventricular system. On occasion, however, particularly in patients with papillomas arising in the third ventricle and posterior fossa, the use of positive contrast material may be valuable.

The introduction of air directly into the lateral ventricles in any patient with hydrocephalus may result in the production of bleeding and the subsequent formation of a hematoma from the normal choroid plexus. If factitial hemorrhage occurs, subsequent roentgenograms will demonstrate an intraventricular mass. The appearance of blood tinged cerebrospinal fluid which gradually clears, the presence of noncrenated red blood cells and the tendency for an iatrogenic hematoma to be somewhat smaller than a choroid plexus papilloma might aid in the differential diagnosis between these 2 entities.

The major potential pitfall in the radiographic diagnosis of choroid plexus papillomas occurs because of the association of this tumor with communicating hydrocephalus. In numerous cases in the literature, inadequate "bubble studies" had been performed and benign curable lesions overlooked.[3,5,15] In a few fortunate instances, deterioration of the patient's condition following the initial air study led to repeated radiographic examination and the subsequent establishment of the correct diagnosis. In others, efforts to correct the "congenital communicating hydrocephalus" usually failed and the diagnosis of a choroid plexus papilloma was only made at postmortem examination.

The inadequacy of "bubble" ventriculography is particularly germane to the papillomas found within the lateral ventricle. These tumors have a specific gravity greater than that of the surrounding ventricular fluid. The tumor accordingly will tend to gravitate to a dependent position while the small bubble of air rises to the topmost portion of the dilated ventricle. The demonstration of a communicating hydrocephalus, particularly in young children and infants, should serve as an indication for the introduction of additional air in whatever quantity necessary to obtain thorough delineation of the entire ventricular system. The danger of restricting complete ventriculographic studies to only those cases of communicating hydrocephalus exhibiting certain other abnormalities will almost certainly result in overlooking some cases of papillomas. An especial indication for performing thorough ventriculographic studies is the presence of congenital communicating hydrocephalus in any patient having any of the following features: disproportionate enlargement of one lateral ventricle; bloody or xanthochromic cerebrospinal fluid; cerebrospinal fluid pleocytosis or increased cerebrospinal fluid protein; and deterioration of the patient's neurologic condition following "bubble" examination.

CEREBRAL ANGIOGRAPHY

Until recently cerebral angiography was not usually performed in young patients who presented with an enlarging head and displayed no specific lateralizing neurologic signs or had not had clinical or laboratory evidence of intracranial bleeding. In a few cases where angiography had been performed in patients who have had choroid plexus papillomas, apart from the angiographic signs of enlargement of the ventricular system, no specific changes were demonstrated.[3,11,22] This is surprising since the angiographic architecture of these tumors would suggest that they would demonstrate a "vascular stain" on angiography. This, in fact, has not been well documented.

On occasion, however, cerebral angiog-

raphy has been extremely rewarding and the specific diagnosis has been established by means of this contrast examination.[23] We have had a case of this type. The patient was a 3 year old child with calcification in the region of the trigone. Previous pneumography revealed ventricular dilatation and a mass in the trigone and temporal horn of the left lateral ventricle. Left carotid angiography revealed evidence of ventricular dilatation, but more specifically there was marked hypertrophy of the anterior choroidal artery and a vascular stain outlining the tumor mass (Fig. 4*E*). The specific diagnosis of a choroid plexus papilloma was made and this was proven at surgery.

Vertebral angiography in patients with choroid plexus papillomas of the posterior fossa has not been frequently performed and little reliable data are available.[16]

GAMMA ENCEPHALOGRAPHY

There are few published data regarding gamma encephalography in patients with choroid plexus papillomas. One reason for the paucity of this information may be that scanning has not been widely used in the evaluation of children with hydrocephalus. Because of the increased vascularity of these tumors, one would expect papillomas to display a fair degree of uptake of the isotopes used in brain scans, particularly with technetium 99m which is known to be concentrated by the normal choroid plexus.[17,27]

The potential value of gamma encephalography in patients with choroid plexus papillomas may be illustrated by reference to one of our patients (Fig. 8, *A–D*). This child was originally subjected to surgery at the age of 7 years, when a "tumor of the right cerebral hemisphere" was partially resected. Surgery was again performed 2 years later and additional tumor was resected from the right lateral ventricle. On this second occasion, histologic examination confirmed that this was indeed a choroid plexus papilloma. Recurrent symptoms of vomiting and headache prompted a third operation in March, 1967, and an experienced neurosurgeon was confident that the tumor had finally been entirely resected. In May, 1968, the patient was again readmitted to the hospital with signs of increased intracranial pressure, and a radioactive scanning using technetium 99m revealed an area of dense uptake in the posterior parietal region (Fig. 8, *A–D*). It is highly unlikely that the positive scan in this case was the result of the earlier surgical procedures since the last tumor resection had been performed 14 months previously.

With recent improvements in the isotope compounds used in brain scanning, as well as the availability of more sophisticated scanning equipment, gamma encephalography should be used more frequently, not only in the initial evaluation of patients with an unexplained hydrocephalus, but in the follow-up regimen to patients who have had total or subtotal resections of choroid plexus papillomas.

DISCUSSION

Although choroid plexus papillomas are relatively uncommon, hydrocephalus in children is almost ubiquitous on a neurologic or neurosurgical service. In a busy clinic especially, one tends to follow a set schema in the investigation of children with hydrocephalus. Such a "routine," while undoubtedly valuable in the majority of instances, may, if not modified, militate against the establishment of the correct diagnosis in patients with unusual lesions. This is apt to occur in patients having choroid plexus papillomas and if so would be doubly unfortunate since a benign potentially remediable lesion may be overlooked. The diagnosis of a choroid plexus papilloma will rarely be made unless the condition is thought of and the appropriate diagnostic procedures planned and executed in a predetermined fashion. There is no place for short cuts such as "bubble" pneumography in the investigation of these patients. Although the cerebrospinal fluid may be, and frequently is, grossly and

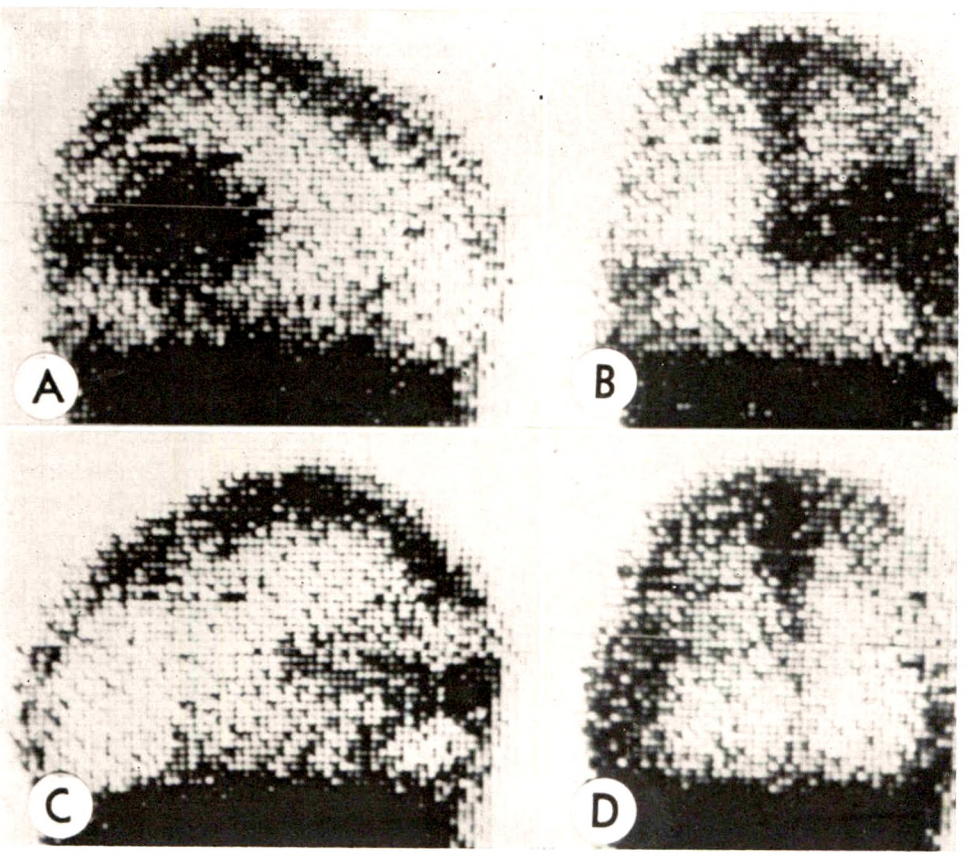


Fig. 8. Patient S.L.J. (*A–D*) Gamma encephalograms using technetium 99m. Note area of activity in posterior parietal region on right side. (*A*) Right lateral; (*B*) posteroanterior; (*C*) left lateral; and (*D*) anteroposterior.

microscopically normal in patients with choroid plexus papillomas, the presence of xanthochromic or blood-stained cerebrospinal fluid, pleocytosis, or increased cerebrospinal fluid protein, should alert one to the possibility that an intraventricular tumor may be present. Especially when these cerebrospinal fluid changes are present, meticulous care must be exercised to ensure demonstration of the entire ventricular system so that the presence or absence of a tumor may be determined unequivocally.

Cerebral angiography may be useful not only in demonstrating the presence of large ventricles, but in delineating a specific arterial supply to a tumor. Recently, gamma encephalography has revealed uptake in these tumors and it will be important to see whether additional confirmatory observations are reported. Positive contrast studies may occasionally help when air cannot be manipulated into portions of the ventricular system or cerebellopontine angles. In one instance a patient with a choroid plexus papilloma had the correct diagnosis established preoperatively on the basis of cytologic examination of the cerebrospinal fluid which had been removed during pneumography. Although cerebrospinal fluid cytology in the past has not proven to be generally valuable in the diagnosis of most intracranial neoplasms, additional refinements may allow this technique to be employed to better advantage in the future.

It should be emphasized that choroid plexus papillomas may recur if not entirely

excised. We have delineated such an example previously in this report. It may, therefore, be wise to follow these patients after surgery with serial brain scans even if they are symptom-free, and, of course, repeat contrast studies should be performed when symptoms warrant. In this latter regard, it is recognized that "seeding" of benign papillomas within the cerebrospinal fluid axis occurs and the secondary implants are often discovered at sites frequently far removed from the original tumor.[19,25,28] This situation is found with other tumors having direct access to the ventricular system (*e.g.*, medulloblastoma) and follow-up examinations of patients with choroid plexus papillomas may require the utilization of studies such as myelography under appropriate circumstances.

SUMMARY

A review is made of 234 cases of choroid plexus papillomas collected from the literature, to which 11 unpublished personal cases are added.

The clinical features, plain roentgenographic findings of the skull, and the value of pneumography, cerebral angiography and gamma encephalography in establishing the diagnosis are discussed in detail.

The majority of choroid plexus papillomas occurred within the first decade of life and were invariably associated with hydrocephalus.

Suture separation, craniofacial disproportion, increased digital markings and other roentgenographic changes of increased intracranial pressure provide no specific proof as to the nature of location or the underlying pathology.

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NORMAL VALUES OF THE VERTEBRAL BODY AND INTERVERTEBRAL DISK INDEX DURING GROWTH*

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THE morphologic aspects of the vertebral body (vb) in the various osseous dysplasias have been widely described and well summarized.[12] In view of the different mechanisms of the vertebral growth—by enchondral ossification in the vertical and by periosteal in the horizontal direction[12,14]—there exist conditions of more or less selective involvement of these 2 ossification patterns. For a better understanding of such modifications, a morphologic description alone is too subjective, and is valuable only in distinct entities; *i.e.*, in achondroplasia, in thanatophoric dwarfism, in severe forms of congenital osteogenesis imperfecta and in others. Measurements of vertical and sagittal vertebral body diameters might enhance recognition of various pathologic features of the vertebra and permit comparative studies of enchondral and periosteal growth.

Rabinowitz and Moseley[11] measured, without statistical analysis, the vertical and sagittal vb diameter of a small number of newborns. Hurxthal[5] gives results of vertical vb diameter of a small population of adult women. Transverse and sagittal measurements were made by Knutsson[8] for the first lumbar vertebra. His results cannot be incorporated in this study because his data were given without statistical evaluation. Measurements of intervertebral disks for adults were available from the paper of Hurxthal[5] and from the work of Schinz.[13] No comparison between men and women was made. No determinations were available for children.

MATERIAL AND METHOD

The material consists of 187 roentgenograms of dorsal and lumbar spines from newborns up to adolescents. Careful attention was paid to exclude children with virtual vb diseases, especially neurologic cases with known muscular hypotonia, endocrine disorders, or degenerative syndromes with or without chromosomal anomalies. Traumatic lesions of the spine were included in this investigation if they were outside of the studied region. The principal diagnoses are tabulated in Table 1. Only good lateral projections of vb were measured. Roentgenograms were always made with a Bucky table, even in newborns. The film-focus distance was gener-

TABLE I

DIAGNOSES OR INDICATIONS FOR ROENTGENOGRAPHY

	No.
Exclusion of traumatic or infectious lesions	58
Functional scoliosis without anatomic lesions	42
Exclusion of vertebral malformations	23
Normal spine in healthy children	2
Neurologic diseases without muscular hypotonia	15
Psychologic disorders	6
Tumors outside of the spine	9
Sacral teratoma or lipoma	2
Myelomeningocele with slight neurologic symptoms	2
Lung diseases	6
Kidney diseases without metabolic disorders	4
Immunologic disorders	5
Pyloric stenosis	1
Intoxications	2
Osteochondrosis	4
Storage diseases without skeletal signs	3
Spondylolysis L5/S1	3
Total	187

* From the Institut de radiologie médicale de l'hôpital cantonal universitaire de Lausanne (Directeur: Prof. Dr. G. Candardjis), Lausanne, Switzerland.

ally 110 cm.; however, since the roentgenograms were taken in different divisions of the Radiological Institute, slight variations might be possible. The factor of enlargement was calculated and found to be 1.13 for newborns and 1.25 for pubertal children.

MEASUREMENTS

The measurements consisted of determination of the highest vertical diameter and the smallest anteroposterior distance of all the vb from the 12th thoracic to the 3rd lumbar vertebra (Fig. 1). For technical reasons not all these vb were always measurable. In newborns with persistence of anterior or posterior vb blood vessels, a determination was only possible when at least a faint ossification of these borders was visible. Furthermore, minimal intervertebral body distance was measured from D 11/12 to L 2/3. The measurements were made with a fine mm. scale with a precision of ± 0.25 mm.

The following indices were formed (Fig. 1):

$$I_{vb} = \frac{v}{s} = \frac{\text{vertical vb diameter}}{\text{sagittal vb diameter}}$$

$$I_{d} = \frac{d}{v} = \frac{\text{intervertebral disk thickness}}{\text{vertical vb diameter}}.$$

These indices are independent of focus-film distance. Only disks of the same embryonal segment as the vb, *i.e.*, upper disk and lower vb, were compared in the index.

Measurements were also made on posteroanterior roentgenograms, but the singular vb was rarely seen in a strict frontal projection; therefore, the height of vb could very often not be determined. Horizontal diameter was also rarely measurable, especially in infants, because of a lack of clear visibility or superimposition with the pedicles on the lateral borders.

In 92 cases the height of the children at the time of roentgenography was available. The skeletal age could be determined in only 25 cases. No generalized conclusions may be drawn from such a small number of observations.

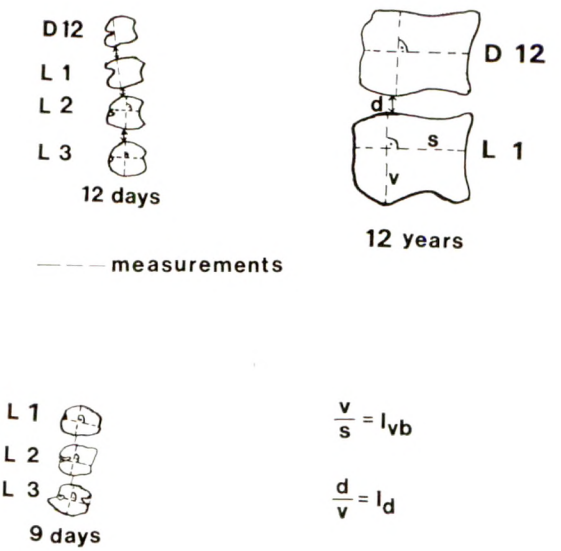


Fig. 1. Method of measuring vertebral bodies and disk spaces. Only dotted lines were measurable. This is the method used to form the indices.

STATISTICS

From each group mean values and standard deviations were calculated after proving that the measurements were normally distributed. Student tests were made between most groups.

The error of measurements was evaluated by repeated measurement on 10 roentgenograms some weeks after the first investigation. In this way 87 double measurements were made, preferentially of the newborns where the percentage of error is larger. The differences between the duplicates ranged from zero to ± 1.5 mm. The mean absolute error was ± 0.28 mm. In 46 per cent the second measurement was equal to the first one, in 89.5 per cent the maximum error was less than ± 0.5 mm., in 97.5 per cent less than ± 1 mm. From these double measurements 42 double indices v/s were calculated, in order to estimate the influence of the error on the indices. The mean difference between the duplicates was 0.032. In 19 per cent no difference was observed, in 59.5 per cent it was less than 0.03, in 85.5 per cent it was less than 0.05 and in no case was the difference more than 0.10.

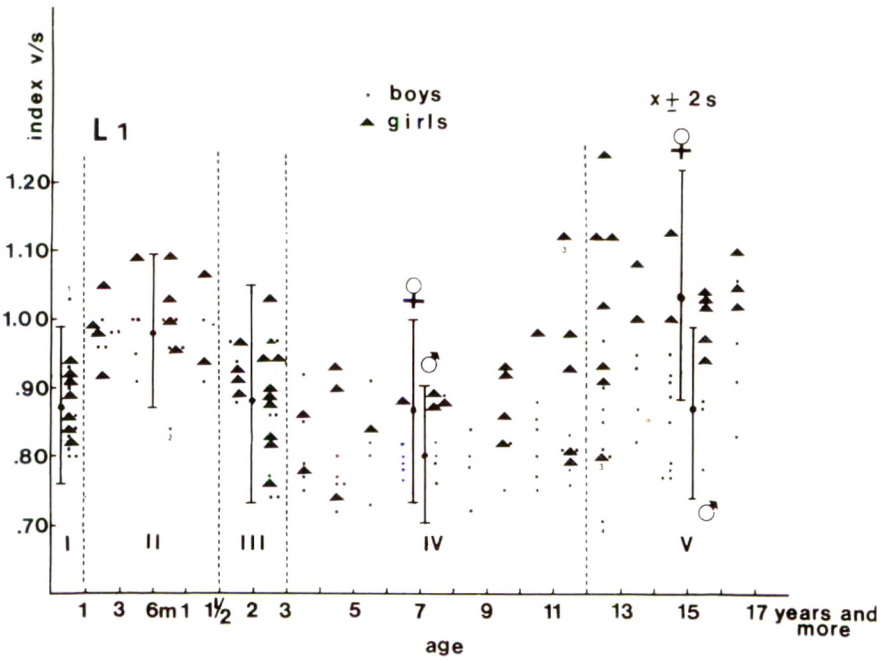


FIG. 2. Scattergram of the indices (I_{vb} = vertical/sagittal diameter) of the first lumbar vertebral body and double standard deviations in the different age groups.
Numbered cases see text.

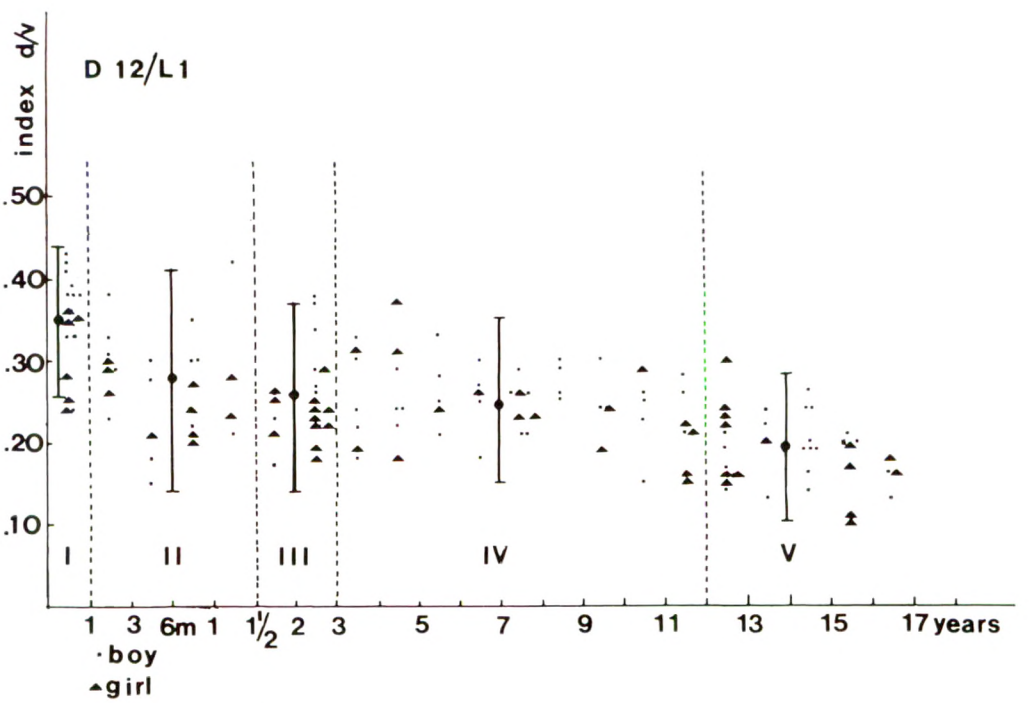


FIG. 3. Scattergram of the intervertebral disk indices (*d*isk thickness/*v*ertical vertebral body diameter) between D12 and L1 and double standard deviations in the different age groups.

Table II

AGE GROUPS

Age Group	I	II	III	IV	V
Age	0–1 month	2–18 months	19–36 months	4–12 years	13 years and more

RESULTS

According to statistical analysis by Student tests, the results were grouped by age as seen in Table II. The results of I_{vb} are summarized in Table III and those of I_d in Table IV. Diagrammatic presentation of the results is given in Figure 2.

The *Index of vertebral body* (I_{vb}) increases during infancy. In infants of Group II a slight difference between sexes is statis-

Table III

$I_{vb}(v/s)$ OF DIFFERENT VERTEBRAL BODIES

(f = girls; m = boys)

Vertebral Body	Age Group	n	Mean v/s	s	Range $x \pm 2s$	p
D12	I	13	0.81	0.061	0.69–0.93	
	II	26	0.91	0.077	0.75–1.06	<0.005
	III	22	0.86	0.066	0.73–0.99	≅0.10
	IV f	18	0.86	0.062	0.74–0.98	
	IV m	35	0.78	0.052	0.67–0.88	<0.001
	V f	7	0.93	0.148	0.64–1.23	
	V m	20	0.84	0.116	0.60–1.07	<0.05
L1	I	16	0.87	0.060	0.76–0.99	<0.001
	II f	11	1.02	0.066	0.88–1.15	<0.02
	II m	16	0.96	0.043	0.87–1.05	
	II m+f	27	0.98	0.055	0.87–1.09	<0.001
	III	23	0.89	0.080	0.73–1.05	
	IV f	20	0.87	0.068	0.73–1.00	<0.001 (IV f/m)
	IV m	40	0.80	0.048	0.70–0.90	<0.001 (IV f/Vf)
	V f	19	1.03	0.095	0.88–1.22	<0.001 (IV m/Vm)
	V m	27	0.87	0.063	0.74–0.99	<0.001 (V f/m)
L2	I	10	0.92	0.060	0.80–1.04	<0.01
	II	21	1.01	0.090	0.83–1.19	<0.001
	III	20	0.91	0.060	0.79–1.03	<0.001
	IV	49	0.82	0.076	0.67–0.97	<0.001
	V f	15	1.03	0.096	0.84–1.22	<0.001
	V m	25	0.88	0.086	0.70–1.05	
L3	I	11	0.95	0.068	0.81–1.08	<0.25
	II	17	0.98	0.084	0.81–1.15	<0.005
	III	16	0.88	0.081	0.72–1.04	<0.001
	IV	35	0.79	0.072	0.67–0.91	<0.001
	V f	11	1.00	0.101	0.80–1.20	<0.001
	V m	17	0.87	0.094	0.68–1.03	

n = number; v/s = vertical/sagittal.

TABLE IV

$I_d(d/v)$ OF DIFFERENT VERTEBRAL SEGMENTS

	Age Group	n	Mean d/v	s	Range $x \pm 2s$	p
D 11/12	I	12	0.37	0.060	0.25–0.49	
	II	26	0.30	0.065	0.16–0.43	<0.005
	III	19	0.25	0.089	0.08–0.43	
	IV	49	0.24	0.053	0.13–0.34	
	V	21	0.18	0.042	0.10–0.26	≪0.001
D 12/L1	I	17	0.35	0.063	0.22–0.48	
	II	27	0.28	0.068	0.14–0.41	<0.01
	III	20	0.26	0.057	0.14–0.37	
	IV	53	0.25	0.050	0.15–0.35	
	V	37	0.19	0.043	0.10–0.28	≪0.001
L 1/2	I	15	0.35	0.046	0.26–0.44	
	II	26	0.26	0.073	0.12–0.41	<0.001
	III	19	0.27	0.055	0.15–0.38	>0.25
	IV	44	0.28	0.047	0.18–0.37	
	V	37	0.20	0.056	0.09–0.31	≪0.001
L 2/3	I	9	0.38	0.075	0.23–0.53	
	II	18	0.28	0.089	0.10–0.46	~0.01
	III	15	0.30	0.083	0.13–0.47	
	IV	32	0.30	0.049	0.20–0.40	
	V	22	0.21	0.051	0.11–0.31	≪0.001

n = number; d/v = disk thickness/vertical; s = sagittal.

tically significant only in L 1 ($p < 0.02$), whereas in the other vb no significant difference exists. In L 2 and L 3 no sexual difference could be detected in Group IV. On the other hand, there exists a highly significant difference in D 12 and L 1. A significant sexual difference was always observed in Group V, girls having higher indices than boys. In the latter age group the range of distribution is much higher than in the other groups, in part because the samples are relatively small. No conclusions concerning adult I_{vb} are possible. Only 7 cases over 17 years of age could be measured. When comparing the development of vb at different levels, it becomes evident that children of Groups I and II have higher indices in the distal vb. Between D 12 and L 2 the difference is at $p < 0.005$ significant for Group I, and $p < 0.001$ for Group II.

The *Index of intervertebral disk* (I_d) diminishes significantly after birth (Fig. 3). In the subsequent groups no statistically proved differences were observed, although the scattergram (Fig. 3) suggests an inverse development in comparison to that of the vb (Fig. 2). After the twelfth year, I_d diminishes once more in all investigated age groups. Only slight differences exist between the I_d at different levels of the spine in the same age group: p is <0.001 only between D 11/12 and L 2/3 for Group IV; among the other groups p is insignificant. The absolute values of disk thickness of D 12/L1 were found between 2.5–4 mm. in Group I; 3–5 mm. in Group II; 3–6 mm. in Group III; 4–7 mm. in Group IV; and 3–8 mm. in Group V.

Although few measurements of the height of children were available, the indices of vb of 83 cases (47 boys and 36 girls) are examined as a function of the height. Figure 4 suggests an even better

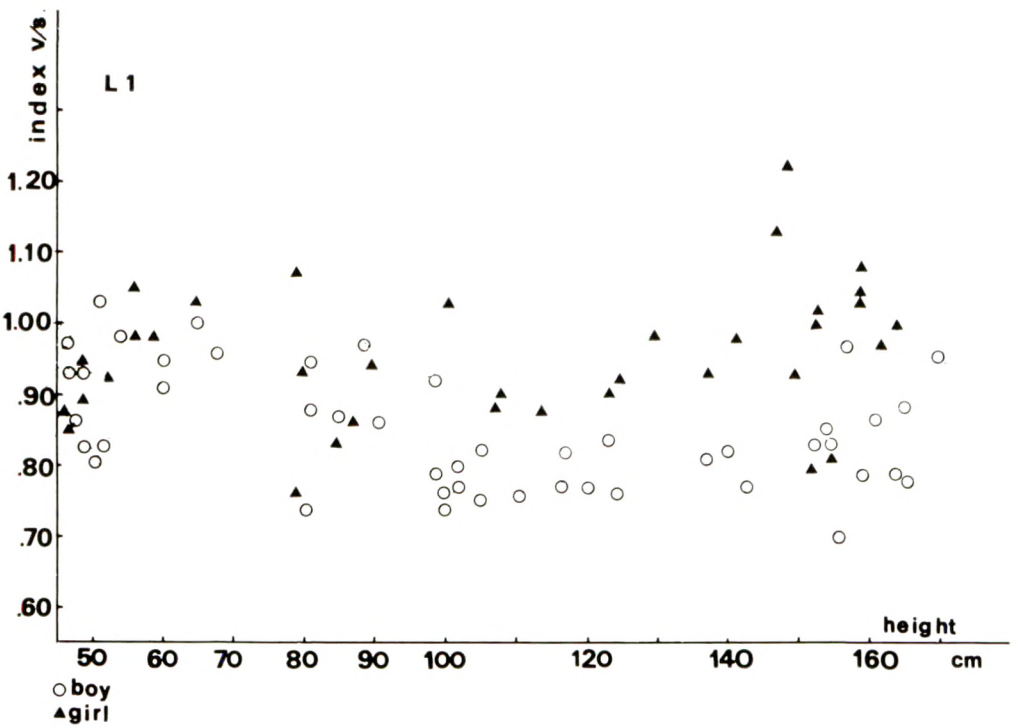


Fig. 4. Scattergram of the indices of the 1st lumbar vertebral body (I_{vb}) in function of the height of children (83 children).

relationship between the indices and the height as was found between the indices and the age (Fig. 2). With increasing height (more than 90 cm.) the difference between the sexes is more pronounced, girls having higher indices than boys.

Discussion

The inclusion of 2 roentgenograms of one case with myelomeningocele who had only distal neurologic damage might be controversial; however, they were included because of the absence of lesions in vb and especially of muscular hypotonia, which is best known to be associated with higher vb.[3] The inclusion of 3 cases of spondylolysis was regarded as permissible, since Toendury[14] failed to show an involvement of the ossification center of the vb. The 4 cases of osteochondrosis show only small lesions outside of the investigated region.

The scattergram of I_{vb} (Fig. 2) shows some values other than the statistical variation. These cases are numbered. Index of Case 1 was found in a child with hyperbilirubinemia due to incompatibility between B and O blood groups. Case 11 was hospitalized for hiatal hernia. No explanation for these abnormal indices could be found. Both cases had normal indices of the other vb. Cases 111 may correspond to a transitional group between the age Group 1v and v.

The present results require an explanation of the variations of the indices during growth and between the sexes.

In Figure 5 the absolute mean values and standard deviations of the measured vertical (v) and sagittal (s) diameter of the vb L 1 are plotted in function of the age. Girls and boys were not separated. It can be seen that in the first month of life v increases more rapidly in relation to s and then is approximately equal from 3 to 18 months. However, more detailed work will be necessary to show whether age Group 1 should be further subdivided according to a group of prematures, of dysmatures and

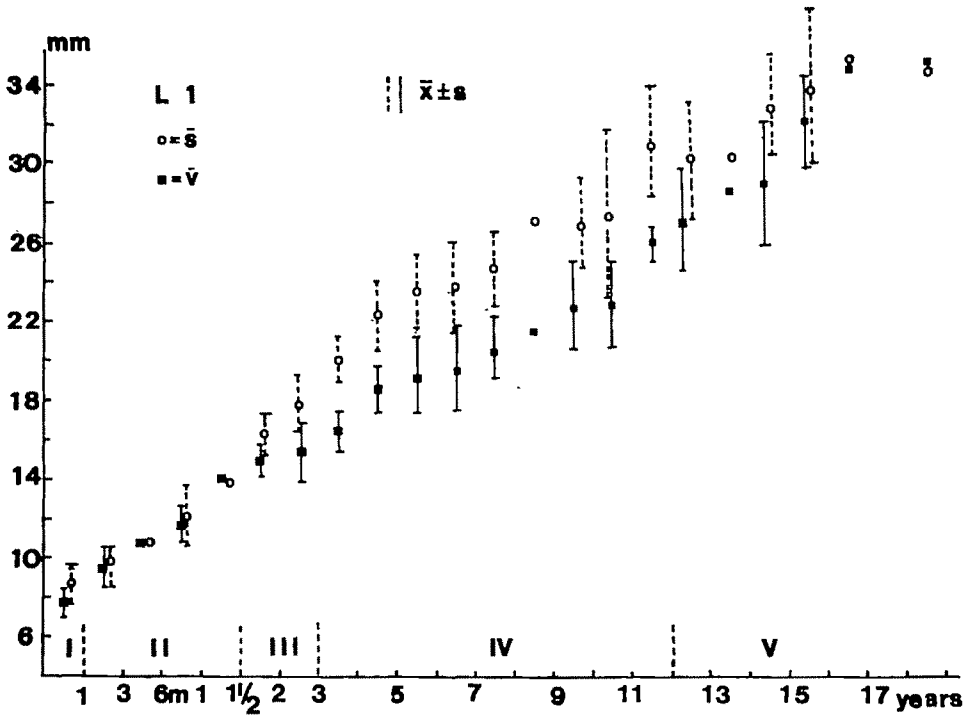


Fig. 5. Measured values of the vertical (v) and sagittal diameter (s) of the 1st lumbar vertebral body. By reason of too small samples, the standard deviation could not be calculated in some groups.

of normal fullterm babies. In children of Group III, v increases more slowly, whereas s increases faster than before. Thus the 2 curves diverge. It is improbable that this divergency may be explained solely by a slow-down of the growth of v or by an accelerated growth of s in boys only. In fact, both boys and girls begin to have lower indices and greater s in Groups III and IV. Group III is apparently a transitional group.

Until the age of 12 years the curves are parallel in Figure 5. It can be seen that the division of the results into the 5 age groups is well reflected by the behavior of these 2 curves. During the growth spurt after the twelfth year, the development of v is accelerated, whereas the increase of s diminishes and the curves rejoin. As the measurements were not corrected for the enlargement factor, the values in higher age groups are magnified by about 10 per cent.

The *sexual difference* of I_{vb} is better documented by its correlation to the height than to the age of the children (Fig. 4), and it seems to be due to a relatively increased s in boys. This interpretation is still better supported if the s values are plotted in function of the height of the children (Fig. 6), whereas for v no visible sex difference until a height of about 140 cm. exists (Fig. 7). Among the 4–12 year old children, the body height is equal or very slightly higher and the skeletal age is increased in girls,[8] who have lower s values. Thus one possible hypothesis is that the growth of s is enhanced by androgen factors.

Among children of more than 140 cm. in height, girls have a more increased vb height than boys (Fig. 7), who show a less important vertical growth of vb. This trend can be followed until a body height of 160 cm. is reached. Therefore, it seems that the observed differences in sex among children of age Group V is due to the vertical growth increase of vb in girls which is in good correlation with the sexual dif-

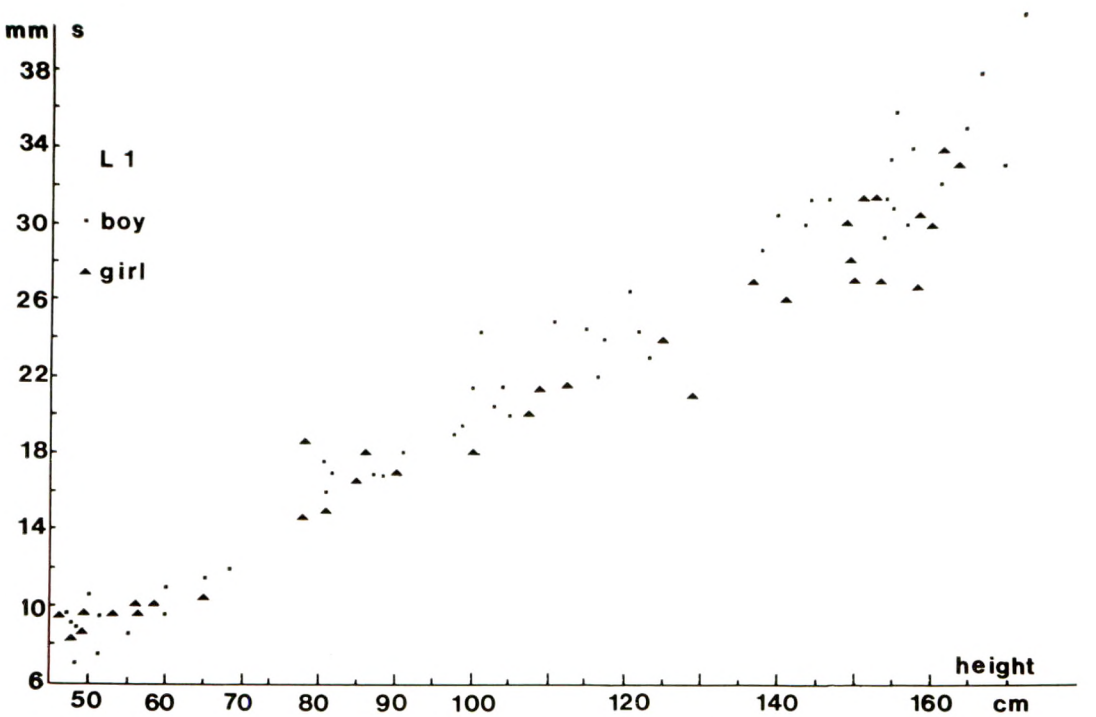


Fig. 6. Measured values of the sagittal diameter (s) of the 1st lumbar vertebral body in function of the height of the children.

ference of sitting height. At the same time boys show constantly slightly higher s values than girls. The fact that with increasing body height v and s show an inverse pattern according to sex explains well the even more significant sex difference, when an index of both is formed.

The present investigation is not quite complete in that no conclusions may be drawn with regard to the development of vb in adolescents taller than 160 cm. Growth spurt begins somewhat later in boys than girls, so that boys become taller than girls only after they reach the height of 160 cm.[4] Thus, the question of whether men will finally have equal I_{vb} as women cannot be answered.

A similar comparative study in children younger than 2 years of age was made by Joseph and Meadow,[6] who also found higher indices in girls when they correlated the sum of the length of the metacarpal bones to the sum of their width. They also demonstrated that girls have narrower and longer tubular bones than boys.

Determination and comparison of s and v of vb in adults and different sexes were not available from the literature. In this respect the papers of Hurxthal[5] and of Knutsson[8] are not conclusive. Maresh[9,10] studied the longitudinal growth of tubular bones during childhood in a large number of roentgenograms and found excellent correlations with longitudinal growth. She also found larger breadth diameters in boys, which was in good correlation with longitudinal growth. Similar findings were reported by Bonnard.[1] The two latter authors give correlations only to height and weight, or both, but do not compare breadth to length.

Although the graphic demonstration (Fig. 4–6) of the obtained results is quite suggestive in the explanation of the sex differences, statistical analysis should be made in identical groups of boys and girls at different ages and heights. For such an approach the present material was too small.

As previously mentioned, Rabinowitz

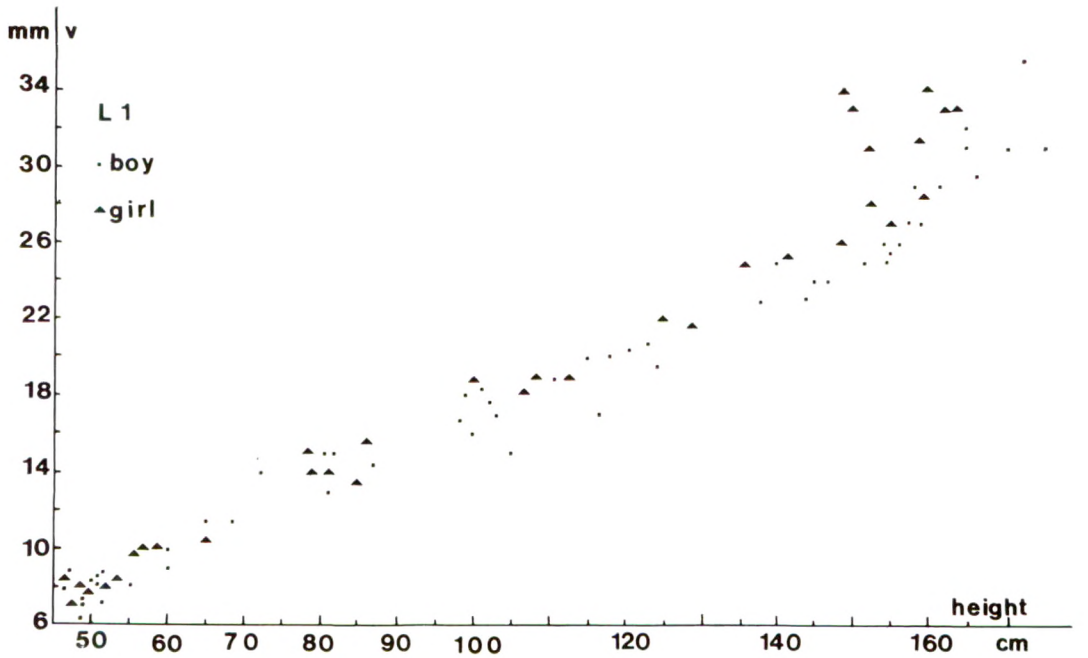


FIG. 7. Measured values of the vertical diameter (v) of the 1st lumbar vertebral body in function of the height of the children.

and Moseley[11] made similar measurements of v and s, but their results are not reliable because vb was measured at different levels of the spine, and only one index (s/v) was calculated; thus a certain error is induced, as proved by the comparison of the different levels of the spine in this study. However, one has the impression that their results correspond to the present findings in that newborns have flat rather than high vertebral bodies.

The *intervertebral disk index* is decreased after the second month of life because of a more rapid vertical growth of vb. It is not fully understood why, in the older age groups, the decrease of I_d is not more significant (Fig. 6). One factor might be a slight thickening of the disk with age. The decrease of I_d in Group v children can probably be explained by the pubertal growth spurt of vb. In a morphologic study Katz[7] suggested a decrease of the disk space in relation to the vb height, and he thought also that this space diminishes with age. This latter suspicion can now be ruled out by the present findings. The measurements of intervertebral disks in adult women by Hurxthal[5] show a slight increase of the disk space with age.

Based upon these results which define the normal pattern of growth of the vb and intervertebral disk, different osseous dysplasias, particularly mucopolysaccharidosis, will be further studied.[2]

SUMMARY

Vertical and sagittal diameters from the twelfth thoracic to the third lumbar vertebral body of newborns, children and adolescents were measured on 187 lateral roentgenograms which had been interpreted as normal. An index between the vertical and sagittal diameter ($I_{vb}=v/s$) was formed and values with double standard deviations were calculated. The index is significantly higher in the second month of life in D 12 and L 2 than in newborns. After the eighteenth month the index decreases, is stable, and from 4–12 years afterwards rises once more. There exists a sex difference which becomes significant in D 12 and L 1 for 4–12 year old children

and in pubertal children. Girls have a higher index than boys, which was explained by higher vertical diameters in girls older than 12 years or taller than 140 cm. and by higher sagittal diameters in boys.

At the same time, disk spaces in the embryologically corresponding segment of the investigated vertebral bodies were measured. An index between the disk and the next lower vertebral body height was calculated ($I_d = d/v$). This index decreases significantly after the first month and is almost stable until 12 years when it decreases once more. No significant sex differences were found.

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THE NOTOCHORD AS AN ABNORMAL ORGANIZER IN PRODUCTION OF CONGENITAL INTESTINAL DEFECT*

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THE purpose of this paper is to trace the role of the notochord in disrupting normal organogenesis of the intestine at the upper and lower poles of the body. Congenital sacral vertebral ageneses have been noted to occur, together with agenesis of the hind gut, from time to time. We have studied 58 consecutive cases of congenital defect of the hind gut for vertebral anomalies. The results are summarized in Table I.

The vertebral deficiencies are of types known to follow abnormal development of the notochord in the cervical region.[1,15] The notochord is usually regarded as a vestige showing a phylogenetic link with elasmobranch fish. It is an inconspicuous thread, and lies immediately ventral to the neural tube, as the earliest semblance of an axial cartilaginous skeleton. Its importance lies in its function of potent organizer, which on mesoderm induces formation of vertebrae. Where it becomes exposed close to primitive endoderm, severe disturbances of organization in the latter occur.

THE NORMAL ROLE OF THE NOTOCHORD AS AN ORGANIZER

During early fetal development of many organs, an exact sequence of changes has been discovered, when appearance of one tissue seems absolutely necessary if the next phase is to be triggered in its surroundings. The role of the notochord has been shown experimentally in such an organogenetic sequence. If parts of the neural tube are excised from early chick embryos, complete failure of vertebra formation follows in those segments. However, if the neural tube and its associated mesoderm segments are isolated from the embryo by transfer and graft to chorioallantoic membrane, notochord forms, then vertebral cartilage develops. Grafts of the mesoderm alone remain totally cartilage-free, although they must contain notochord precursor. Holzer[6] showed that it was the ventral embryonic spinal cord which had an "organizer" effect upon primitive somite mesoderm.

Notochord had a similar inducing effect, but other embryonic tissues did not induce chondroitin formation. Even these two tissues would only trigger induction for a particular, and exceptionally limited, period of fetal life. It is therefore reasonable to suspect than any interruption of this sequence, or even delay in its timing, will lead to major defects in the subsequent formation of vertebrae. It will be shown later that if the notochord organizer is abnormally exposed by faulty formation of vertebrae, then severe inhibition of contiguous endodermal fore and hind gut development is likely to occur.

The biochemical nature of the organizer activities is important but poorly understood. From isotope tracer studies it would seem that such inducer cells are sometimes sources of chemical monomers.[5] The latter are polymerized by the cells of contiguous tissues. For example, in some animal embryos, primitive ectoderm seems to produce a monomer necessary for final production of collagen by a polymerizing action in adjacent mesoderm. Collaborative activity of apparently widely different tissues gives insight into many other cir-

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Table I

Intestinal Anomaly	Male	Female	Vertebral Anomalies			Other Congenital Anomalies
			Thoracic	Lumbar	Sacral	
Extrophy of cloaca		3			1	3
Rectovesical fistula (above verumontanum)	11*		0	2	2	10
Rectovaginal fistula		8	1	1	3*	4
Anal ageneses	21	15	1	1 2	1 1	8 7

* Diabetic mothers in 1 case of rectovesical and 1 case of rectovaginal fistula only.

cumstances. For example, it has been puzzling that cultures of adult basal cell carcinomas of skin will not grow unless some of their closely adjacent dermis is included in a tissue culture with them. These examples show intertissue dependencies. It is for such reasons that each step in an organogenetic sequence is thought to be essential.

In the embryo, sequentially oriented cell growth over frameworks of matrix which has been functionally laid down becomes organogenetic. The older static ideas of strict division of embryonic tissues into ecto-, meso- and endodermal types must therefore be abandoned functionally.

The sequence of organogenetic steps for vertebrae appears to be the formation of a ventral spinal cord which induces notochord, and which in turn induces chondrification. The next steps are also important. Inhibition of further notochord development then occurs in segmental fashion, which permits fusion of the lateral chondrification centers, enclosing the thread of notochord, and cartilage is then replaced by bone. In between inhibited segments, notochord continues growing at the same rate as the rest of the body, forming nuclei pulposi and Schmorl's nodes in the intervertebral disk tissue.

It is now easier to understand why continued abnormal development of the thread-like segments of inhibited notochord into the nucleus pulposus diameters is usually associated with failures in full formation of vertebrae. This enlargement may be detected roentgenologically as "persistent notochordal canal." It is not a true persistence but overgrowth of the notochord to noninhibited size. Anterior fusion of chondrification centers is prevented, producing either "anterior spina bifida," butterfly or hemivertebral fusion anomalies, "block" vertebra, or even congenital absence of vertebra as a result. These anomalies may therefore be thought to be consequences of disorders of the notochord.[15]

Associations of cervical anomalies of these types with congenital malformations of fore gut have been noted. To our mind, they provide the basis for understanding of similar associations with hind gut ageneses. The associated cervical anomalies are now described as prototypes, with this aim in mind.

The Relationship of Notochord-Determined Vertebral Anomalies with Defects of Fore Gut Development

Ehrenhaft,[1] and Fallon and associates[3] showed, for example, that cervical "anterior spina bifida" (noninhibition of notochord) was often associated with fibrous bands connecting the exposed giant notochord with anomalies of fore gut. These

have included intramural esophageal cysts, gastrogenous cysts, and reduplications along the path of descent of primitive pharynx into the upper abdomen. Fibromuscular bands occasionally connect tracheoesophageal diverticula or atresias to cervical vertebral anomalies. The microscopic constitution of these bands is seldom described,[14] but they require study for it is not known whether they represent undeveloped minute residua of endoderm cords or abnormal segmental diversions of a vascular supply. In any event, they sometimes connect isolated patent gut segments, described as developmental cysts, with the rest of the fore gut, as well as with cervical vertebral congenital abnormalities of the types described above. It is as though the dominant connective tissue inductive capacity of the notochord produces inhibition of endoderm segments, should they lie in proximity.

In another example, esophagus has sometimes been found adherent to a fissure in the anterior aspect of the vertebral column. The most severe form of this is iniencephaly where there are congenital deficiencies, often with reduction in the number of cervical vertebrae, anterior spina bifida, and adhesion of gastric or esophageal extensions to hind brain. On any other basis, it is difficult to account for retention cysts of intestinal type that have sometimes been described in intrathecal positions.[14] In a cranial site, disturbance of another organogenetic sequence is sometimes evident in those cases where the upper end of the notochord has been found defective in the basisphenoid. Bifid optic cup induction is defective and cyclops appears, with separated nares. The priority of defect is uncertain. It is probably a primary ventral central nervous system failure, blocking proper notochord formation and thereby bone, in that sequence.

The fore gut anomalies must first occur while the fetal pharynx lies in the neck. The situation of cysts and elongation of the bands seem due to the insertion of bulky cardiopulmonary apparatus. This forces true cervical structures distally from their normal segmental position.

In summary, overgrowth of the notochord is a visual reminder of failure of inhibition of segments of this tissue. This may occupy the crucial time interval needed for another step in a rapid sequence. In consequence, subsequent organogenetic steps fail. Certainly contact of the notochord with endoderm seems to inhibit its development into normal fore gut. The so-called fine solid cords which are sometimes described may have the appearance of remains of noncanalized endoderm, but are usually fibrous.

An analogous situation seemed likely to us for sacrum and hind gut but less so for trunk and mid gut, for the following reasons. Normally the thread-like notochord arches far anteriorly at the head and tail poles. It can be traced to normal subperiosteal positions on the basisphenoid and also on the anterior surfaces of cervical and sacral vertebrae. Between, in the thoracolumbar regions, it lies very deeply, buried at the junction between the anterior two-thirds and posterior one-third of the vertebral bodies. The very mass of lateral cartilaginous centers there seems more likely to fuse, preventing exposure of the notochord through sagittal clefts, even when "persistent notochordal canal" appears. Association of mid gut anomalies with thoracic and upper lumbar vertebral anomalies is therefore possible, but less likely. This is confirmed by the scarcity of these reports in the literature.

THE RELATIONSHIP OF LUMBOSACRAL VERTEBRAL ANOMALIES TO DEFECTIVE FORMATION OF HIND GUT

It is now necessary to review evidence connecting these two sets of complicated conditions in the light of notochordal functions related to the opposite fetal pole.

Sacral vertebral defects of the types known to be associated with abnormal development of notochord such as central clefts, hemivertebrae, and total agenesis,

were first reported very infrequently and as isolated examples. In the 100 years up to Freedman's cumulative review[4] in 1950, only 43 cases of sacral agenesis had appeared in the literature. Fifteen of these also had anorectal or genitourinary abnormalities. Similar correlations in short series were recorded by Moore and Laurence[7] in 1952, Williams and Nixon[13] in 1957, and Partridge and Gough[8] in 1961. A noteworthy study was made by Stephens[12] in 1963 using applications of embryology. He distinguished between true rectal as opposed to the anal abnormalities and noted that sacral deficiencies were present in 17 out of 27 rectal (63 per cent) but in only 2 of 21 (10 per cent) anal malformations. From his personal collection he suggested that sacral agenesis was not in fact a rare condition. His series has the highest incidence of sacral anomalies with high rectal agenesis to date. In their series of 192 patients with low anal abnormalities, Partridge and Gough[8] showed that 16 (8 per cent) had sacral malformations compared with 13 (12 per cent) out of 106 cases with anorectal agenesis. The percentages in our series lie between. If lumbosacral vertebral anomalies are combined they were found in 16 per cent of anal ageneses and in 42 per cent of the rectal anomalies. The associations would thus seem real (Table II; Fig. 1–4). The relative variation may be due to a tendency to ignore minor sacral changes which are sometimes difficult to see in the newborn.

Table II

Authors	Percentage with Sacral Abnormalities	
	Anal Malformations	Rectal Malformations
Stephens[12]	10	63
Partridge and Gough[8]	8	12
Elliott *et al.*	6	27

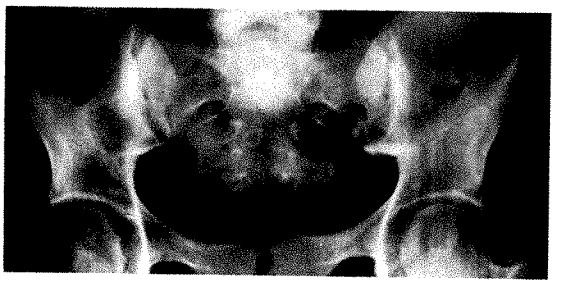


Fig. 1. Defects of S5 and 6 present in a patient showing congenital rectovaginal fistula and double right ureter. The mother was diabetic.

They may equally represent geographic variations in incidence and this requires further study.

In none of the series cited has it been possible to examine the precise state of the notochord in the sacrum. In Stephens' studies[12] there are correlations with sacral nerve distributions, and the neurologic deficits in his cases are known in detail. He showed close correlation between the absence of sacral nerves and deficiency in sacral segments. In 2 instances sacral

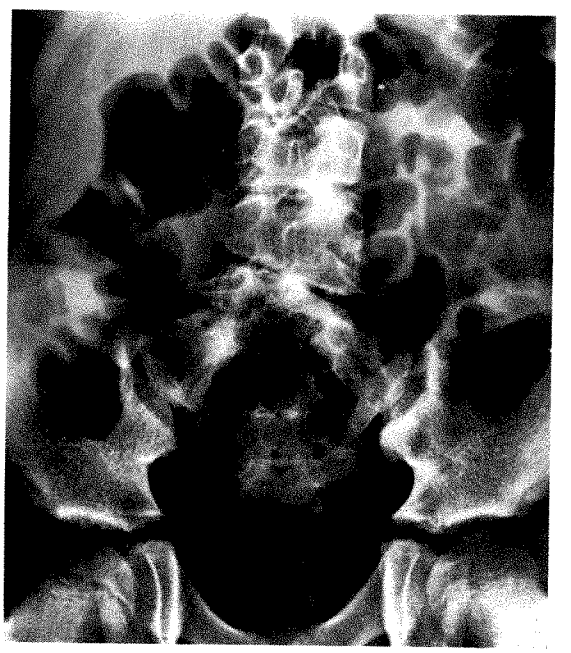


Fig. 2. Multiple vertebral defects in a boy with congenital rectovesical fistula. An anterior cleft is present in L2; L4 is fused with the left half of L5; and the right half is fused with S1. The coccyx and S5 are absent.

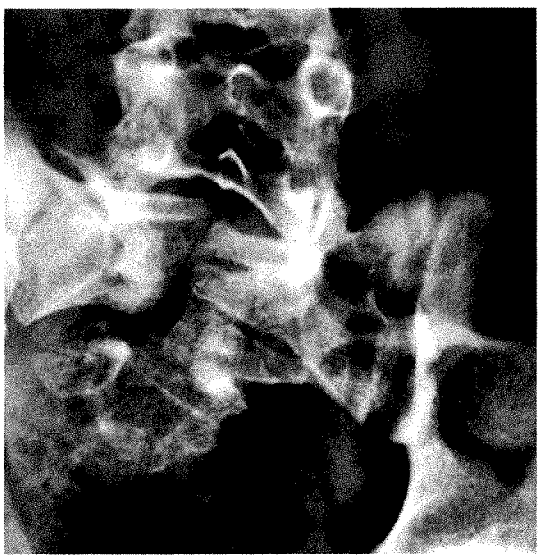


FIG. 3. A boy with rectal agenesis also showed fusion of L_4 and 5, spina bifida of S1, 2, 3 and absence of S_4 and 5.

nerves were present despite absence of corresponding vertebrae. This coincided with the clinical state in those patients. Most of his cases were without spinal cord defect. It therefore seems likely that any block in a sequence, which should lead to formation of lumbosacral vertebrae, occurred at notochordal-organizer, and not neural tube-organizer level.

By analogy with agenesis of the fore gut, it would seem that when sacral vertebral formation is markedly incomplete, then hind gut formation also is often inhibited. In 1 of Partridge and Gough's cases[8] they note an interesting finding that "from the proximal blind loop of bowel a cord of what appears to be fibrous tissue was seen passing back towards the sacrum. On section this proved to be a segment of atretic bowel." No counterparts for the thoracic, gastrogenous and esophageal cysts and reduplications are reported to our knowledge. We suggest that this is so, for in hind gut region no counterpart exists for displacements produced by insertion of a large cardiopulmonary visceral mass into the cervical region. While this forces the elongation of primitive pharynx from a true cervical position, no corresponding chain of events occurs in the sacral region.

Stephens' observations[12] led to a questioning of conventional embryologic belief. He stated "if downgrowth of uro-rectal septum were the only mechanism involved in cloacal division it would be expected that the commonest form of fistula in the male would be located at the level of the triangular ligament. The extreme rarity of a fistula at this level suggests that the subdivision of cloaca below this line is effected by a different process." In clinical terms, the inferior limit of the triangular ligament may be higher. Rectourethral fistulae at or above the level of verumontanum are common. In consequence, Stephens suggested a return to the earlier teachings of Wood-Jones,[16] that below Müller's hillock and the pubococcygeal line, insurge of mesenchyme from the lateral pelvic regions completes the separation which is begun superiorly by the urogenital diaphragm.

Although embryologic views on the development of this region vary considerably and are controversial, it must be considered that differentiation is taking place in minute structures. For example, the urorectal system begins at the 5 mm. stage and cloacal separation is completed in the 16 mm. fetus.

Partridge and Gough[8] opposed some portion of these views and noted, with reason, that "if the perineal portion of the rectum" developed from a postallantoic outgrowth of the cloaca "one might find a recto-urethral or a recto-vesical fistula without ano-rectal agenesis." This they state is not recorded. However, it may exist classified under another name as nonextrophic extrophy of cloaca as discussed below. For this reason we support Stephens' views.

A counterpart for iniencephaly is present in patients who have complete agenesis of sacrum. While some show high rectal agenesis we have seen one associated with true extrophy of cloaca (extrophia splanchnica). An excellent concise description of this condition is "an extrophic hemi-

bladder where a ureteric orifice lies on each side of the mid-line and between them is a zone of intestinal mucosa with an upper and lower orifice. . . . the upper opening being ileal termination. . . . the lower leads to a short length of large intestine which ends blindly in front of the sacrum." This severe condition seems to correspond with complete failure of cloacal partitioning. In the nonextrophic variety described, the anterior abdominal wall is intact and anomalies are internal. It has been proposed that this may be due to normal formation of the cloacal membrane. However, it may represent the missing type of recto-vesical fistula sought by Partridge and Gough.[8] In her review of 5 personal cases of extrophy of cloaca, with 38 others from the literature, Spencer[11] noted that anomalies of the vertebral column were present in 20 patients and absent in 4. Exact locations are not given. Sacral spina bifida occurred in 32 of 57 cases reviewed by Rickham and Johnston.[9]

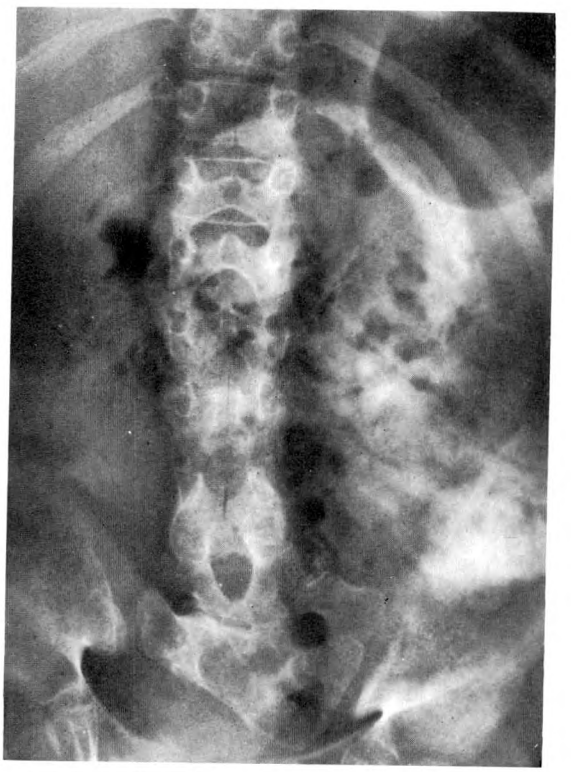


Fig. 4. A girl with extrophy of cloaca showed spina bifida in T11, block vertebra in L3 and 4, fusion of L5 and S1, absence of right hemivertebra in S2 and absence of S3, 4 and 5.

CONCLUSIONS

1. It is a departure in thought to realize that development of intestine may be dependent upon exclusion from influences of organizers for unrelated structures, forming in adjacent sites.

2. While the association of vertebral anomalies in cervical and lumbosacral regions with fore and hind gut ageneses is undoubted, much remains to be explained. The bony vertebral anomalies are of types associated with abnormalities in development of the notochord. This should serve to focus attention upon disorders of this inconspicuous but important organizer.

3. In the control of these lies the prospect of protection from some intestinal ageneses.

4. As the sequence of organogenetic steps is known to be crucially spaced in timing, it would seem likely that there is still a great deal to be learned of disturbed organogenetic sequences, from experimental embryology. With registration of congenital defects, other components of these disordered sequences may be discovered.

SUMMARY

Fifty-eight cases of congenital defects in hind gut were studied for associated lumbosacral vertebral anomalies. These were found in 16 per cent of anal and 42 per cent of rectal ageneses, respectively.

The vertebral deficiencies are of types known to follow abnormal development of the notochord in cervical regions. This inconspicuous thread, besides being the first semblance of a human axial skeleton, is an organizer. Where it overdevelops, it prevents the fusion of chondrification centers anteriorly. Severe inhibition of contiguous endodermal fore and hind gut is likely to occur. It is as though the dominant connective tissue inductive capacity of the notochord produces inhibition of endoderm segments when these lie in proximity.

The disturbances in sequential organogenesis when the notochord is exposed in cervical and sacral regions are reviewed and counterparts identified. Normal development of the intestine is therefore partly dependent upon exclusion from influences of organizers for unrelated structures, which are forming at an early age in adjacent sites.

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ROENTGENOLOGIC CHANGES IN IRON DEFICIENCY ANEMIA*

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THERE have been isolated case reports of iron deficiency anemia with the roentgenologic changes simulating those of chronic hemolytic anemia.[2,3,13] The roentgenologic changes in nutritional iron deficiency anemia described by these authors include widening of the diploic space and atrophy of the outer table of the skull with radial striations running perpendicular to the skull tables. The absence of involvement of the facial bones and, more particularly, of the long bones differentiates the roentgenologic changes of iron deficiency anemia from those of severe Cooley's anemia in infants and young children.

Since no comprehensive study on the subject has appeared in the literature, it was decided to investigate a group of infants and children with iron deficiency anemia as regards the roentgenologic changes.

MATERIAL AND METHOD

One hundred and sixty-four patients with nutritional anemia (hemoglobin level <12.0 gm. per cent) were selected from the Pediatric Hematology Unit, Maulana Azad Medical College, New Delhi, 70 per cent of whom were under 3 years of age. The roentgenograms of the lateral view of the skull and of one hand, including the long bones of the forearm, were chosen for evaluation. The roentgenograms were studied by the radiologists without knowledge of the hematologic data. The protein caloric malnutrition was assessed according to Gomez *et al.*[9]

HEMATOLOGIC INVESTIGATIONS

1. Hemoglobin estimations: cyanmethemoglobin method according to Crosby *et al.*[5]
2. Peripheral smear and bone marrow examinations: Giemsa stain.
3. Bone marrow iron estimation according to Dacie and Lewis.[6]
4. Serum iron estimation according to Ramsay.[11]
5. Unsaturated iron binding capacity according to Ressler and Zak.[12]

OBSERVATIONS

Of 164 cases 147 fulfilled the criteria for iron deficiency anemia: *i.e.*, absence of stainable iron in bone marrow; low iron saturation and lack of evidence of megaloblastic dysplasia in peripheral smear and bone marrow;[10] and response to iron therapy. The abnormal roentgenologic features were detailed in 100 of these 147 children (Table 1).

OBSERVATIONS IN 100 PATIENTS OF IRON DEFICIENCY ANEMIA WITH ROENTGENOLOGIC CHANGES

In all cases mild to moderate osteoporosis was observed; in 8 per cent changes consistent with rickets were also found.

The changes consisting of atrophy of the outer table of the skull, and osteoporosis and thinning of the cortex in the hand bones were observed in 90 per cent of the patients, while atrophy of spongiosa in the hand bones was noted only in 50 per cent of the patients.

Two patients showed widening of the diploic space in the skull, while loss of concavity in the mid-shaft region and thinning of the cortices of the metacarpals and phalanges were found in 1 of these (Fig. 1, *A* and *B*; and 2). There was no evidence of increased destruction of the red blood cells,

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TABLE I

CORRELATION BETWEEN ROENTGENOLOGIC CHANGES AND HEMOGLOBIN LEVEL

Hemoglobin (gm. per cent)	Skull			Hands					Long bones	
	Widening of diploic space	Outer table atrophy	Radial striations	Shaft osteoporosis	Thin corticalis	Widening of shaft	Destruction of spongiosa	Coarse trabeculated spongiosal pattern	Transverse metaphyseal lines	No. of patients
0–5	1	31	2	32	23	1	19	4	3	32
5–8	—	52	2	56	34	4	26	13	6	56
Above 8	—	12	—	12	3	—	1	—	—	12
Total	1	95	4	100	60	5	46	17	9	100

presence of abnormal hemoglobin, or rickets in this case.

The roentgenologic changes were not related either to the hemoglobin levels (mean 5.8 ± 2.1 gm. per cent; Table 1), or to the serum iron levels (46.5 ± 14.9 μg. per cent).

The transverse metaphyseal lines which are believed to be present in malnutrition were seen in 6 per cent of the patients.

DISCUSSION

The incidence of iron deficiency in childhood anemias is very high in India.[7] The possible etiologic factors could be: faulty and prolonged breast feeding; under and/or malnutrition; and poor socioeconomic status. Therefore, in overpopulated countries children suffer from various degrees of chronic iron deficiency anemia.

In the present study of 147 patients with iron deficiency anemia, the significant abnormality encountered consisted of osteoporotic changes in 100 patients. The changes of hemolytic anemia were present in only 1 patient, which would suggest that the roentgenologic changes observed in patients with hemolytic anemia are rare in iron deficiency anemia (incidence of 0.68 per cent).

It seems that there is a latent period of several months between the clinical onset of anemia and the roentgenologic evidence suggestive of marrow hyperplasia.[4] Eng[8] was first to demonstrate the relationship between iron deficiency anemia and the roentgenologic changes in the skull (*i.e.*, cortical thinning and diploic widening in the frontal and parietal bones). Similar marrow hyperplasia has also been reported

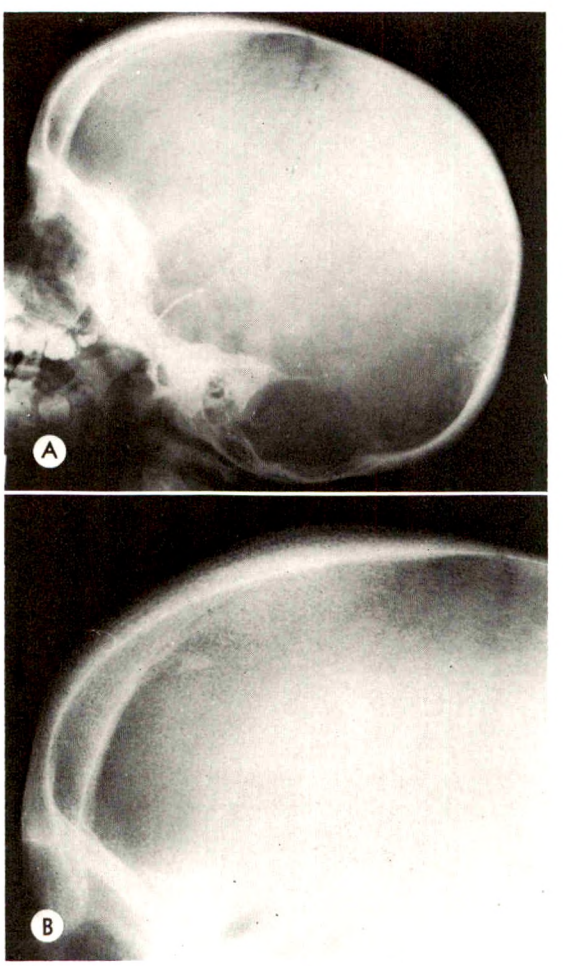


FIG. 1. (*A*) Lateral view of the skull showing widening of the diploic space with outer table atrophy in the frontal region. (*B*) Localized and magnified view.

in patients with congenital cardiac anomalies associated with cyanosis and polycythemia.[1]

The exact cause of this consistently observed osteoporosis in the present study is difficult to explain. In protein caloric malnutrition, delayed roentgenologic appearance of ossification centers and local or generalized rarefaction of bones may be seen. The patients in the present study had Grade I–II protein caloric malnutrition which could be responsible for the osteoporosis observed. However, the presence and degree of osteoporosis did not correlate with the serum iron and hemoglobin levels.

SUMMARY

In a roentgenologic survey, 147 children with iron deficiency anemia were found to show variable degrees of osteoporosis and only 1 patient presented changes suggestive of hemolytic anemia.

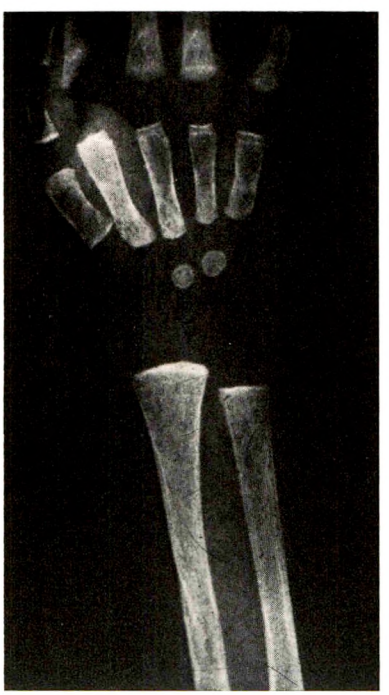


FIG. 2. Anteroposterior view of the right hand showing loss of concavity in the mid-shaft region of the metacarpals, with thinning of their cortices.

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EDITORIALS

COMPUTERS AND RADIOLOGIC PRACTICE*

COMPUTERS and radiology at the outset of their association seemed ideally mated for the creation of exceptional offspring. The phenomenal ability of these machines to accept, retain, retrieve, display, manage and analyze alphermeric data selectively, mathematically and logically astounded all radiologists who worked with them. Certainly strong mutual attractions and great potentialities for real advances in radiologic practice do exist in this association as shown by the few sound contributions which have been realized. But so far these modest attainments have produced no major improvement or substantial change in daily radiologic practice, nor indeed does any seem imminent. As a matter of fact, formidable obstacles, some apparently unrecognized, or at least not fully appreciated, remain to be overcome before the potent, latent abilities of the computer can materially influence the specialty clinically and benefit patients directly.

As persuasive, often exacting and demanding entrants into an increasing number of the processes and procedures of radiology, computers now deservedly warrant prime consideration. Perceptively employed, they can act to broaden the scope of radiologic practice in many ways and to strengthen its role in medicine. They encourage long overdue efforts towards the development of more precise radiologic terminology, better classification of disease and more satisfactory systems of coding. The formulation of thesauri for segments of radiologic reporting has begun. In special limited situations these versatile machines can now be made to indicate the proper sequence for carrying out examinations, perform simple measurements, pass on the thoroughness and selectivity of data gathering and check on the completeness of a radiologic work-up. Computers can reinforce certain kinds of decision making. They cope with more data than the mind can successfully handle, and they do so without bias and emotion. Multivariate statistical analysis depends upon them. They have achieved real progress in scheduling, payroll, billing and similar business activities, all matters of some concern in radiologic practice. Minor inroads have even been made into the radiologist's special preserve, data gathering, an intrusion furthered by image enhancement and similar technical innovations. The applications of Bayesian calculations and related formal ways of arriving at medical decisions have been applied in selected circumstances. Computer based calculations now speedily furnish precise, complicated mathematical determinations for treatments employing interstitial and external irradiation.

Nevertheless, much of this output has had little direct clinical impact, and some actually represents elegantly derived answers to situations that simply do not apply in day-to-day work. The capability of radiophysicists with computers to calculate doses and to formulate complex data into treatment plans have been as impractical in some clinical settings as most of the machine based radiologic diagnoses have been in other spheres of patient care.

Why have inadequacies such as these arisen? Why has practical help for the radiologist been so meager? What accounts for the computer's present lack of influence on daily radiologic work?

Stated most generally, the obstacles to greater computer productivity in radiologic practice relate to input, the interface between the radiologist-investigator, program-

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mer, systems analyst and biostatistician on the one side and the machine and its requirements on the other. Most prominently operative here are many vexing conditions inherent in the varied decision processes of diagnosis and therapy including the built-in, inescapable limitations of both radiology and of medicine itself including its imperfect taxonomy. It is chiefly within these complex general areas where the opportunity to make real headway lies.

The reliability and the completeness of the raw data supplied to the computer deserve first serious thought. These qualities continue to depend upon the motivation, training and experience of the radiologist. As of now and for the foreseeable future, computers cannot compete effectively with the eye and the mind of the trained radiologist in most types of data gathering. Consequently, strenuous efforts should be made toward improvement of crucial data identification through the use of improved methods of visual perception guided by more clinically astute mental recognition. When dealing with controversial material and equivocal findings as radiologists often must do, then concerted attempts to lessen the serious drawbacks of both interexaminer and intraexaminer variability of reporting are also required. Finally, radiologists eventually must learn how to eradicate most types of judgement errors in diagnosis and treatment. These three broad fundamental approaches will provide the best immediate ways to increase the practicality of computer output by ensuring the validity of the raw data. In other words, the primary responsibility toward eventually achieving real clinical usefulness for computers begins with the radiologist, not the machine.

Once assembled, these data, largely nonnumeric and somewhat subjective in character, must be digitized or similarly prepared for machine consumption —no small task in itself. It requires clinical perceptiveness based upon an extensive experience with the particular disease under consideration plus knowledge of the demands of the computer itself.

Lastly, a sound program for the productive handling of this information by the computer needs to be devised by the investigator-radiologist and his co-workers. Not the awesome speed of the machine or its operative perfection, but the clinical acumen used in data selection, the reliability of its preparation for computer use, and the medical soundness of the programming determine the worth of the information the machine will eventually extrude.

In regard to the use of computers for radiologic decision making, important guiding principles have now emerged. First, radiologists must not embrace the false premise that their specialty always offers the only source or commonly serves as chief supplier of necessary data for decision formation in medicine. Consequently, an initial test of the practicality of any computer-radiology program for clinical application requires that as a minimum the specialty possess the potentiality of competing successfully with, or furnishing urgently needed confirmation to, the other relevant sources available to physicians for medical decision making. For example, in the management of any disease, where informational providers less hazardous, more reliable, and more specific than radiologic ones can be utilized, computer based probability calculations lose their fullest justification. Similarly, when life threatening situations in patient care confront the responsible physician (*e.g.*, the possibility of a malignant bone tumor, a cancerous gastric ulceration, or a malignant pulmonary nodule), proper definitive treatment ordinarily demands the highest possible degree of diagnostic reliability and specificity. Radiology often is inherently incapable of satisfying these two rigid requirements. Nevertheless, other medical methods may be quite qualified to produce them.

Another vital point: radiologic diagnosis should not be considered merely as a single, indivisible item, the selection of the name of one disease which best fits the patient's findings. Rather it always consists of the detection or the exclusion of an abnormality, a description of the gross pathology

and of any dysfunction caused by the lesion, and lastly a judgement as to its nature or etiology. Computer programs dealing with diagnosis largely pass over the first mentioned contributions by concentrating upon the last one. Furthermore, even the best of final diagnostic and therapeutic judgements may have to be staged or graded.

Radiologic decisions often stand complete and self-sufficient, unaffected by any other medical information. Many are simple to make, entailing no complex reasoning; some are pathognomonic; many, while not specific etiologically, nevertheless rest upon absolute certainty, not upon probability. Consequently, much of the radiologist's daily work as it is now done, and apparently must continue to be done for the future, needs little or no backing from computers, probability calculations or indeed from any other kind of ready reference material including texts.

Additional important considerations remain. Evidence indicates that for computers to serve in formulating decisions which rely upon Bayesian probability concepts, certain stringent conditions must be met. There needs to be an established taxonomy of the disease, essential data sources must be readily available from which to extract the patterns that characterize the disease, and these data must be quantifiable. However, even when these requirements have been satisfied, more or less serious, built-in objections to the use of this probability theorem must be faced. These relate to the presumed independence of the findings and the difficulty of determining the extent of the population under study.

So far, computers cannot really duplicate the intuitive capacity of the mind, that rather mysterious ability to leap to new insights without the conscious employment of formal reasoning processes. The total abandonment of intuitive radiologic decision making in favor of logical, methodical reasoning would be quite unrealistic at this juncture. This substitution, no matter how preferable in ensuring the reliability of decision making, cannot be made until our most intricate thought processes have been more clearly defined than they are at the present time.

In the future how can more substantial and realistic support be obtained from computers to help the radiologist meet the crucial problems of daily patient care?

In order to avoid clinically inefficacious output from computers, the really critical decisions of radiologic practice must first be searched out and clearly labeled on the basis of broad agreement among radiologists. To give one example: most radiologists believe that it is vastly more important to ascertain from their examination that the possibility of a malignant bone tumor exists than to have determined by formal probability calculations that the likelihood of the correct diagnosis being fatigue fracture rests at 95 per cent. Clearly, this last opinion in spite of its elaborate derivation overlooks the really critical clinical problem, the chance, slight though it may be, of a life threatening disease. It follows from this that unless the computer's abilities are thoughtfully and productively applied to the really basic problems of radiologic practice, then no matter what innovations arise in information processing and in decision solving, the results can never be useful and valid.

When the proper clinical situation for constructing probability based decisions does appear, radiologists have not one, but two general approaches to consider. The philosopher defines probability as the likelihood of an event's taking place as based upon certain observations and as related to the rationality of the situation. The mathematician's route to the possibility of a disease being present rests upon Bayes' theorem which states that the chance is directly proportional to the incidence of the disease and the frequency of a given set of findings presented by the patient for that disease. The philosopher's concept with its reference to rationality, *i.e.*, clinical feasibility, must be thoroughly considered first before proceeding to any mathematical determination of probability. Full

awareness of the clinical consequences of the result contemplated must always form an essential part of the total responsibility of any decision making process.

Clearly much more has to be learned about the basic nature of radiologic diagnosis and therapy, especially their roles in medical practice and how each relates to the care of the individual patient and to the management of each particular disease. We should seek better delineation and further appreciation of the clinical significance of the inherent limitations of diagnostic and therapeutic radiology and of medicine itself. At the same time we must continue to make progress in refining and extending the more promising of the work with computers which we referred to previously.

The pressing clinical problems of radiotherapy including how to determine the exact extent of the neoplasm, the effect of host resistance on tumors, the biologic nature of the individual neoplasm under treatment, the vagaries of the human anatomy, the action of adjunctive chemical agents on the tumor response to radiotherapy, the obscurities of the cancer cell migration and growth in the body and the results of irradiation on normal tissues demand better elucidation before computers can become more effective in the management of patients with cancer.

Present trends favor the use of smaller machines for specific tasks over earlier attempts to employ more costly, complex computers on a time sharing basis. After initial processing, the refined data can then be transferred into central computer systems. As to the machines themselves, if they could be adapted to collect and select data in more productive ways than the radiologist himself now can, then a major breakthrough would be attained. Also, if computers could be used to develop fresh data sources otherwise inaccessible to radiology, another valuable advance would result. Providing the radiologist with the significant facts for decision making from a mass of detail too vast or too complex for ready memory recall offers another substantial contribution, and one which seems nearer to accomplishment. Certain radiologic measurements when supported by electronic techniques certainly could be made more dependably than by present visual means. Since a definite change in size or shape often constitutes significant medical information influencing patient management, here again would be another area for very practical assistance to the radiologist from the computer. The use of subtraction techniques with matching of all subsequent films to the normal, original roentgenogram which would be used as the standard, offers the possibility of automated film reading in periodic screening projects.

When the radiologist does need to seek information from a computer, it would be advantageous to have it delivered to him on the basis of whatever body system, anatomic part or disease category he might specify. Also needed are computer listings of those diseases in which radiologic decisions have special significance or pivotal influence upon the management of the patient. Labelling the clinical limitations of radiology for computer recall as they might bear upon the diagnosis and treatment of certain diseases would also be extremely helpful. For example, most practicing radiologists and physicians would welcome information indicating the radiologic latent period of certain bone diseases, *i.e.*, the time between the appearance of symptoms and the first possible film abnormality. Crucial diagnostic points needed for the identification of many uncommon conditions offer still another means of supporting the work of the clinical radiologist, although problem cases could be referred by computer-communication systems to experts in that particular category of disease. A listing of the significant medical and laboratory findings needed to support any particular radiologic diagnosis under consideration would also be possible and helpful. So, too, would an outline of the indicated radiologic examinations to be

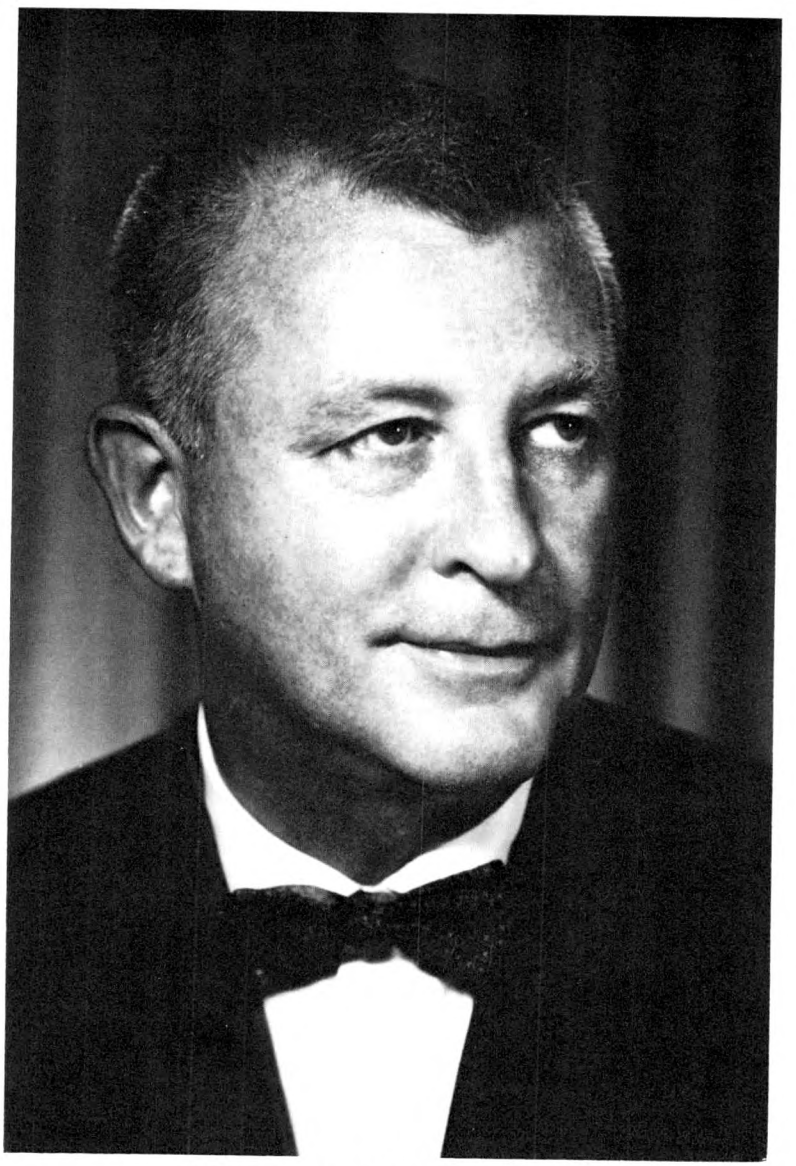
done at any particular time of the patient's work-up. Review courses at the post-graduate level could be made easily available to radiologists through computers, no matter the distances involved. Computers could be used in numerous ways to help ease the manpower shortages plaguing radiology at this time.

Diagnostic and therapeutic radiology have to recognize one vitally important principle, one which will always govern a significant part of their association with the computer. Applications of this machine to too narrow, impractical questions will deliver answers wholly inadequate for use at the bedside as shown by past computer research in radiologic decision making, much of which seems to have issued from sources having a very restricted concept of the nagging problems of day-to-day radiologic practice. The result has been that mathematical skills have overbalanced radiologic perceptiveness and technical triumphs have been made at the expense of clinical common sense.

We must now recognize that some radiologic decisions fall into a special class of problems referred to as complex, multiple loop, non-linear feed-back systems, where not isolated decisions of a simple nature, but system structure, multiple decision points and policy changes govern the outcome. Only by developing fully integrated, effective interrelationships between practicing radiologists of proven ability in the particular subjects being investigated and the computer with its supporting personnel will sound programs of decision making for the individual patient eventually be realized. Furthermore, in order for radiology's own computer outputs to be acceptable for integration into the broader ones of general medical practice, they must be carefully and discernedly formulated and accurately executed. As a matter of fact, computers properly programmed could actually enable a radiologist to diagnose a disease he had not known existed. Since computers tend to downgrade demands on memory, theoretically they could eventually do away with the need for most of it.

In the next decade dramatic innovations in the computer-radiology association probably will not come from any improvement in the machine itself, but rather from more practical, thoughtful and ingenious applications of this amazing device. For the future, instead of procuring a computer and then trying to find ways to apply it to radiologic practice, investigators should identify problems first, see if they are solvable by computer techniques, and then select the machine best suited to do the work. The period of false starts and expansive prophecies draws to a close as model systems, clinical experiments and trial runs under actual practice conditions begin to operate. Providing the talents of these machines are employed with ingenuity and sagacity, no recent development promises to be as effective as the computer in bringing radiologists and other physicians closer together in the delivery of patient care. Radiologists and their colleagues in medicine may have to learn several fresh abilities including a new language in order to communicate with each other through centralized information centers.

Robert S. Sherman, M.D.



JOHN E. GARY, M.D.
1916-1970

THE sudden, completely unexpected death of Dr. John E. Gary, probably from coronary disease, came as a shock to his many friends. A valuable man who had so much to offer in the years ahead died prematurely.

John Gary was born at Coquille, Oregon in 1916. He spent his youth and the greater part of his training in the Northwest, going to college at Willamette University and to the medical school at the University of Oregon. A leader in medical school, he finished at the top of his class. He took his internship at the Philadelphia General Hospital, where he came under the influence of Dr. Herman Ostrum and decided to become

a radiologist. After a short training period in the Army X-Ray School in Memphis, he became my assistant at McGuire General Hospital in Richmond, Virginia. He then spent the rest of the war years as Chief of Radiology at a Station Hospital in the Aleutian Islands. Fortunately for John, the Commanding Officer of the hospital, Dr. Henry Moehring, was a radiologist himself.

After almost two and a half years of radiology in the service, he started his regular residency with Dr. Golden at Columbia's Presbyterian Hospital in New York. At the end of 1949, he joined me at the Mount Auburn Hospital in Cambridge and became my partner in private practice. The fruitful and highly enjoyable years of our association were also years of constant growth of the Department. In January 1967 he took over as the new Chairman and was immediately confronted with a marked expansion of the Department of Radiology. He died before the new department was finished.

John was an excellent clinical radiologist, and his advice was greatly valued by the physicians with whom he came in contact. He had a great ability of condensing problems and coming to simple and practical conclusions.

Although he preferred clinical to academic radiology, having refused the offer of an academic career, he wrote a number of valuable publications. Several of them concerned the gastrointestinal tract. He was particularly interested in the radiology of occupational diseases. This interest was stimulated further by his connection as radiologist to the Massachusetts Institute of Technology and resulted in publications about beryllium and radium poisoning. A long term extensive study on the effects of mesothorium and radium had not been completed at the time of his death.

John was Assistant Clinical Professor of Radiology at Harvard Medical School. He loved to teach. Many students remember gratefully the elective month in radiology with him at the Mount Auburn Hospital.

John was an extremely fair person. He basically believed people to be good and—when in question—gave them the benefit of doubt. On the other hand, he did not hesitate to make decisions, even if they were unpopular ones. Although often pleasantly boisterous, particularly when he was with his friends, he was basically a humble man. This combination explains the great influence that he had as an arbiter. He became President of the staff of the hospital. The New England Roentgen Ray Society made him its Secretary and its President.

John enjoyed life. He was attractive, friendly, liked to have fun with others, whether he played golf or just sat with them and chatted. He was loved by many and I cannot remember a single person who did not like him. He became my friend very shortly after we met at McGuire General Hospital.

He leaves behind his charming wife, Maida, and three children, John, Janet, and Wendy. He also leaves two sisters as well as his parents. He was their only son.

Richard Schatzki, M.D.

1180 Beacon Street
Brookline, Massachusetts 02146

NEWS ITEMS

FOURTEENTH ANNUAL POSTGRADUATE COURSE IN DIAGNOSTIC RADIOLOGY

This course will be held Monday, March 8–Friday, March 12, 1971, in Medical Sciences Auditorium, University of California, San Francisco.

The course is designed for the radiologist in clinical practice to provide a survey of selected subdivisions of diagnostic radiology.

All registrations must be made in advance, as attendance is limited. No daily registration is available.

The Guest Faculty includes: Roy Astley, M.D., Birmingham, England; Morton A. Bosniak, M.D., New York, New York; Benjamin Felson, M.D., Cincinnati, Ohio; Robert H. Freiberger, M.D., New York, New York; E. Robert Heitzman, M.D., Syracuse, New York; M. T. Khilnani, M.D., New York, New York; Ronald O. Murray, M.D., London, England; Stewart R. Reuter, M.D., San Diego, California; and Frederic N. Silverman, M.D., Cincinnati, Ohio, in addition to the Faculty, Department of Radiology, University of California, San Francisco, California.

For further information please write to Department of Radiology and Continuing Education, Health Sciences, University of California, San Francisco, California 94122.

COLUMBIA UNIVERSITY COLLEGE OF PHYSICIANS AND SURGEONS

A Postgraduate Course on "Radiology of the Head and Neck" will be held under the direction of Guy D. Potter, M.D., March 18–20, 1971.

The topics to be considered will be: Radiological diagnosis of lesions of mandible, salivary gland, pharynx, larynx, swallowing, paranasal sinuses, and ear. Emphasis will be placed on differential diagnosis and the role of radiology in surgical planning.

The Faculty will consist of: Drs. Conley, Baker, Valvassori, Seaman, Potter, Basek, Rankow, Hanafee, Zizmor, Waltner, Mandel, and Zegarelli.

For information and registration contact: Melvin D. Yahr, M.D., Associate Dean, 630 West 168th Street, New York, New York 10032.

WORKSHOP IN PEDIATRIC RADIOLOGY

The Fifth Annual Workshop in Pediatric Radiology will be held at Children's Mercy Hospital, April 26–30, 1971. This Workshop is being held in cooperation with the University of Missouri School of Medicine.

Registration will be restricted to 5 pediatricians and 5 radiologists. This limited enrollment will permit the curriculum to include participation in daily patient care, special procedures, guest professor lectures, teaching film seminars, and scientific exhibit reviews. The Guest Professor will be John Kirkpatrick, M.D., St. Christopher's Hospital for Children, Philadelphia, Pennsylvania.

For additional information contact Chas. E. Shopfner, M.D., Head, Department of Radiology, Children's Mercy Hospital, 1710 Independence Avenue, Kansas City, Missouri 64106.

SEMI-CONDUCTOR DETECTORS FOR NUCLEAR MEDICINE

A Symposium on the role of "Semi-conductor Detectors for Nuclear Medicine" will be held at the Marriott Inn, Chicago, Illinois on February 12 and 13, 1971.

The symposium is intended for physicians and medical physicists desiring to learn about the properties and uses of these new radiation detectors.

The topics will include. 1. Physical principles of semi-conductor detectors; 2. recent advances in detector materials; 3. nuclear imaging devices utilizing semi-conductors; and 4. fluorescent counting and scanning techniques.

Application forms and further informa-

tion may be obtained by writing to Dr. Paul B. Hoffer, Department of Radiology, University of Chicago, 950 East 59th Street, Chicago, Illinois 60637.

LASER SAFETY CONFERENCE AND APPLICATIONS COURSE IN CINCINNATI

Under the combined auspices of the U. S. Public Health Service, Bureau of Radiological Health, and the Laser Laboratory of the Medical Center of the University of Cincinnati, there will be an International Conference on Non-Ionizing Radiation Safety in Cincinnati on March 29–31, 1971.

This is to include laser, microwave and ultraviolet. Literature on current safety programs for laser, microwave and ultraviolet will be sent in advance to all registrants.

Following this, through the Continuing Medical Education Division of the College of Medicine, University of Cincinnati, there will be a course on The Applications of the Laser in Biology, Medicine and Dentistry, with laboratory and operating room exercises, on April 1–3, 1971.

Requests for information should be directed to: Laser Laboratory, Children's Hospital Research Foundation, Cincinnati, Ohio 45229.

SOCIETE EUROPEENNE DE RADIOLOGIE PEDIATRIQUE

The Société Européenne de Radiologie Pédiatrique meets in Elseneur, Denmark, May 12–15, 1971.

The President is Dr. Gregers Thomsen, Rigshospitalet (University Hospital), Blegdamsvej 9, DK 2100 Copenhagen, Denmark.

The Permanent Secretary is Dr. Clément Fauré, Hôpital des Enfants-Malades, 149, rue de Sèvres, Paris 15e, France.

FIRST WORLD CONGRESS OF NUCLEAR MEDICINE AND BIOLOGY

The First World Congress of Nuclear Medicine and Biology will take place in Montreal, P.Q., Canada, August 20–September 4, 1971.

The Congress is sponsored by the World Federation of Nuclear Medicine and Biology and by the Society of Nuclear Medicine (U.S.A.).

The scientific program of the Congress is expressed by its theme. "Nuclear Medicine —The Second Generation."

The thematic program will attempt to evaluate the progress achieved by the biomedical specialties during the past generation as well as the contribution of nuclear sciences to this progress. This will be developed in a number of panels covering the major clinical and fundamental aspects, as well as some timely topics such as aerospace medicine, environmental medicine, etc. The conclusions of the panels will, in turn, be discussed in plenary sessions, where the broad lines of development will then be outlined and the most urgent needs defined.

Other specialized subjects will be treated in separate symposia, where well defined technical subjects will be analyzed in depth. The Scientific Program Committee is considering a number of topics, such as: computer sciences and nuclear medicine; high resolution radio-autography; tracer elements, etc. Suggestions for other topics will be considered until December 15th, 1970.

Problems of teaching and training in nuclear medicine and biology will be discussed in plenary sessions.

Free papers will be accepted, only insofar as they report original contributions or new developments in radionuclidic procedures. Also, a limited number of films or videotapes will be accepted, when specific techniques are presented.

Technical and commercial exhibits are accepted; the Administration of the Society of Nuclear Medicine (U.S.A.) is in charge of the arrangements with the organizations who contemplate such exhibits.

All those interested in attending the Congress and in contributing to its program are requested to apply for information and for

application forms to: Administrative Office, The Society of Nuclear Medicine, 211 E. 43rd Street, New York, New York 10017, U.S.A.; or Secretariat Général du Premier Congrès Mondial de Médecine et Biologie Nucléaires, Université de Montreal, B.P. 6128, Montréal, Canada.

FOURTEENTH W. EDWARD CHAMBERLAIN LECTURE CORRELATING RADIOLOGY AND PHYSIOLOGY

The Fourteenth W. Edward Chamberlain Lecture will be given Wednesday, November 25, 1970–4:00 P.M., on "Bronchial Dynamics in Health and Obstructive Airway Disease: Physiology and Roentgenology" by Robert G. Fraser, M.D., Professor of Radiology, McGill University, Montreal, Canada.

The Lecture will be held in the Lecture Room A, Kresge Building, Temple University Health Sciences Center, Philadelphia, Pennsylvania.

FIRST ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY

Although the International Society of Radiology has not been concerned directly with the promotion of zonal activities, two large regional groups, the European Association of Radiology and the Inter American Society, conduct regional congresses at four year intervals about midway between the International Congresses.

As a step towards filling a need which exists in those areas not catered to by these Societies, at the instigation of Professor Kempo Tsukamoto, President of the International Society of Radiology, the Asian and Oceanian Society of Radiology was formed at the conclusion of the Twelfth International Congress of Radiology (Tokyo, 1969).

The sphere of influence of the new Society will extend from the Hawaiian Islands in the East to Pakistan in the West and as Far North as Japan and South Korea and as far South as Australia and New Zealand.

At the Inaugural Meeting in Tokyo delegates from 12 countries in the region accepted the invitation of the College of Radiologists of Australasia to conduct the First Congress of the Society in Melbourne, Australia, from November 22 to 26, 1971. Professor W. S. C. Hare of Melbourne, Australia, was appointed President of the Society and Dr. J. J. Martin, also of Melbourne, Honorary Secretary.

Although this is a Regional Congress, an invitation is extended to Radiologists in other parts of the world to attend.

For further literature concerning the Congress write to: The Secretary, First Asian and Oceanian Congress of Radiology, Box 805F, G.P.O., Melbourne, 3001, Australia

BOOK REVIEWS

DIAGNOSIS OF DISEASES OF THE CHEST: AN INTEGRATED STUDY BASED ON THE ABNORMAL ROENTGENOGRAM. Two Volumes. By Robert G. Fraser, M.D., F.R.C.P.(C), Professor of Diagnostic Radiology, McGill University, and Diagnostic Radiologist-in-Chief, Royal Victoria Hospital, Montreal, Canada; and J. A. Peter Paré, M.D., Associate Professor of Medicine, McGill University, Joint Cardiorespiratory Service, Royal Victoria Hospital, Montreal, Canada. Cloth. Pp. 1,446, with 1,268 illustrations. Price, $48.00 U.S.; $51.85 Canada. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1970.

This text dedicated to Dr. Felix G. Fleischner is most outstanding and its excellence is a fitting memorial to this great scholar, teacher and pioneer in Radiology.

As stated by the authors, the book was written with the aim of defining an approach to the diagnosis of diseases of the chest based on the abnormal roentgenogram. Knowledge of structural changes thus obtained is integrated with pertinent clinical findings, results of pulmonary function tests, and other ancillary diagnostic procedures, enabling one to arrive at a confident diagnosis.

Tables of differential diagnosis, listing etiologic classifications of diseases which produce specific roentgenographic patterns, as well as briefly describing the clinical and laboratory characteristics of each disease to facilitate recognition of disease states, were designed for use with the text. When a specific pattern of disease is recognized the appropriate table is then scanned, and only those conditions which correspond most closely with the clinical picture presented by the patient, are selected. Additional information about the most likely diagnostic possibilities can be obtained by referring to the detailed discussions in the relevant sections of the text, as the page numbers are cited after each diagnosis. The tables relate to 17 basic patterns of bronchopulmonary, pleural and mediastinal disease, and each table is preceded by a detailed description and representative illustrations of the specific roentgenographic pattern.

The book, consisting of 2 Volumes, contains 7 Chapters in Volume I, which include: The Normal Chest; Methods of Roentgenologic and Pathologic Investigations; Roentgenologic Signs in the Diagnosis of Chest Disease; Tables of Differential Diagnosis; Pulmonary Abnormalities of Developmental Origin; and Infectious Disease of the Lung. In this Volume the presentation and accompanying illustrations of normal anatomy, physiology and pulmonary function studies are magnificent and can be used as a reference work by practitioners in all branches of medicine.

The remaining 12 Chapters in Volume II consist of: Presentations of Neoplastic Diseases of the Lungs; Embolic and Thrombotic Diseases of the Lungs; Pulmonary Hypertension and Pulmonary Disease of Cardiac Origin; Diseases of Altering Immunologic Activity; Inhalation Disease of the Lungs; Diseases of the Airways; Pleuropulmonary Disease Caused by External Physical Agents; Diseases of the Lung of Unknown Origin; Diseases of the Pleura; Diseases of the Mediastinum; Diseases of the Diaphragm and Chest Wall; and Respiratory Disease Associated with a Normal Chest Roentgenogram. An outstanding and most convenient bibliography of 4330 references is supplied in the second volume and an index of first authors of cited literature is supplied in both volumes. In addition, the subject index is also duplicated in both volumes.

The reproduction of the roentgenograms both by the logEtronic method and by direct photography again demonstrates the excellence of the preparation of this monumental work. Furthermore, sufficient space is allotted for reproduction of the roentgenograms in a size adequate for good detail recognition. The number of diagrams, drawings, and tables accompanying the text is most generous, thus allowing for easier comprehension of many difficult areas of subject material.

The authors humbly recognize that inevitably some of the observations of a text of this magnitude will prove erroneous in time or will find disagreement among many of the knowledgeable readers, but this does not diminish in any way from the great worth of this textbook. Pulmonary diseases peculiar to children are

mentioned only in passing as complete descriptions would demand treatment by a fully separate text.

The purpose of this book, emphasizing the value of the roentgenogram as a first, rather than the major step, in the diagnosis is most adequately fulfilled. In spite of the fact that the clinical morphologic and laboratory aspects of many diseases are described at greater lengths in many areas than the roentgenologic changes, this should not detract from the fact that this book should be an indispensable part of every roentgenologist's library. In fact, this text is recommended as a valuable addition to the library of all practicing physicians.

E. Nicholas Sargent, M.D.

Textbook of Nuclear Medicine Technology. By Paul J. Early, B.S., Physicist, Nuclear Medicine Institute, Cleveland, Ohio; Muhammad Abdel Razzak, M.B., B.Ch., D.M., M.D., Assistant Professor, Medical Unit and Division of Nuclear Medicine, Faculty of Medicine, Cairo University, Cairo, U.A.R.; and D. Bruce Sodee, M.D., F.A.C.P., Associate Professor of Radiology (Nuclear Medicine), George Washington University, Washington, D.C., Director, Nuclear Medicine Institute, Cleveland, Ohio, Director, Nuclear Medicine Department, Hillcrest Hospital, Cleveland, Ohio. Cloth. Pp. 378, with 241 illustrations. Price, $15.50. C. V. Mosby Company, 3207 Washington Blvd., St. Louis, Mo. 63103, 1969.

Individuals enter the field of nuclear medicine technology with varied backgrounds which make it very difficult to assemble a general purpose text on the subject. The manpower shortage is at all levels in nuclear medicine and the need for a text aimed primarily at the nuclear medicine technologists is great. The authors are to be congratulated in that they are the first to publish a book for this stated purpose and, at least temporarily, this text is unique.

The general factual content of the book is acceptable, although many of the illustrations are difficult to interpret and a few are incorrect if taken at face value. Chapters 1 and 2 are standard explanations of basic concepts. In Figure 3–2 amplitude is labelled wavelength, but the remainder of the chapter as well as Chapter 4 has no major inaccuracies. Chapter 5 presents the philosophical concept of checking for Mo^{99} in Tc^{99m} eluate as "health physics poppycock." We would agree that this point has been overemphasized but the above statement is true only if patient dose is not a consideration. Also, a beginning technologist in the field of nuclear medicine should not be prejudiced with a casual attitude regarding any radiation dose. The discussion of dosimetry in Chapter 6 is too limited even for a technologist early in training. A brief discussion of the absorbed fraction method would also be useful as this concept seems to be gaining wide acceptance. Figure 6–2 is a faulty illustration of the relation between half life and mean life. Not included in Chapter 6, but desirable, would have been a list and short discussion of factors that reduce patient dose.

The chapters on biologic effects of radiation and health safety give the technologist a good view of the subject. In Chapter 8, the explanations of amplifier gain and attenuation are awkward and tortuous. Since these are very important concepts, failure to present them clearly in a manner easily remembered raises a major criticism. Figure 8–12 illustrating the effect of a time constant on the response of a count rate meter could only be true if time is reversible. While illustrations consisting of photographs of imaging devices have some informational content, line drawings demonstrating how they operate are to be preferred.

A chapter on considerations of counting and imaging, such as Chapter 9, is an excellent idea. In general the explanations are good except for the discussion of stationary imaging techniques. Statements such as "the size of the dot on the image is directly dependent upon the number of counts to be accumulated" is simply not true. Considering "information density" as an absolute and not in its relation to count rate and other parameters is misleading.

We were pleased to see a glossary included which could prove of great benefit to the technologist. The definitions for cumulative dose, daughter, gas amplification, Geiger region, linear amplifier and personnel monitoring need revision or future explanation.

Part II of the text is titled "Clinical nuclear medicine." This gives the technologist an outline on the basic physiology, pathology and nuclear medicine principle involved in the various tests. The illustrations in general are good and the nuclear medicine principles clearly outlined. For a text aimed at technologists inclusion of a short discussion of how to perform

each diagnostic procedure would be invaluable. A concise, if one will pardon the trite expression, "cookbook" approach outlining the standard method (with usual variations mentioned) used to perform each study would add tremendously to the usefulness of the text. Since many nuclear medicine laboratories will prefer to use a single reference as a guideline for the technologists, a complete text on the subject will contain this type of information even though it must, of necessity, be periodically updated.

In summary, a text of this type is needed. At present this is the only current one which attempts to address the nuclear medicine technologist. Unfortunately, the faults in certain critical portions of this text are rather numerous.

A. Everette James, Jr., M.D.
Edward U. Buddemeyer, Ph.D.
James K. Langan, R. T.

Electrical Impedance Plethysmography. The Electrical Resistive Measure of the Blood Pulse Volume, Peripheral and Central Blood Flow. Second edition. By Jan Nyboer, D.Sc., M.D., Director of Research, Rehabilitation Institute, Department of Physical Medicine, Wayne State University, Detroit, Mich.; Professor, Department of Physiology and Pharmacology, Wayne State University Medical School, Detroit, Mich.; Former Chief, Division Cardiovascular Physiology Research; Electrocardiographer and Associate Physician, Department of Medicine, Harper Hospital, Detroit, Mich. Cloth. Pp. 390, with some figures. Price, $22.00. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1970.

This monograph on Bioelectrical Impedance methodology uniquely involves techniques for studying vascular and respiratory dynamics. It demonstrates minute and gross plethoric phenomena and thoroughly justifies the role of measuring electrical impedance of the body segments and fluids. It is applicable in legions of conditions found in man and animals diagnostically and produced therapeutically.

Contrast studies of bilateral respiratory function and pulmonary vascular pulsations are made by roentgendensitometry and electrical impedance plethysmography with an excellent degree of correlation. Although the chest is diffusely dense to roentgen rays, pulsatile function is still detectable from residual lung and blood tissue by impedance. Impedance signals may also be used in children to trigger equipments at any phase of respiration defined by impedance.

The theory is well-supported that parallel mechanical changes in volume of the segment are associated with parallel changes in resistance improving or decreasing electrical conductance of the part. The pulse produces about 0.1 of 1 per cent changes in resistance. Thus, based on the laws of Ohm and Kirchoff, one may calculate or instruct a computer to derive pulsatile blood volume or volume flow indices from physical and geometric parameters of a given segment.

The author highlights impedance plethysmography as a multi-disciplinary physical technique applicable to monitoring non-invasively the heart, the vasculature of the kidneys, the brain and the lungs as well as the extremities of a subject. It is of interest to researchers in biology and medicine as well as clinicians demanding functional definition within the roentgen field. References 259, 260 and 261 from our Journal indicate its relevance to interests in radiology.

The format of the monograph is elegant, the printing is in latest style and the illustrations are beautifully reproduced. The composition is of the highest quality and most artistically conceived. As in the First Edition, there are four main chapters, of which the third, devoted to "Applications in Electrical Plethysmography," has been greatly expanded. Correspondingly, the number of references has been increased from 225 to 455. As Dr. Franklin D. Johnston states in the Foreword to the Second Edition, this is not surprising, because "the field involved in impedance studies is almost unlimited, and since the technique is painless and without risk to patients, the method is almost certain to be increasingly used in the future."

Traian Leucutia, M.D.

BOOKS RECEIVED

Tumours of the Testicle. By John P. Blandy, D.M., M.Ch., F.R.C.S., Professor of Urology, London Hospital Medical College; Consultant Urological Surgeon, London Hospital and St. Peter's, St. Paul's, and St. Philip's Hospitals, London; H. F. Hope-Stone, M.B., B.S., D.M.R.T., F.F.R., Consultant Radiotherapist, the London Hospital; and Anthony D. Dayan, B.Sc., M.D., M.R.C.P., M.R.C.Path., Consultant Pathologist, the Hospital for Sick Children, Great Ormond

Street, and the National Hospital, Queen Square, London, England. Cloth. Pp. 199, with many illustrations. Price, $7.00. Grune & Stratton, Inc., 757 Third Avenue, New York, N. Y. 10017, 1970.

ATLAS OF ROENTGEN ANATOMY OF THE SKULL. Revised third printing. By Lewis E. Etter, B.S., M.D., F.A.C.R., Professor of Radiology, Falk Clinic and Western Psychiatric Institute and Clinic; Presbyterian-University Hospital; School of Medicine, University of Pittsburgh, Pittsburgh, Pa. With a section on "The Radiographic Anatomy of the Temporal Bone" by J. Brown Farrier, M.D., F.A.C.S., Farrier Clinic, Tampa, Fla., and a section on "The Roentgen Anatomy of the Skull in the Newborn Infant" by Samuel G. Henderson, M.D., F.A.C.R., and Louise S. Sherman, B.S., M.D., School of Medicine, University of Pittsburgh, Pittsburgh, Pa. Cloth. Pp. 215, with many illustrations. Price, $24.75. Charles C Thomas Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1970.

DECONTAMINATION OF NUCLEAR REACTORS AND EQUIPMENT. Edited by J. A. Ayres, Ph.D., Pacific Northwest Laboratory, Battelle Memorial Institute. Prepared under the auspices of the United States Atomic Energy Commission. Cloth. Pp. 825, with many figures. Price, $22.50. The Ronald Press Company, 79 Madison Avenue, New York, N. Y. 10016, 1970.

RANDOM NOISE TECHNIQUES IN NUCLEAR REACTOR SYSTEMS. By Robert E. Uhrig, Ph.D., University of Florida. Prepared under the auspices of the United States Atomic Energy Commission. Cloth. Pp. 490, with some figures. Price, $20.00. The Ronald Press Company, 79 Madison Avenue, New York, N. Y. 10016, 1970.

ECOLOGY OF THE CANCER PATIENT. Proceedings of Three Interdisciplinary Conferences on Rehabilitation of the Patient with Cancer: The Head and Neck, Princeton, N. J., Feb. 12–15, 1967; The Breast, Extremities, and Nervous System, Princeton, N. J., April 21–24, 1968; The Pelvic Area, Elkridge, Md., April 20–23, 1969. Edited by John E. Healey, Jr., M.D. Paper. Pp. 184. Price, $2.00. Interdisciplinary Communication Associates, Inc., 1353 28th Street, N.W., Washington, D. C. 20007, 1970.

INTRAMEDULLARY NAILING AFTER REAMING: an Investigation on the Healing Process in Osteotomized Rabbit Tibias. By Göran Danckwardt-Lilliestrom, Gian Lorenzo Lorenzi, and Sven Olerud, Departments of Surgery and Orthopedics, University Hospital, Uppsala, Sweden. Paper. Pp. 78, with many illustrations. Acta Orthopaedica Scandinavica, Supplementum No. 134. Munksgaard, Copenhagen, 1970.

PREVENTION OF CONGENITAL DISLOCATION OF THE HIP JOINT IN SWEDEN: Efficiency of Early Diagnosis and Treatment. A symposium held at the annual meeting of the Swedish Orthopaedic Association in Stockholm, April 5, 1967. Paper. Pp. 64, with many illustrations. Acta Orthopaedica Scandinavica, Supplementum No. 130. Munksgaard, Copenhagen, 1970.

STATEMENT OF OWNERSHIP, MANAGEMENT AND CIRCULATION

(Act of October 23, 1962: Section 4369, Title 39, United States Code)

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NAME	ADDRESS
The American Roentgen Ray Society,	A non-profit organization incorporated in the
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Dr. Ted F. Leigh, Secretary	Emory Univ. Clinic, Atlanta, Georgia 30322
Dr. Paul Riemenschneider, Chairman	Publication Committee-Cottage Hosp., Santa Barbara, CA

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I certify that the statements made by me above are correct and complete.

(Signature of editor, publisher, business manager, or owner)

Paul A. Riemenschneider

POD Form 3526 May 1968

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual meeting: Deauville Hotel, Miami Beach, Fla. September 29–October 2, 1970.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Jerome M. Vaeth, Saroni Tumor Institute, 1600 Divisadero St., San Francisco, Calif. 94115. Annual meeting: Mexico City, Mexico, March 15-18, 1971.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary-Treasurer, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., November 29–December 4, 1970.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: St. Louis, Mo., Chase-Park Hotel, March 30–April 3, 1971.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga., 30322. Annual meeting: Atlantic City, N. J., June 20–24, 1971.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Denver, Colo., Dec. 7–11, 1970, Denver-Hilton Hotel; Bal Harbour, Fla., June 7–11, 1971, Americana Hotel; and Dallas, Tex., Dec. 6–10, 1971, Statler-Hilton Hotel.

Written examinations are scheduled in June of each year in 12 large centers, and applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Beginning in 1970 the deadline for receiving applications for any examination is Sept. 30 of the year preceding.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, Leonard Stanton, Hahnemann Medical College, 230 N. Broad St., Philadelphia, Pa. 19102. Annual meeting to be announced.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Carl R. Bogardus, Jr., University of Oklahoma Medical Center, Oklahoma City, Oklahoma 73104 Fall meeting: Mountain Shadows, Scottsdale (Phoenix), Ariz., November 11-13, 1970.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, F. J. Fry, M.Sc., Bioacoustics Lab., University of Illinois, Urbana, Ill.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary-Treasurer, Dr. Eugene V. Leslie, Edward J. Meyer Memorial Hospital, 462 Grider St., Buffalo, N. Y. 14215.

THIRTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Meeting: Madrid, Spain, Oct. 13–19, 1973.

TENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Manuel Viamonte, Jr., University of Miami School of Medicine, Jackson Memorial Hospital, Miami Fla. 33136.
President, Dr. Victor A. Marcial, Puerto Rico Nuclear Center, Caparra Heights Station, San Juan, Puerto Rico 00935.
Meeting: San Jeronimo-Hilton Hotel, San Juan, Puerto Rico, May 16-22, 1971.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.

ALABAMA CHAPTER OF ACR
Secretary, Dr. William V. Weldon, Medical Arts Building, Birmingham, Ala. 35205. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY
Secretary, Dr. Bruce C. Wright, Providence Hospital, Anchorage, Alaska. Meets third Wednesday each month.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Wesley S. Fee, 2421 E. 6th St., Tucson, Ariz. 85719. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. Wilma C. Diner, Univ. of Arkansas Medical Center, Little Rock, Ark. 72201. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Elliott C. Lasser, University Hospital of San Diego County, San Diego, Calif. 92103. Annual Meeting: Durham, N. C., May 13–15, 1971, with the Duke University and University of North Carolina Radiology Departments serving, as co-hosts.

ATLANTA RADIOLOGICAL SOCIETY
Secretary, Dr. Richard S. Colvin, Emory University Clinic, Atlanta, Ga. 30322. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Advisor, Colonel Paul E. Sieber. *Secretary*, LTC Peter B. Riesz, USAH Bad Cannstatt, APO 09154, New York, N. Y. Meets quarterly.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. R. John Gould, 41 Lombardy Rd., Drexel Hill, Pa. 19026.

BLUEGRASS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Joseph A. Sayeg, Ph.D., Radiation Physicist, University of Kentucky, Lexington, Ky. 40506. The Society meets once each month during the school year.

BRONX RADIOLOGICAL SOCIETY, NEW YORK STATE, CHAPTER ACR
Secretary-Treasurer, Dr. David Bruce Hayt, 600 E. 233rd St., Bronx, N. Y. 10466. Meets 4 times a year.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Rubem Pochaczevsky, 300 Skillman Ave., Brooklyn, N.Y. 11211. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Glen M. Ebersole, 405 Spring St., Jamestown, N.Y. 14701. Meets second Monday evening each month, October to May inclusive, at University Club.

CALIFORNIA RADIOLOGICAL SOCIETY, CALIFORNIA CHAPTER of ACR
Secretary-Treasurer, Dr. John L. Gwinn, Childrens Hospital of Los Angeles, P.O. Box 54700, Los Angeles, Calif. 90054.

CATAWBA VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Emmett R. White, P. O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 P.M.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Central New York Radiological Society
Secretary-Treasurer, Dr. David N. Cheris, Community General Hospital of Greater Syracuse, Broad Road, Syracuse, N. Y. 13215. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary, Dr. James V. Blazek, 2586 Lane Rd., Columbus, Ohio 43220. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Chicago Roentgen Society
Secretary-Treasurer, Dr. William T. Moss, 250 E. Superior St., Chicago, Ill. 60611. Meets third Thursday of each month, October to April, except December, at the Bismarck Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. James Farmer, 18869 Canyon Rd. Fairview Park, Ohio 44126. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Marvin L. Daves, Univ. of Colorado Medical Center, 4200 E. Ninth Ave., Denver, Colo. 80220. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. V. V. Kirby, 1722 Spring Lake Dr., Arlington, Tex. 76010. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. James H. Taylor, Wilmington Medical Center, Wilmington, Del. 19899.

East Bay Radiological Society
Secretary-Treasurer, Dr. S. Walter Kran, Doctors' Hospital of San Leandro, 13855 East 14th St., San Leandro, Calif. 94578. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. Eberhard F. Besemann, Baroness Erlanger Hospital, Chattanooga, Tenn. 37403. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary, Dr. Wm. F. Lindsey, 1215 Hodges Dr., Tallahassee, Fla. 32303. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Allen L. Sheer, University Community Hospital, 13505 N. 31st St., Tampa, Fla. 33612. Meets in January, March, May, July, September and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. Walker Harris, The Medical Center, Columbus, Ga. 31902. Meets in spring and fall at Annual State Society Meeting.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. John Kathe, North Shore Hospital, Miami, Fla. 33150. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. Roland P. Ernst, 3720 Washington Ave., St. Louis, Mo. 63108.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Virgil R. Jobe, Jr., 888 South King St., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, John H. Pingel, Argonne National Laboratory, 9700 S. Cass Ave., Argonne, Ill. 60439. Annual Meeting: Waldorf Astoria Hotel, New York City, July 11–15, 1971.

Houston Radiological Society
Secretary, Dr. Kenneth M. Jensen, 1615 St. Joseph Prof. Bldg., Houston, Texas 77002. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025

Idaho State Radiological Society, Chapter of ACR
Secretary, Dr. Hugh P. Smith, Jr., 130 E. Bannock, Boise, Id. 83702. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. Jack L. Melamed, 1230 Sunset Rd. Winnetka, Ill. 60093. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. Dale B. Parshall, Elkhart General Hospital, P.O. Box 1329, Elkhart, Ind. 46514.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Wm. R. Allen, 155 S. 18th St. Kansas City, Kan. 66102. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Joan R. Hale, 402 Heyburn Bldg. Louisville, Ky. 40202. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N.Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Harold L. Atkins, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Harry T. Vanley, St. Mary's Long Beach Hospital, Long Beach, Calif. 90083. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Annual Midwinter Conference: Jan 30–31, 1971.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. Nathan Stofberg, 4519 Hawksbury Rd., Pikesville, Md. 21208.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Norman L. Siadowsky, The Faulkner Hosp., 1153 Centre St., Jamaica Plain, Mass. 02130

Memphis Roentgen Society
Secretary-Treasurer, Dr. Webster Riggs, Jr., The University of Tennessee College of Medicine, Department of Radiology, Walter F. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Ind. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary, Dr. David P. Corbett, Harper Hospital, De-

troit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary-Treasurer, Dr. James E. Bell, 8700 W. Wisconsin Ave., Milwaukee, Wis. 53213. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Warren L. Kump, 4243 Glenwood Ave., Minneapolis, Minn. 55422. Meets twice annually, fall and winter.

MISSISSIPPI STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ottis G. Ball, 5356 Balmoral Drive, Jackson, Miss. 39211. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

MISSOURI RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Arthur A. Porporis, 100 N. Euclid Ave., St. Louis, Mo. 63108.

MONTANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Jon A. Anderson, Doctor's Building, 1231 N. 29th Street, Billings, Mont. 59101.

NEBRASKA CHAPTER OF ACR
Secretary-Treasurer, Dr. Gordon F. Johnson, 4239 Farnam, Omaha, Neb. 68131. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEVADA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Harris W. Knudson, 2020 W. Charleston Blvd., Las Vegas, Nev. 89102.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Stefan C. Schatzki, 1180 Beacon St., Brookline Mass. 02146. Meets third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY, CHAPTER OF ACR
Secretary, George Farmlett, 33 Round Bay Rd., Keene, N. H. 03246. Meets four to six times yearly.

NEW MEXICO SOCIETY OF RADIOLOGISTS CHAPTER OF ACR
Secretary, Dr. Donald A. Wolfel, Albuquerque, New Mexico. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

NEW YORK ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Samuel H. Madell, 1. E. 82nd St., New York, N. Y. 10028. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference: Waldorf Astoria Hotel, New York, April 29–May 1, 1971. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

NEW YORK STATE CHAPTER OF ACR
Secretary-Treasurer, Dr. John J. Magovern, 520 Franklin Ave., Garden City, N. Y. 11530.

NORTH CAROLINA CHAPTER OF ACR.
Secretary-Treasurer, Dr. James F. Martin, 300 S. Hawthorne Road, Winston-Salem, N. C. 27103. Fall Meeting, Mid Pines Hotel, Southern Pines, N. C., Oct. 31, 1970, 2 P.M.

NORTH DAKOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Marshall Landa, 1702 13th St., So., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

NORTH FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. John W. Morris, III., Department of Radiology, Halifax District Hospital, Daytona Beach, Fla. 32015. Meets quarterly in March, June, September and December.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Barbara Chick, Glens Falls Hospital, Glens Falls, N.Y. 12801. Meets in Albany area on third Wednesday of October, November, March, April, and May.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Kevin Ryan, Woodland Medical group, Woodland, Calif. 95695. Meets fourth Monday of Sept., Nov., Jan., March and May at Aldo's Restaurant in Sacramento.

NORTHWESTERN OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

OHIO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Joseph Hanson, 1544 South Byrne Road, Toledo, Ohio 43614.

OKLAHOMA STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Richard B. Price, 204 Medical Tower Bldg., Oklahoma City, Okla. 73112. Meets in January, May and October.

ORANGE COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Roderick A. Silveira, 100 E. Valencia Mesa Dr., Fullerton, Calif. 92632. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at the Orange County Medical Association Bldg., Orange, Calif.

OREGON STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Gerald L. Warnock, 11699 E. Glisan St., Portland, Ore. 97220. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd.; Beaverton, Oregon 97005. Meets annually in Portland, Oregon, Seattle, Washington or Victoria or Vancouver, British Columbia, in early May.

PENNSYLVANIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Theodore A. Tristan, Harrisburg Polyclinic Hosp., Harrisburg, Pa. 17105.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. Norman Williams, Department of Radiotherapy, Montefiore Hospital, 3459 Fifth Ave., Pittsburgh, Pa. 15213. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

RADIATION RESEARCH SOCIETY
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016.

RADIOLOGICAL SOCIETY OF CONNECTICUT, INC., CHAPTER OF ACR
Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meetings are held quarterly

RADIOLOGICAL SOCIETY OF GREATER CINCINNATI
Secretary-Treasurer, Dr. Donald E. Gunderson, 3553 Bayard Dr., Cincinnati, Ohio 45208. Meets first Monday of each month at Cincinnati Academy of Medicine.

RADIOLOGICAL SOCIETY OF GREATER KANSAS CITY
Secretary, Dr. Ken C. Davidson, St. Luke's Hospital of Kansas City, Kansas City, Mo. 84111. Meets 5 times a year on given dates.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

RADIOLOGICAL SOCIETY OF LOUISIANA, CHAPTER OF ACR
Secretary, Dr. Ralph B. Bergerson, 154 Brockenbraugh Ct. Metairie, La. 70005. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

RADIOLOGICAL SOCIETY OF NEW JERSEY, CHAPTER OF ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic

City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. John J. O'Brien, 292 Merrymount Dr., Warwick, R.I. 02888.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Gladden V. Elliott, 5565 Grossmont Center Dr., Suite 1, La Mesa, Calif. 92041. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. M. Pinson Neal, Jr., Medical College of Virginia, 1200 E. Broad St., Richmond, Va. 23219. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Kenneth E. Robinson, Rochester General Hospital, 1425 Portland Ave., Rochester, N. Y. 14621. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo. Aug. 20–22, 1970.

San Antonio-Civilian-Military Radiological Society
Secretary, Dr. Lee F. Rogers, Department of Radiotherapy, Bexar County Teaching Hospital, 4502 Medical Drive, San Antonio, Texas. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

San Diego Radiological Society
President-Secretary, Dr. Q. H. Lehmann, 5565 Grossmont Center Dr. Suite 1, La Mesa, Calif. 92041. Meets first Wednesday of each month at the Town & Country Hotel.

San Francisco Radiological Society
Secretary-Treasurer, Dr. Warren M. Russel, Franklin Hospital, Castro & Duboce, San Francisco, Calif. 94114. Meets quarterly at various hospitals (contact Secretary).

Santa Clara County Radiological Society
Secretary, Dr. Emory G. West, 285 S. Drive, Mt. View, Calif. 94040. Meets monthly at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Southern Medical Association
Secretary, Dr. Phillip W. Voltz, Jr., 120 Medical Professional Bldg., San Antonio, Tex. 78212. Annual Meeting: Dallas, Tex., Nov. 16–19, 1970.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles 27, Calif. Annual meeting: Deauville Hotel, Miami Beach, Fla., September 28, 1970.

Society of Nuclear Medicine
Secretary, Dr. James J. Smith, 140 E. 54th St., New York, N. Y. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting: Los Angeles, Calif., June 26–July 2, 1971.

South Bay Radiological Society
Secretary, Dr. Emerson C. Curtis, University Dr., Menlo Park, Calif. 94025. Meets second Wednesday of each month.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Haakon O. Haugan, 716 Quincy St., Rapid City, S. D. 57701. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Aaron G. Fingerhut, 1000 W. Carson St., Torrance, Calif. 90502. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Annual meeting: Grand Hotel, Pointe Clear, Ala. Jan. 29-31, 1971.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Lawrence R. Nickell, Maury County Hospital, Columbia, Tenn. 38401. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Herman C. Sehested, 815 Medical Tower, Room 100, 1550 W. Rosedale St., Fort Worth, Tex. 76104. Annual meeting at the Flagship Hotel on Pier, Galveston, Tex.

The Fleichner Society
Secretary-Treasurer, Eric N. C. Milne, M.B., Medical Sciences Bldg., University of Toronto, Ontario, Canada. Meets in Williamsburg, Va., March 1971, in conjunction with a course on "Modern Trends in Roentgenology of the Chest," sponsored by the Virginia Commonwealth University, Richmond, Va.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Leon M. Neal, St. Benedict's Hospital, 3000 Polk Ave., Ogden, Utah 84403. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. Edward A. Kupic, Mary Fletcher Hosp., Burlington, Vt. 05401.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. James S. Redmond, Suite 7, Medical Center, Lynchburg, Va. 24501.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Joan Wohlgemuth, 5021 Seminary Rd., Alexandria, Va. 22311.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Paul S. Paulson, 1001 Broadway Seattle, Washington 98122. Meets quarterly.

West Virginia Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. J. Dennis Kugel, 510-517 Med. Arts Bldg. Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

WESTCHESTER COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Edgar Palmer, 650 Main St., New Rochelle, N. Y. 10801. Meets on third Tuesday of January and October and on two other dates.

WISCONSIN RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert E. Douglas, 1209 S. Commercial St., Neenah, Wis. 54956. Meets twice a year, May and September.

WYOMING RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. J. D. Grant, Memorial Hosp., Sheridan, Wyo. Meets in fall with State Medical Society and in spring on call of President.

MEXICO, PUERTO RICO AND CENTRAL AMERICA

ASOCIACIÓN COSTARRICENSE DE RADIOLOGÍA
Secretary, Dr. Jorge Vargas Segura, Apartado 5367, San José, Costa Rica.

ASOCIACIÓN DE RADIÓLOGOS DE CENTRO AMERICA Y PANAMÁ. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Roberto Calderón, Calle Central Oeste No. 218, Managua, Nicaragua, Central America. Meets annually in a rotating manner in the six countries.

ASOCIACIÓN PUERTORRIQUEÑA DE RADIOLOGÍA
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

SOCIEDAD DE RADIOLOGÍA DE EL SALVADOR
Secretary, Dr. Julio Astacio, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

SOCIEDAD DE RADIOLOGÍA DE GUATEMALA
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

SOCIEDAD MEXICANA DE RADIOLOGÍA, A.C.
Coahuila No. 35, México 7, D.F.
Secretary-General, Dr. Bernardo Serviansky. Meets first Monday of each month.

SOCIEDAD RADIOLÓGICA PANAMEÑA
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

SOCIEDAD RADIOLÓGICA DE PUERTO RICO
Secretary, Dr. Heriberto Pagán Sáez, Apt. 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

BRITISH COMMONWEALTH OF NATIONS

ASSOCIATION OF RADIOLOGISTS OF THE PROVINCE OF QUEBEC
Secretary, Dr. Pierre Archambault, Hôpital Charle Le Moyne, 121 Boul. Taschereau, Greenfield Park, P.Q., Canada. Meets four times a year.

BRITISH INSTITUTE OF RADIOLOGY
Honorary Secretary, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.

CANADIAN ASSOCIATION OF PHYSICISTS, DIVISION OF MEDICAL AND BIOLOGICAL PHYSICS.
Honorary Secretary Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada. Annual Congress, to be announced.

EDMONTON AND DISTRICT RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

FACULTY OF RADIOLOGISTS
Honorary Secretary, Robert Morrison, 47 Lincoln's Inn Fields, London, W.C.2, England.

FACULTY OF RADIOLOGISTS, ROYAL COLLEGE OF SURGEONS IN IRELAND
Registrar, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

CANADIAN ASSOCIATION OF RADIOLOGISTS
Honorary Secretary, Dr. F. Robert MacDonald, *Associate Honorary Secretary*, Dr. Champlain Charest, 1555 Summerhill Ave., Montreal 25, Que., Canada. Annual meeting: Calgary, Alberta, March 15–19, 1971.

MONTREAL RADIOLOGICAL STUDY CLUB
Secretary, Dr. Leonard Rosenthall, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

SECTION OF RADIOLOGY, CANADIAN MEDICAL ASSOCIATION
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

SOCIÉTÉ CANADIENNE-FRANÇAISE DE RADIOLOGIE
Secretary General, Dr. Guy Duckett, 1385 est, rue Jean Talon, Montréal, P.Q., Canada. Meets every third Tuesday from October to April. Annual meeting: Montreal, P.Q., Canada, November 5–7, 1970.

TORONTO RADIOLOGICAL SOCIETY
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September, through May.

COLLEGE OF RADIOLOGISTS OF AUSTRALASIA
Honorary Secretary, Dr. T. P. Loneragan, 147 Macquarie St., Sydney, N.S.W., Australia.

SOUTH AMERICA

ASOCIACIÓN ARGENTINA DE RADIOLOGÍA
Secretary, Dr. Lidio G. Mosca, Avda. Fral. Paz 151, Córdoba, Argentina. Meetings held monthly.

ATENEO DE RADIOLOGIA
Secretary, Dr. Víctor A. Añaños, Instituto de Radiologia, Urqiza 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional de Centenario, Santa Fe 1300, Rosario.

COLÉGIO BRASILEIRO DE RADIOLOGIA
Secretary-General, Dr. Miguel Mario, Céntola, Caixa Postal 5984, São Paulo, Brazil.

SOCIEDAD ARGENTINA DE RADIOLOGÍA
Secretary-General, Dr. Juan R. Heilbuth Pacheco, Santa Fe 1171, Buenos Aires. Meets first Wednesday evening, April through December.

SOCIEDAD BOLIVIANA DE RADIOLOGÍA
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

SOCIEDADE BRASILEIRA DE RADIOLOGIA
Secretary, Dr. Armando Rocha Amoédo, Cxa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

SOCIEDADE BRASILEIRA DE RADIOTERAPIA
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigaderio Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

SOCIEDAD CHILENA DE RADIOLOGÍA
Secretary, Dr. Carlos Vildósola, Casilla, 13426 Santiago, Chile. Meets fourth Friday of each month.

SOCIEDAD COLOMBIANA DE RADIOLOGIA
Secretary-General, Dr. Raul Fernández Angula, Calle 43 No. 44-70 Barranquilla, Colombia. Meets last Thursday of each month.

SOCIEDAD EQUATORIANA DE RADIOLOGÍA
Secretary, Dr. Carlos Palau Jimenez, Casilla 4569, Guayaquil, Ecuador.

SOCIEDAD PARAGUAYA DE RADIOLOGÍA
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

SOCIEDAD PERUANA DE RADIOLOGIA
Secretary-General, Dr. Julio Ormeño del Pino, Instituto de Radiología "Cayetano Heredia" Hospital Arzobispo

Loayza, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41–110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología de la Plata
Secretary. Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Alejandro Glausiuss, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Secretary, Dr. Mario A. Moya, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Modesto Rivero González, Apartado No. 9213 Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Second Congress of the European Association of Radiology.
President, Professor Dr. J. R. von Ronnen, State University of Leiden, The Netherlands.
Secretariat, c/o Holland Organizing Centre, 16 Lange Voorhout, The Hague, The Netherlands. Congress Meeting: Amsterdam, The Netherlands, June 14–18, 1971.

Österreichische Rontgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
President, Dr. George Thomsen, Rigshospitalet (University Hospital), Blegdamsvej 9, DK 2100 Copenhagen, Denmark. Meets in Elseneur, Denmark, May 12–15, 1971.
Permanent Secretary, Clément Fauré, Hôpital des Enfants-Malades, 149, rue de Sèvres, Paris 15e, France.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru Paris 8e, France.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Rontgengesellschaft
President, Professor Dr. med. Hanno Poppe, Radiologische Univ.-Klinik, Gossler Str. 10, 34 Göttingen, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano Torino, Italy. Meets annually.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. C. B. A. J. Puijlaert, Prof. Dondersstraat 73, Tilburg, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. E. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägen 5 C., Finland.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. D. José Bonnati, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Schweizerische Gesellschaft Für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. L. R. Parthasarathy 44, Constable Road, Madras—23, India.

Indonesian Radiological Society
Secretary, Professor Sjahriar Rasad, Taman Tjut Mutiah I Diakarta, Indonesia

Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Africa

Association of Radiologists of West Africa
Honorary Secretary, Dr. S. B. Lagundoza, University College Hospital, Ibadan, Nigeria.

South African International radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa. Second Congress: President Hotel, Johannesburg, September 3-9, 1970.

ABSTRACTS OF RADIOLOGICAL LITERATURE

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ROENTGEN DIAGNOSIS

Head

Picard, L., Renard, M., and Montaut, J. Aspects radio-anatomiques des ruptures de la carotide intracrânienne. (Radiologic-anatomic aspects of rupture of the intracranial portion of the carotid artery.) *J. de radiol., d'électrol. et de méd. nucléaire*, May, 1970, *51*, 217–222. (From: Clinique Neuro-Chirurgicale et Clinique Neurologique, C.H.U. Nancy, France.)

Traumatic rupture of the siphon of the internal carotid artery results in the formation of false aneurysms and of cavernous sinus fistulas. The exact mechanism of the physiopathology of the development of the carotid cavernous fistula is not clearly understood. It is still to be determined whether it is due to a tear, traumatic or spontaneous, which has occurred at the site of a pre-existing lesion in the arterial wall, or whether it is due to a re-opening of an arteriovenous communication between arterioles arising from the internal carotid artery and the venules of the cavernous plexus, secondary to a previous trauma.

In the 6 cases which they report, the authors have endeavoured to demonstrate the exact site of the rupture of the wall of the carotid artery. This has been possible through the technical advances made in angiography utilizing a rapid cassette changer at 6 to 8 exposures per second.

The cases reported include 2 false aneurysms, 3 carotid cavernous sinus fistulas and 1 "dry" carotid artery rupture.

The roentgenographic examinations revealed the extent and location of multiple fractures at the base of the skull and the angiographic studies demonstrated the exact location and the appearance of the vascular lesion. These were confirmed at autopsy in 5 of the 6 cases.

The false aneurysm develops from a rupture at the anterior convexity, while the cavernous sinus fistula develops from a rupture of the posterior convexity of the carotid siphon.

In Case v the fistula originated from the internal carotid artery in the subarachnoid space immediately outside the cavernous sinus.

Case vi is that of a "dry" rupture of the internal carotid artery with retrograde thrombosis. This case is a particularly striking and rare form of rupture of the carotid artery. A metallic projectile entered through the right orbit and emerged in the left parieto-occipital region. In its path it perforated the orbital roof, dislocated the sella and the anterior clinoid processes. Left carotid angiography demonstrated complete occlusion of the internal carotid artery in the neck, at the level of C2. Angiography of the contralateral side resulted in opacification of the right carotid-cerebral vessels and, following compression, in opacification of the cerebral branches of the left carotid artery. Both anterior cerebral arteries were displaced to the right, with crowding and lateral displacement of the sylvian group of vessels indicating deep intracerebral hemorrhage. Thrombosis of the left internal carotid artery, with retrograde occlusion may have resulted from an actual "cautery" by the metallic projectile.

Reproduction of roentgenograms using the subtraction technique, and 2 tables, accompany this article.—*William H. Shehadi, M.D.*

Hugh, A. E. Trickle arteriography: demonstration of thrombi in the origin of the internal carotid artery. *Brit. M. J.*, June 6, 1970, *2*, 574–576. (Address: Consultant Radiologist, North Staffordshire Hospital, Stoke-on-Trent, England.)

Thrombi developing within the origin of the internal carotid artery may be demonstrated by a slow trickle injection of highly concentrated contrast medium, which lingers in pools around the thrombus on the dependent wall of the vessel. With this technique, thrombi not visualized in conventional arteriography have been detected, because they were obscured by the density of the vascular shadow produced by the forceful injection usually employed in carotid angiography. In addition, trickle arteriography is useful for assessing the position and extent of atheromatous plaques and the contrast medium stasis associated with them.

Conventional arteriography is first performed. If the intracranial vessels are displayed satisfactorily, further injections are made. The technique is described in detail.

The trickle technique has several advantages in demonstrating intraarterial stasis and thrombi. In a healthy carotid artery, the dense contrast medium is seen to spread out along the dependent wall of the vessel but has almost completely cleared a few seconds later. Where pathologic stasis exists, possibly in relation to a plaque of atheroma, its persistence is noted on the roentgenograms taken in the next 8 to 10 seconds. Most striking advantage of the trickle technique is in the demonstration of intraarterial thrombi.

Occasionally irregular thrombi are outlined by a coating of contrast medium, which persists for a number of seconds on their surface with the trickle technique.

Complete mixing of the contrast medium occurs sometimes, and sleeve filling is not obtained. This is usually due to the needle tip being placed in the rapidly flowing blood in the center of the vessel, and can be overcome by advancing the position of the needle tip slightly.

Success of the technique depends on the following factor: the tendency of intravascular contrast

media to enter and to linger in the slowly-moving sleeve of fluid which comprises the boundary layer of flow in the vessel, since the origin of the internal carotid artery is usually dependent during carotid arteriography. Entry of contrast medium into the boundary layer of flow depends on both the nature of the medium and the mode of injection.

Slow speed of injection is also of vital importance. Forceful injections cause pronounced local turbulence and more thorough mixing of contrast media than do slower injections. Unless thorough mixing is immediate, gravitational layering will occur, with subsequent boundary-layer visualization. Further, injections made with the contrast jet directed at the vessel wall are more likely to produce boundary-layer filling. This explains why the needle is introduced in a near vertical direction rather than along the horizontal course of the artery.

The use of highly concentrated contrast media has been thought to be contraindicated in carotid angiography, for high concentrations are known to damage nervous tissue. If the injection is made slowly, however, the rate of iodine passage through the small vessels of the brain would be lower than in conventional techniques, and no damage should result. None of the 12 patients examined in this way have shown any adverse response. Over 50 patients have been examined by the techniques described, and no complications have been noted.—*Stephen N. Tager, M.D.*

Tlusty, T. Neonatal cranial malformations. *Proc. Roy. Soc. Med.*, May, 1970, *63*, 459–460. (From: St. James's Hospital, Balham, London SW 12, England.)

A 2,020 gram neonate is reported who was born with a marked Pierre-Robin type deformity of the lower part of the face, with no palpable mandible. The infant had respiratory distress and died shortly after birth.

Additional anomalies at postmortem examination were: marked hypoplasia and deformity of the right lung, a very small malformed mandible situated behind the esophagus and larynx, hemivertebral spinal deformities and left rib cage deformities.

The mandible was a small, single arcuate bone, attached to the cranial base by a formed articulation into the right pterygoid fossa and on the left side by a loose ligamental junction. Normally the mandible develops by a bilateral deposition of membranous bone to form a complete lower jaw at about 6 weeks of uterine life and the midline synosteotic junction occurs during the second year of extrauterine life. The case reported represents an arrest in development during intrauterine life with midline fusion occurring prior to delivery.

Also discussed is the subject of cranial lacunae. Twelve dry skull preparations were selected from several hundreds of neo- and perinatal postmortem cases which had a high incidence of congenital deformities. Some of these cases were associated with co-existent deformities of spina bifida or hydrocephalus, but other cases were found without association of spina bifida, meningocele or myelomeningocele. Apparently cranial lacunae can occur as an isolated malformation.—*Lionel W. Young, M.D.*

Neck and Chest

Dalquen, P., Dabbert, A.-F., and Hinz, I. The epidemiology of pleural mesothelioma: a preliminary report on 119 cases from the Hamburg area. *German Med. Monthly*, Feb., 1970, *15*, 89–95. (Address: Dr. P. Dalquen, Hebelstr. 24, 4056 Basel, Switzerland.)

Diffuse pleural mesothelioma is an uncommon malignant tumor derived from the cells which cover the pleural surface. The authors discuss the occurrence of pleural mesothelioma from an epidemiologic standpoint in the area of Hamburg.

A total of 119 cases of pleural mesothelioma (79 males and 40 females) were discovered in the Bergedorf district of Hamburg associated with an asbestos factory in that area. They point out the importance of a carefully taken occupational history, the problems of occupational, neighborhood or domestic exposure to asbestos, and the relatively high risk among shipyard workers and seamen.

The average latent period was 35.2 years.

In 17 cases there was definite asbestos exposure, in 38 patients it was incidental or merely probable, and in 64 cases asbestos exposure was uncertain, absent or not ascertainable.

Asbestos exposure is an unusually frequent item in the histories of patients with pleural mesothelioma and the remarkable fact is that the exposure may be very short (minimum 14 days) or so slight that it has not been enough to cause pulmonary asbestosis.

The problems revealed by the study are: (1) asbestos cancer should be defined as cancer following asbestos exposure and not necessarily associated with asbestosis; (2) in spite of the control of emission of asbestos dust from major factories, there is still a substantial and largely uncontrolled emission of dust from numerous small manufacturers and users of asbestos products; (3) the ubiquitous abrasion of materials which contain asbestos is a factor which must not be underestimated; and (4) the available data throw no light on the question of co-carcinogenesis.—*Luther W. Brady, M.D.*

Ferrané, J., Guermonprez, J. L., Vasile, N., and Maurice, P. Silhouette cardiaque anormale et tumeurs thymiques: à propos de 11 observations. (Abnormal cardiac silhouette and thymic tumors: a report on 11 patients.) *J. de radiol., d'électrol. et de méd. nucléaire*,

May, 1970, *51*, 207–216. (From: Clinique cardiologique, Hôpital Bouçicaut, Paris 15, France.)

Tumors of the thymus constitute approximately 50 per cent of tumors of the anterior mediastinum and 12–16 per cent of all mediastinal tumors.

Tumors of the thymus located in the lower mediastinum overshadow the heart and great vessels and, often, may be difficult to recognize or identify.

The authors report a series of 11 cases seen in the cardiac clinic between 1956 and 1967. There were 7 male and 4 female patients between the ages of 17 and 63 years. The examinations on each patient included routine clinical and laboratory studies, and roentgenologic examination in multiple projections. Angiography was performed in 7 cases.

Ten patients experienced dyspnea without exertion and 1 patient complained of precordial pain. Myasthenia gravis occurred in 1 patient only.

Auscultation revealed the presence of a mild systolic murmur in 1 patient; the remaining 10 were normal. On routine roentgenograms of the chest the appearance of the heart and mediastinum was consistent with cardiac enlargement in 1 patient, enlargement of the right antrum in 2 patients, dilatation of the descending aorta in 3 patients, mitralization of the heart in 1 patient, and pleuropericardiac cyst in 1 patient.

Roentgenograms made in multiple projections, including overexposed posteroanterior films, demonstrated the apparent abnormal configuration of the heart and great vessels to be due to soft tissue tumor masses extraneous to these structures; this was confirmed by angiography which showed the heart and great vessels to be normal. A careful study of the roentgenograms, especially those made in oblique and lateral projections, showed the density of the shadow of the tumor to be different from that of the cardiovascular silhouette. Furthermore, the tumor usually formed an acute angle with the heart and great vessels. These criteria should suggest that the tumor is most probably of thymic origin.

The final diagnosis can be made only by surgical exploration and a histologic examination of the tissues removed. This was done in all the cases reported by the authors. Of the 11 cases reported, the diagnosis of carcinoma of the thymus was made in 1 case and of thymoma in 10 cases.

Each case report is admirably illustrated with reproductions of several roentgenograms.—*William H. Shehadi, M.D.*

Misra, Kabi P., Hildner, Frank J., Cohen, Lawrence S., Narula, Onkar S., and Samet, Philip. Aneurysm of the membranous ventricular septum: a mechanism for spontaneous closure of ventricular septal defect. *New England J. Med.*, July, 1970, *283*, 58–61. (From: Division of Cardiology, Department of Internal Medicine, Mount Sinai Hospital of Greater Miami, Miami Beach, Fla.)

The report records 3 cases of aneurysm of the membranous ventricular septum in a 5 year old, 14 year old and a 22 year old patient. All have had previous catheterizations and angiographic studies without the aneurysms being evident. It is thus assumed that these aneurysms were acquired and not congenital in origin. This evidence is contrary to the usually accepted origin of the aneurysms which have been accepted by most authors as being congenital.

All the patients had small ventricular septal defects and small left-to-right shunts.

The authors draw attention to recent reports that aneurysm of the membranous septum may occur in association, and as a prelude to, closure of small ventricular septal defects. They point out that the mechanism is not definitely understood. The relationship of the aneurysm to the mechanism of closure of the ventricular septal defect is not adequately discussed and would be of some interest to your reviewer. It would also have been pertinent to the article to have seen illustrations of the ventricular septal area on the initial angiographic studies.—*Roger Pyle, M.D.*

Abdomen

Hughes, William, and Pierce, William S. Surgical implications of gastric diverticula. *Surg., Gynec. & Obst.*, July, 1970, *131*, 99–102. (From: Department of Medicine, Section of Gastroenterology and the Department of Surgery, Hospital of the University of Pennsylvania, Pittsburgh, Pa.)

The diverticula of the stomach are uncommon. An incidence of approximately 1 patient per 20,000 admissions has been recorded.

The authors report 23 cases in a 15 year period. Of these, 19 complained of pain in the upper portion of the abdomen; 2 patients had melena; 1 had hematemesis; and 1 had abdominal distention.

The diverticulum was discovered by roentgenographic means in 21 instances, by operation in 1 instance, and at autopsy in another.

In 21 cases the diverticula were located near the esophagogastric junction, while 2 were found at operation and at autopsy in the prepylorus.

There is considerable associated gastrointestinal pathology in patients with gastric diverticula and a thorough evaluation of the gastrointestinal tract is necessary before the symptoms can be attributed to a gastric diverticulum. In most cases, peptic ulcer disease, biliary tract disease, hiatal hernia or esophageal carcinoma are associated.

Frequently diverticula are located elsewhere in the gastrointestinal tract, as for example, in the colon. One patient had diverticulum in the esophagus, 1 in the stomach, 2 in the duodenum and 1 in the jejunum associated with multiple diverticula of the colon.

None of the patients had multiple gastric diverticula.

Perforation of the gastric diverticulum is exceedingly rare.

A gastric diverticulum is subject to diseases of the gastric mucosa including malignant lesions, hemorrhage, and inflammation.

Operation for a gastric diverticulum is to be recommended only if medical management is unsuccessful. —*David C. Alftine, M.D.*

Parks, T. G. Malignant carcinoid and adenocarcinoma of the stomach. *Brit. J. Surg.*, May, 1970, *57*, 377–379. (From: The Royal Victoria Hospital, Grosvenor Road, Belfast, Northern Ireland.)

Primary carcinoid tumors may arise in any part of the gastrointestinal tract from the cardia to the anus. They have been found in the gallbladder and in the ovarian and testicular teratomas.

A case is presented having an association of adenocarcinoma of the stomach and carcinoid tumor. Only 1 case of this type of association has been reported previously.

The report is supported by macroscopic and microscopic reproductions.—*David C. Alftine, M.D.*

Sherbon, K. J. The radiology of ischemic colitis. *Australasian Radiol.*, Feb., 1970, *14*, 46–55. (Address: Staff Radiologist, Royal Prince Alfred Hospital, Sydney, Australia.)

The roentgenologic findings in ischemic colitis are described and illustrated.

The plain abdominal roentgenographic findings are gas in the bowel wall, "thumb-printing," and thickening of the bowel wall. Although gas in the bowel wall is not frequently seen, it is the most helpful sign in the plain roentgenograms.

Early in the disease the barium enema examination shows a narrowed segment and marginal defects due to submucosal hemorrhages. In later stages, fine mucosal ulcerations and deep transverse indentations are observed. Even later, smooth tapered strictures and sacculations may develop.

Arteriography is disappointing and has shown poor correlation in this disease. Serial barium enema studies, demonstrating the progressive changes in the bowel in different stages, are the most important method in ischemic colitis.—*John H. Harris, M.D.*

Biolcati, R. A., and Cardona, P. L'impiego della metodica perfusionale nella pratica radio-diagnostica delle vie biliari. (The use of the perfusion method in the radio-diagnostic practice of the biliary paths.) *Ann. radiol. diagn.*, 1970, *42*, 369–381. (From: Istituto di Radiologia dell'Arcispedale Sant' Anna di Ferrara, Italy.)

A new contrast medium for intravenous potentiated cholangiography, SH 20977 or biligram, has been used in 25 patients chosen at random.

Biligram is a salt of methylglumine of the diglycolic acid; its iodine content is 8 gm., compared with 5.5 gm. of bilivistan; and comes already diluted in 100 cc. of a hypertonic glucose solution.

From an analysis of the results obtained, the opacification of the extrabiliary system was excellent in 3 cases, good in 10, moderate in 8, and poor or nil in 2 cases each. Collateral reactions were minimal and noted only in 6 cases. Vicarious renal excretion, from moderate to minimal, was noted in 15 cases. The time of infusion was 15 minutes, from an initial time of 30 minutes. The first cholangiogram was taken 60 minutes from the end of the infusion, and 30 minutes in those patients who presented a history of cholecystectomy (13 cases).

In concluding, the authors are of the opinion that biligram by infusion presents definite advantages over bilivistan in the demonstration of the gallbladder and/or the extrabiliary ducts, and a better tolerance to the contrast medium, most likely because of its dilution in a glucose solution.—*A. F. Govoni, M.D.*

Wangermez, A., Wangermez, J., and Debot, P. (Bordeaux, France.) Les risques de la cholangiographie-perfusion et les dangers des examens biliaires itératifs. (The risks of infusion cholangiography and the dangers of repeated examinations of the biliary tract.) *J. de radiol., d'électrol. et de méd. nucléaire*, May, 1970, *51*, 287–290.

The authors warn against the initial overenthusiasm in reporting the results of new techniques and procedures. There may be a tendency, in good faith, to overemphasize the apparent advantages while minimizing or even neglecting to stress the shortcomings and risks. The authors of such published reports bear the responsibility for the accidents or adverse reactions which may be experienced or observed by other physicians who have not been adequately alerted and forewarned.

The authors express regret over their initial over-

enthusiasm, as regards the use of large doses of contrast material in infusion cholangiography.

Adipiodone (cholografin-biligrafin) is 6 times more toxic than contrast media ordinarily used for angiography and excretion urography. Doses of 60 ml. adipiodone initially used for cholangiography can be dangerous, particularly in the presence of insufficiency of the liver and/or kidney function.

In their investigation the authors performed the following laboratory tests before and 14 hours after roentgenologic examination of the biliary tract: transaminase, lactic-dehydrogenase (L.D.H.), bilirubin, bromsulphalein and creatinine clearance. The last 2 tests are considered to be the most reliable in determining liver and kidney functions, respectively.

The examinations performed were: single dose oral cholecystography; double dose oral cholecystography; infusion cholangiography with 40 ml. adipiodone; infusion cholangiography with 60 ml. adipiodone; and oral cholecystography followed the next day by infusion cholangiography. There was elevation in the values of all 5 laboratory tests performed following double dose oral cholecystography, infusion cholangiography using 60 ml. adipiodone, and oral cholecystography followed by infusion cholangiography.

In the authors' experience the incidence of adverse reactions is less with infusion cholangiography than with regular cholangiography.

In the preparation of infusion cholangiography 40 ml. adipiodone is added to 250 ml. 5 per cent glucose solution. In case of nonvisualization of the gallbladder following oral cholecystography they recommend that infusion cholangiography be performed after an interval of at least 6 days.—*William H. Shehadi, M.D.*

Gynecology and Obstetrics

Aquilini, E., and Arena, G. C. Indagine isterosalpingografica preoperatoria e postoperatoria per gli interventi di miomectomia. (Pre- and postoperative hysterosalpingography in surgical intervention of myomectomy.) *Radiol. med.*, 1970, *56*, 37–51. (Address: Dott. Emilio Aquilini, Corso G. Garibaldi, 59, Vercelli, Italy.)

The authors performed hysterosalpingography before and after surgery in 30 patients in whom myomectomy was done because of myomata of the uterus.

Described are the known roentgen findings demonstrable in the hysterograms, and analyzed are the changes produced in the morphology of the uterine cavity following myomectomy.

Emphasized also is the importance of hysterosalpingography performed postoperatively, in the demonstration of complications, such as uterine adhesions, fistulous tracts in the myometrium, and in the evaluation of possible late pathologic changes involving the salpinges.—*A. F. Govoni, M.D.*

Genitourinary System

Samuel, Eric. Radiology in the diagnosis of renal rejection. *Clin. Radiol.*, April, 1970, *21*, 109–118. (From: Department of Diagnostic Radiology, University of Edinburgh, Edinburgh, Scotland.)

An analysis of 35 patients who received renal transplants reveals that the acute rejection phases occurred at approximately the 7th and the 32–37th day postoperatively. These acute rejection episodes probably represent exacerbations in a continuing process of chronic rejection.

The basic pathology of rejection is not fully understood, but it appears that antibody sensitized lymph cells attack the vascular endothelium of the transplant which initially results in increased permeability of the vessel wall with passage of the cells into the interstitial tissues, and ultimately leads to thrombosis and vessel obstruction. The tubules appear to be particularly vulnerable and the swelling of the interstitial tissue between the tubules resulting in enlargement of the pyramids produces one of the earliest radiographic findings of rejection. Meanwhile the glomerulus becomes infiltrated with round cells and the lumens of the intralobular vessels are reduced by proliferation of the endothelium and thrombus, and result in ischemia of segments of the kidney.

The clinical symptoms of rejection are nonspecific and include malaise, generally feeling unwell, fever, and a fall in urinary output. The biochemical features are equally nonspecific and consist of a raised blood urea, nonprotein nitrogen, and increased blood creatinine. A marked decrease in the creatinine clearance ratio indicates the onset of renal failure.

The radiographic findings in a rejection crisis include a greater than 20 per cent increase in kidney size as compared with its pretransplant size, a delay in the runoff through the main arterial and intrarenal branches, edema and swelling of the renal pyramids and ultimately progressive failure of excretion of the contrast medium with poor nephrographic and pyelographic phases.

Some authorities have adopted a pragmatic approach to the diagnosis of rejection by implying that if radioisotope tests indicate that perfusion of the kidney is occurring, a decrease in renal urinary output must imply renal rejection. One must be careful, however, as the clinical and biochemical features of rejection can be produced by obstructive uropathy, renal artery obstruction, or renal vein thrombosis.

Obstruction to the ureter most likely develops at the site of implantation into the bladder, with sloughing of the lower end of the ureter. Intravenous pyelography with tomography, and occasionally retrograde pyelography, are valuable aids in studying the distal ureter.

Possible renal artery obstruction is evaluated by percutaneous puncture and catheterization of the ipsilateral femoral artery and use of a rapid film changer following positioning of the tip of the catheter in the common iliac artery. An injection of 20 cc. of contrast medium was used. Some degree of narrowing is always present at the site of arterial anastomosis, but if any dilatation of the renal artery is present beyond the anastomosis, then significant narrowing of the artery is considered to be present.

The clinical findings of heavy proteinuria associated with a diminished renal output is strong evidence of a renal vein thrombosis. Retrograde catheterization of the common iliac vein and a forced injection during a Valsalva maneuver will often fill the anastomosed vein. While filling of the anastomosed vein is positive evidence of its patency, nonfilling could be due either to technical factors or rapid washout through normal veins.

The appearance of pulmonary symptoms or radiologic signs should raise the question of renal rejection, as the bronchial epithelium is antigenically similar to the epithelium of the kidney glomerulus. Clinically, respiratory function tests reveal a capillary block syndrome before there are changes on the roentgenograms. The chest roentgen findings have been described as ill-defined pulmonary opacities which must be distinguished from patchy pulmonary edema which may be associated with the underlying renal condition or with infection.—*Donald M. Monson, M.D.*

Kaftori, J. K., Munk, J., Better, O. S., Erlik, D., and Barzilai, A. The pyelographic appearance of the transplanted cadaver kidney. *Clin. Radiol.*, April, 1970, *21*, 119–123. (From: Departments of Diagnostic Radiology, Nephrology and Surgery, Rambam Government Hospital, Haifa, Israel.)

The material consists of 19 drip infusion pyelographies performed on 11 recipients of cadaver kidneys out of a series of 17 cadaver grafts.

The first postoperative pyelographies were usually performed about 6 to 8 weeks after the transplantation, and by this time the most severe rejection reactions had been overcome. All transplanted kidneys so examined revealed clear evidence of pyelographic function.

The transplanted kidneys appeared large, the measurements being 14–17 cm. in length and 7–11 cm. in width, measured on films exposed with the patients in the prone position as the kidneys were transplanted into the iliac fossa of either side. The big size of the kidneys may be due to hypertrophy, but edema of rejection may be a factor.

There was good to excellent visualization of the pyelocalyceal system in all but the first 3 cases examined. The ureteropelvic or ureteroureteric anastomosis was well shown in most of the cases. In 5 patients caliectasis and clubbing involving the transplanted kidney were noted. It is thought that this might be due to disease inherent to the transplant or to damage of the graft before or after operation. Infection after the transplantation was probably not a cause as none of the patients in the series had signs of urinary tract infection.

Retrograde flow of the contrast medium from the renal pelvis of the transplant into the original ureter cranial (and dependent) to the anastomosis was a constant feature in all patients. Spontaneous reflux into the ureter opposite the site of the transplant was seen in 2 cases. No reflux from the bladder was noted into the cadaveric ureter if such was transplanted with the kidney.

The existing theories dealing with reflux up the original ureter are discussed. The reflux may be due to a lack of ureteric tonus caused by long standing absence of normal kidney function. So-called ureteric inertia as well as disruption of neural influences by the transplantation procedure also are considered.

There were no marked changes in the pyelographic appearance in the cases followed up for 2 years. No urologic complications were observed.—*Donald M. Monson, M.D.*

Rosenberger, A., Munk, J., Better, O. S., Erlik, D., and Barzilai, A. The angiographic signs of rejection in cadaver kidney transplants. *Clin. Radiol.*, April, 1970, *21*, 135–143. (From: Departments of Diagnostic Radiology, Nephrology and Surgery, Rambam Government Hospital, Haifa, Israel.)

The most common diagnostic problem in cadaveric renal transplants in the early postoperative period is the oliguria which may be the result of acute renal failure secondary to ischemia, vascular thrombosis, ureteral perforation or obstruction, or tissue rejection. The isotope renogram may show the delayed blood flow through the kidney which is considered to represent the sign of rejection, but the anatomic causes of the delayed blood flow are not shown by this method.

The technique of angiographic evaluation in the 8 cases being reported was to catheterize the ipsilateral femoral artery with a 240 polyethylene catheter using the Seldinger technique. The tip of the catheter was positioned near the site of the anastomosis, but the renal artery was not selectively cath-

eterized. Supine roentgenograms were made with a rapid film change for a period of 22 seconds in order to achieve visualization of the renal veins.

The angiogram after a successful implantation was that of a normal kidney. The anastomosis and main renal artery, the first and second degree vessels and the arcuate arteries were well visualized and there was rapid runoff through the kidneys. The outline of the kidney was smooth and the cortex was extremely well shown.

The signs of rejection of slight degree were seen to be a prolonged arterial phase suggesting delayed blood flow. In general, the iliac artery, the main renal artery and the anastomosis had a normal appearance. There was stretching of the first and second degree renal arteries as a manifestation of edema, and the arcuate and small arteries were not visualized. The underlying pathologic process is known to be subintimal proliferation causing obliteration of the small arteries. The outline of the kidneys appeared smooth in all cases; the cortex however, was not well shown.

In rejection of severe degree the main angiographic sign was shown to be the narrowing of the main renal arteries and the bigger arterial branches, indicating that the proliferative obliterating process had involved these vessels. The other arterial branches were either faintly visualized or completely missing. The arterial phase was short because there was no filling of the small arteries. There was no obvious nephrographic phase. The outline of the kidney was either faintly visible and serrated, or was not seen at all.—*Donald M. Monson, M.D.*

Fletcher, E. W. L., Lecky, J. W., and Gonick, H. C. Selective phlebography of transplanted kidneys. *Clin. Radiol.*, April, 1970, *21*, 144–149. (From: U.C.L.A. School of Medicine, Los Angeles, Calif.)

Renal transplant phlebography may be requested when a patient has marked proteinuria which is not accounted for by urinary obstruction or infection, or when there are signs and symptoms of acute rejection and venous thrombosis of either the vein of the transplanted kidney or the vein of the recipient to which it is anastomosed. This report is on 8 patients who had 9 selective phlebographies of their transplanted kidneys.

The operative notes should be reviewed to find the exact site of the anastomosis before puncturing the common femoral vein on the side of the transplant by a Seldinger needle and the introduction of a No. 5 Fr. brachio-vertebral polyethylene end and sidehole catheter. A hand injected test dose is used to fluoroscopically monitor the iliac veins and inferior vena cava for abnormalities, and if an abnormality is seen, 20 cc. of contrast medium is injected and a single roentgenogram of the iliac veins and the lower inferior vena cava is taken. A large amount of thrombus in the iliac veins is a contraindication for further catheterization and pertrochanteric phlebography may be necessary. If there is only a little thrombus distal to the site of the renal vein anastomosis and no thrombus is seen proximally, selective catheterization is performed either from the contralateral common femoral vein or from the jugular vein.

If there is no evidence of thrombosis an attempt is made to selectively catheterize the transplanted renal vein. If successful, the tip of the catheter is positioned just proximal to the bifurcation of the renal vein, and 20 cc. of renografin 76 is then injected by hand, while 10 films are exposed on a serial changer at a speed of $1\frac{1}{2}$ films per second with the patient supine.

In 2 of the patients in this series there was extremely good filling of the intrarenal veins, and this suggests very poor renal arterial perfusion. Rejection was diagnosed on the grounds of the diminished renal blood flow and this diagnosis was confirmed by renal arteriography and renal biopsy. In 3 patients there were varying degrees of thrombus formation at or near the anastomotic site but this either did not interfere with renal function or there had been recanalization with collateral circulation opened up through the presacral veins to the opposite internal iliac vein. In the other 3 patients there was failure of filling of the peripheral branches of the renal veins as the washout of the contrast medium was extremely fast. This suggests that the blood flow was good and is interpreted to represent a normal phlebogram.

Thrombus is not difficult to detect in the main renal veins, but care must be taken to differentiate it from washout by blood flowing from veins not filled by contrast material. Venous filling can be improved by epinephrine injection through a selective renal artery catheter, but the authors do not feel that this risk to the transplanted kidney is warranted.

In this series 3 kidneys which had been transplanted into the left pelvis were examined by phlebography and 2 of these were shown to have renal vein thrombosis, whereas only 1 of the 5 kidneys transplanted into the right side of the pelvis was affected. This predisposition to thrombosis of the left common iliac vein has been demonstrated by previous workers to be due to the flattening of the left common iliac vein where it is crossed by the right common iliac artery.

The authors conclude that retrograde renal transplant phlebography is a safe and useful procedure in experienced hands, and is not technically difficult, although metal clips at the site of anastomosis would make location of the transplant renal vein easier. In no case was thrombus dislodged and there was no clinical evidence of additional thrombosis after selective catheterization.—*Donald M. Monson, M.D.*

Wilkinson, Robert H., Jr., and Goodrich, Jack K. Scintillation camera renography in the identification of an uncommon renal transplant complication. *J. Urol.*, July, 1970, *104*, 36–40. (From: Division of Nuclear Medicine, Department of Radiology, Duke University Medical Center, Durham, N. C.)

The scintillation camera allows simultaneous recording of bilateral radionuclide renographic curves while obtaining sequential images of radionuclide transit through renal structures. Eighteen μc of sodium orthoiodohippurate labeled with I^{131} is injected intravenously. Radionuclide activity over the renal areas is recorded on a strip chart for 25 minutes and serial 1 minute scintiphotographs are obtained for 11 minutes. At the termination of this study an image is obtained over the bladder area.

The authors emphasize the advantages of this dual investigative technique to monitor renal transplants. They illustrate this by citing an example of the correct diagnosis of an operative complication: extravasation of urine in the pararenal area. The images obtained provided a method of differentiating rather nonspecific renographic curves.

This combined study is considered an extension of the conventional renogram in evaluation of renal transplant function.—*A. Everette James, Jr., M.D.*

Christiansen, N., Hansen, H., and Madsen, P. O. Renal isotope clearance by external monitoring and feedback technique: a new catheter-free clearance method. *J. Urol.*, July, 1970, *104*, 26–35. (From: Urology Section, Veterans Administration Hospital, and the Department of Surgery, University of Wisconsin School of Medicine, Madison, Wisc.)

The most reliable method to determine renal clearance of an exogenous substance is by a continous infusion technique. Single injection methods have been devised but, while more convenient, do not overcome the inaccuracies due to distribution of the single injection. Continuous infusion techniques have, in the past, required multiple measurements and adjustments to assure a steady state plasma concentration.

The authors describe a catheter-free clearance method utilizing external monitoring of radioactivity to assure constant plasma concentration. The principle of this new technique is to measure the amount of diodrast labeled with I^{131} that must be intravenously infused to keep the plasma concentration constant. From these determinations the renal clearance can be calculated. Adjustment of the amount of radiopharmaceutical infused is made automatically by technical innovations described in the article.

This method of determining renal clearance rapidly detects changes in renal function, is more accurate than a single injection technique, and avoids the use of a catheter for urine collection.—*A. Everette James, Jr., M.D.*

Rodriguez-Antunez, Antonio, Gill, Wilfrid M., Jr., and Egleston, Thomas A. Assessment of function in the transplanted kidney with ^{131}I hippuran. *J. Urol.*, May, 1970, *103*, 574–576. (From: Department of Radiation Therapy and Nuclear Medicine, The Cleveland Clinic Foundation, Cleveland, Ohio.)

In post transplantation assessment of kidney function, 3 procedures are utilized by the authors: scintigraphy; $T_{\frac{1}{2}}$ blood clearance; and renography. I^{131} hippuran (30–50 μc) is employed as the radiopharmaceutical.

The scintigraphic image is useful in determining kidney viability and to detect infarction but may be normal early in rejection. The $T_{\frac{1}{2}}$ blood clearance reflects blood flow and is the most sensitive test for renal rejection. Due to the incidence of false positives, the renogram was found to be of little value.

Although the authors' experience with evaluation of renal transplants is considerable, they do not present the data upon which these observations are based.—*A. Everette James, Jr., M.D.*

Hale, J. E., and Whitehouse, G. H. Spontaneous extravasation of urine during emergency intravenous pyelography. *Brit. J. Surg.*, April, 1970, *57*, 302–306. (From: Westminster Hospital, London, England.)

Spontaneous extravasation of contrast medium from the pelvicalyceal system into the renal sinus and beyond was observed in 4 patients with ureteric colic—apparently a spontaneous "back-flow" due to sudden increase in intrapelvic pressure causing minute tears of the calyceal fornix during ureteric colic. Complications are rare and no treatment is required, thus making it important to distinguish this from true rupture of the renal pelvis or ureter which may present as an acute abdomen and require surgical treatment.

For the diagnosis of spontaneous extravasation it is essential that there be no history of trauma, ureteric instrumentation, recent surgery or abdominal compression (extravasation occurs in about 27 per cent of cases of intravenous urography with compression, but usually, only after tight compression for 20 minutes or longer). Destructive renal lesions are likely to cause true rupture and therefore spontaneous extravasation cannot be diagnosed if there is evidence of such a lesion.

The density in spontaneous extravasation is usually pericalyceal, but occasionally may spread into the perirenal fat and even downwards into the retro-

peritoneal space. The ureter is usually well demonstrated. The extravasated contrast material is rapidly absorbed by lymphatics, and therefore transient. In contrast, following true rupture, radiologic appearances remain unchanged for longer periods and are reproduced on serial roentgenograms. It is therefore advisable to repeat the examination after a few days.—*Lois Cowan Collins, M.D.*

Hollenberg, Norman K., Adams, Douglass F., Oken, Donald E., Abrams, Herbert L., and Merrill, John P. Acute renal failure due to nephrotoxins: renal hemodynamic and angiographic studies in man. *New England J. Med.*, June, 1970, *282*, 1329–1334. (Address: Dr. Hollenberg, Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston, Massachusetts 02115.)

The mechanism of sustained oliguria and azotemia in patients with acute renal failure has not been conclusively established.

Seven patients with nephrotoxin-induced acute renal failure showed renal hemodynamic and angiographic abnormalities identical to those found in patients with acute failure due to shock and hemolysis, suggesting a pathogenetic final common pathway involving undefined mediators that induce preglomerular vasoconstriction.

These abnormalities include the absence of recognizable cortical arterial vessels and the cortical nephrogram, delayed transit of contrast material through the kidney, and disappearance of the cortical flow component of xenon washout from the kidney.—*Lois Cowan Collins, M.D.*

Skeletal System

Hoover, Galen H., Flatt, Adrian E., and Weiss, Madge W. The hand and Apert's syndrome. *J. Bone & Joint Surg.*, July, 1970, *52-A*, 878–895. (From: Division of Hand Surgery, Department of Orthopedic Surgery, University of Iowa, Iowa City, Iowa.)

This comprehensive article illustrates and describes the group of anomalies constituting Apert's syndrome (acrocephalosyndactyly).

The hand changes in 20 patients with Apert's syndrome, consisting of syndactyly, radially deviated, shortened thumbs, partial separation of the little fingers, varying degrees of limitation of motion at the metacarpal-phalangeal joints, and synonychia, are discussed in detail.

The authors suggest surgical principles designed to restore maximum function of the hands.—*John H. Harris, M.D.*

Cremin, B. J., and Jarrett, J. Diastrophic dwarfism. *Australasian Radiol.*, Feb., 1970, *14*, 84–87. (From: Department of Radiology, Red Cross War Memorial Children's Hospital, Cape Town, South Africa.)

The authors report a case of diastrophic dwarfism first studied roentgenographically at 5 months of age.

Marked sloping of the anterior cranial fossa, thoracic spine scoliosis, bilateral hip dislocation, expansion of the proximal humeri, femora, anterior rib ends, equino-varus deformity of the feet, and dislocated radial heads represent the major findings in this patient.

The stated salient features of the syndrome are scoliosis, dislocations, clubbed feet, short limbs, and ear deformities.

A differential diagnosis includes arthrogryposis multiplex congenita, Morquio's disease, achondroplasia, and metatropic dwarfism.—*Victor G. Mikity, M.D.*

Gupta, S., and Chawla, S. Iliac horns with arthrodysplasia and dystrophy of the nails—Fong's lesion. *Indian J. Radiol.*, Nov., 1969, *23*, 209–214. (From: Department of Radiology, Lady Hardinge Medical College and Hospital, New Delhi-1, India.)

The authors report iliac horns with arthrodysplasia and dystrophy of the nails (Fong's lesion) in a 26 year old mother and her neonate and also review the literature. The neonate showed marked genu recurvatum, limited extension at the elbows and onychodysplasia affecting only the hands. Roentgenograms of the infant's pelvis showed iliac horns.

The most constant deformity in the syndrome is the abnormality of the thumb nails varying from cracks and ridges to complete absence. The nail dystrophy is associated with various combinations of elbow, pelvis and knee dysplasias. The knee dysplasia is seen in 80 per cent of published cases, elbow and pelvis dysplasias in 58 per cent and 30 per cent, respectively.

The genetic importance of the nail-patella syndrome derives from the linkage between the nail-patella locus and the ABO blood group locus both being situated on the same chromosome. The mode of transmission is autosomal dominant.

Although patients with the syndrome are conscious of an abnormality, most go through a normal life span without gross physical disability.—*Lionel W. Young, M.D.*

Saha, M. M., and Bhardwaj, O. P. Hereditary onycho-osteodysplasia (Fong's lesion). *Indian J. Radiol.*, Nov., 1969, *23*, 199–205. (From: Department of Radiology, Maulana Azad Medical College and Associated Irwin and Pant Hospitals, New Delhi, India.)

The authors report 3 cases and review the literature. They mention the original 3 cardinal features

of the syndrome consisting of malformation of the nails, hypoplasia or total absence of the patella and deformities with impaired function of the elbow. Fong's contribution of the fourth feature "iliac horns" emphasizes the current recognition of a tetrad of features. Multiple other associated findings have also been described.

The authors question the validity of the incidence of components of the tetrad as reported by Duncan and Souter (1963). By restricting the series as reviewed by those authors to include only patients who were examined clinically and radiologically, the dysplasia incidence in a total of 120 patients was found to be: nails 97 per cent, knee 93 per cent, elbow 68 per cent and pelvis 66 per cent.

The authors describe the types of defects seen in the nail. The radiologic findings were in the pelvis: steep descent of iliac crest, prominence of the anterior superior iliac spine and deepening of the supra-acetabular notch; in the knee: prominent medical condyles, small or absent patellae; and in the elbow: prominent medical epicondyles with increased valgus. All had nail defects and pelvic dysplasia, while 2 of the 3 cases had knee and elbow dysplasias.—*Lionel W. Young, M.D.*

RADIOISOTOPES

Miraldi, Floro, and DiChiro, Giovanni. Tomographic techniques in radioisotope imaging with a proposal of a new device: the tomoscanner. *Radiology*, March, 1970, *94*, 513–520. (Address: Floro Miraldi, Sc.D., Division of Biomedical Engineering, Case Western Reserve University, University Circle, Cleveland, Ohio 44106.)

The many methods of section scanning now in use may be grouped into 3 scanning procedures depending on whether the collimator is (a) inclined to the plane of interest, (b) in the plane of interest, or (c) of the focusing type. Actually, the latter 2 procedures are special cases of the first. In the present study, both the general method of collimators inclined to the plane of interest and the special case of focusing collimators are analyzed. This has led directly to the development of a new method.

In the case of the inclined collimator, a computer program was written to obtain the response of a single tapered channel system to a point source in air in terms of characteristic collimator parameters. The results of the calculations indicated that the depth resolution distance is least for small angles of inclination to the plane of interest and increases as the inclination angle increases. These results together with the physical characteristics of these systems indicate that there must be 2 or more collimators to work effectively. Their best use is obtained by time-delay summation methods to define the plane of interest. Finally, their depth resolution for a given plane can be controlled by collimator design but, once chosen, the resolution distance increases with depth.

The special case of the focused collimator system was also analyzed by a computer program which summed the response of each individual channel in the collimator. The results clearly show the decrease in resolution distance as one adds more lateral channels at smaller inclination angles and predicts the best tomographic results with the larger focused collimators. The disadvantage of this procedure is that a separate scan has to be made for each plane of interest. However, this limitation may be removed by additional use of the Anger camera system to produce a scan in which by time delay summation one can select a plane of interest. However, like all focused collimator systems, the resolution distance increases with distance from the collimator face. The inclined focused collimator was also analyzed by computer techniques which predicted large changes in resolution with depth of the plane of interest with increased sensitivity due to the exposure of more detector area.

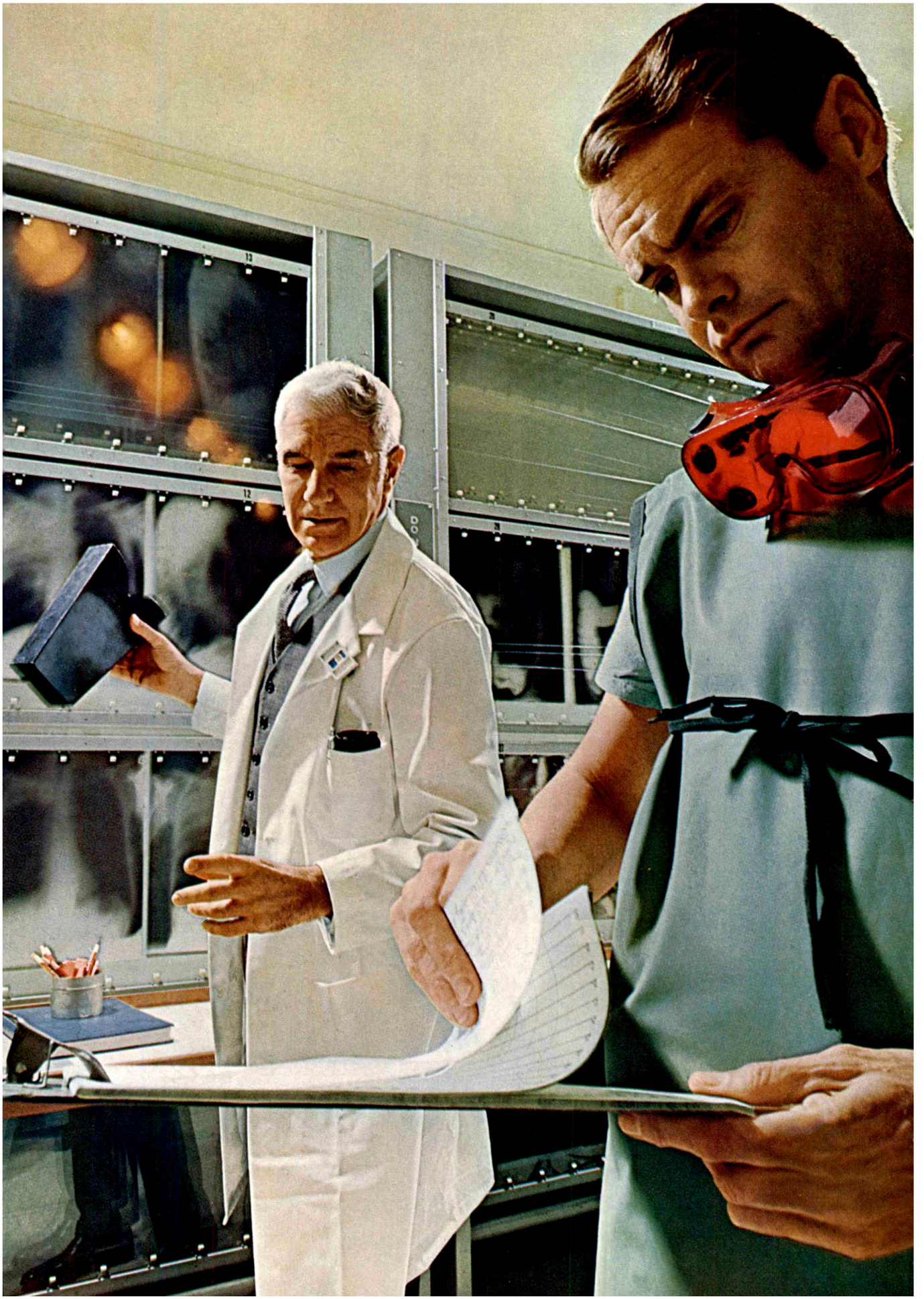
A new system, the *Tomoscanner*, incorporates a multiplane focused slit geometry collimator and revised scanning procedure along with an electronic means for adding certain slit responses to view different layers as desired. It can also be designed to have optimal resolution for each of a number of planes of interest, and by electronic summation techniques can visualize any layer of interest without the need for a rescan. A prototype of this type of scanner is described.—*L. H. Dieterman, Ph.D.*

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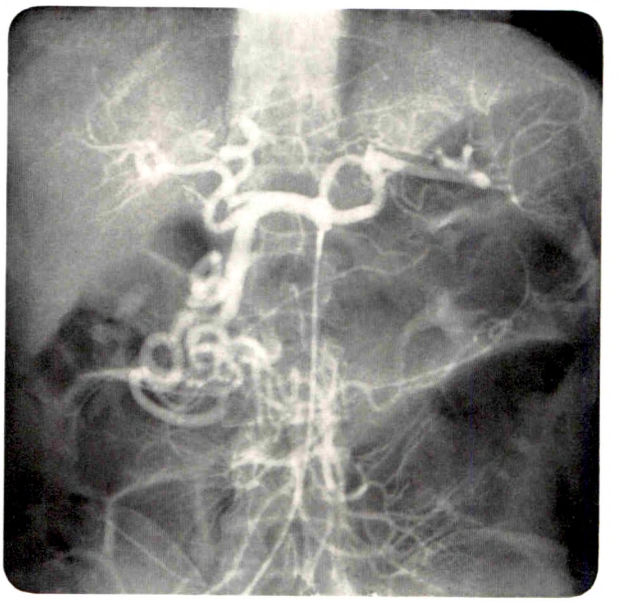
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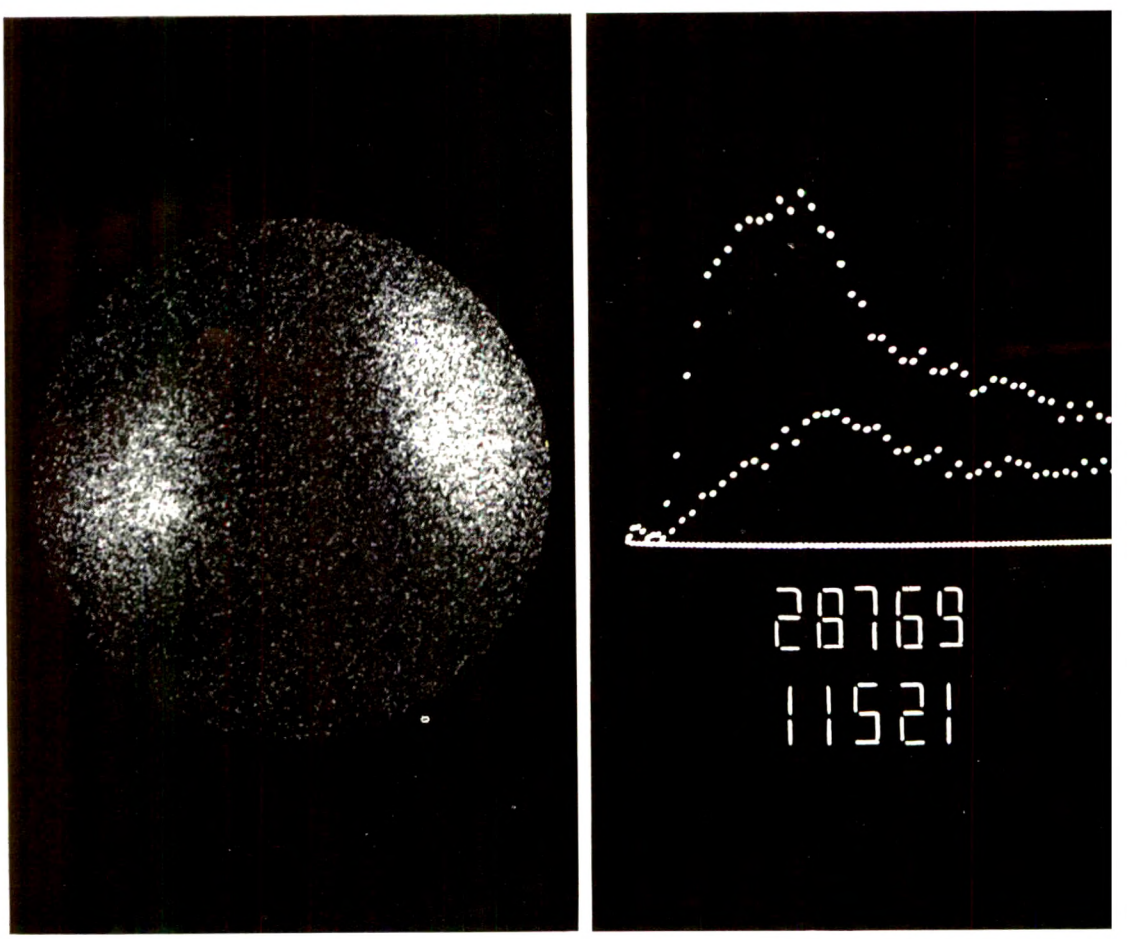


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*F. Clark and D. B. Horn, Journal of Clinical Endocrinology, 25:39-45, Jan. 1965.

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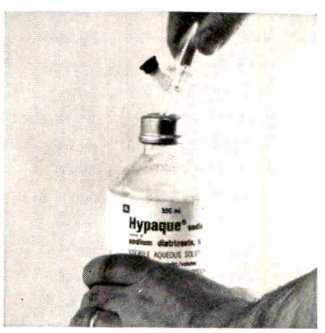
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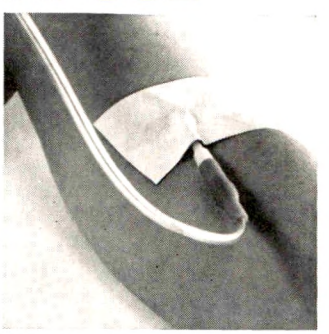
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- may be the only (excretory) method of achieving diagnostic contrast in the azotemic patient
- dehydration and compression not always essential
- 300 ml. dose usually as well tolerated as the conventional dose for intravenous urography . . . no significant changes in BUN reported after use in patients with impaired renal function

PRECAUTIONS

Before a contrast medium is injected, the patient should be questioned for a history of allergy. Although a history of allergy may imply a greater than usual risk, it does not arbitrarily contraindicate the use of the medium. Premedication with antihistamines to avoid or minimize possible allergic reactions may be considered. **Benadryl® (brand of diphenhydramine hydrochloride) however, may cause precipitation when mixed with Hypaque sodium.** The intravenous injection of 0.5 ml. to 1 ml. of the contrast medium approximately 15 minutes prior to infusion of the full dose is frequently used in an effort to detect patient sensitivity. The absence of a reaction to this test dose is not entirely reliable for predicting the patient's response to the full diagnostic dose. Severe reactions, including fatalities, have occurred with a test dose as well as with larger doses. Adequate facilities for treating severe reactions should be available.

Caution is advised in patients with severe cardiovascular disease and in patients with a history of bronchial asthma or other allergic manifestations or of sensitivity to iodine.

Contrast media have been shown to promote the phenomenon of sickling in individuals who are homozygous for sickle cell disease when the material is injected intravenously or intraarterially.

In myelomatosis, urography should only be performed with caution. If a weak protein-binding agent such as a diatrizoate is used for the procedure it is essential to omit preparatory dehydration, administer fluids, and attempt to alkalinize the urine.

The results of PBI and RAI uptake studies, which depend on iodine estimations, will not reflect thyroid function for up to 16 days following administration of iodinated urographic media. However, function tests not depending on iodine estimations, e.g., T_3 resin uptake or direct thyroxine assays, are not affected.

Immediate adverse reactions to drip infusion pyelography have not been reported to occur at a higher frequency or greater severity than with routine excretory urography. However, sequelae of this procedure are possible some hours after the examination. Drip infusion pyelography imposes not only a sudden osmotic load, but also may present as much as 160 meq. of sodium (3.7 Gm.) to patients with established, decreased glomerular filtration and renal tubular damage. In addition, these patients may also have coexisting or associated cardiovascular disease. Therefore, the possibility of the development of congestive heart failure hours after the procedure should be considered.

Because of the possibility of inducing temporary suppression of urine, it is wise to allow an interval of at least 48 hours to pass before repeating drip infusion pyelography in patients with unilateral or bilateral reduction of normal renal function.

Preparatory dehydration or abdominal compression may not be necessary in infusion pyelography. In infants and young children and in azotemic patients, especially those with oliguria, dehydration is considered dangerous.

ADVERSE REACTIONS

Reactions accompanying the use of contrast media may vary directly with the concentration of the substance, the amount used, the technique used, and the underlying pathology.

Adverse reactions, usually of a minor nature, have occurred in 10-14 per cent of patients who have received Hypaque intravenously. Reactions due to **technique** include hematomas and ecchymoses following extravasation from the vein, and pyrogenic reactions. **Hemodynamic reactions** include vasodilatation with flushing, hypotension and, rarely, vein cramp or thrombophlebitis. Serious **cardiovascular reactions** include rare cases of cardiac arrhythmias (e.g., ventricular fibrillation), shock, and cardiac arrest. Transient proteinuria may occur occasionally following the injection of radiopaques and, rarely, oliguria and anuria (renal shutdown) have been reported as a secondary effect of a hypotensive reaction. **Allergic reactions** include asthmatic attacks, nasal and conjunctival symptoms, dermal reactions such as urticaria and, rarely, anaphylactic shock, sometimes with fatal outcome. Severe reactions may also be manifested by signs and symptoms relating to the **respiratory system** (dyspnea, cyanosis, pulmonary or laryngeal edema), or to the **nervous system** (restlessness, confusion, or convulsions). **Other reactions** include nausea, vomiting, excessive salivation, anxiety, headache, and dizziness. Infrequently, "iodism" (salivary gland swelling) from organic compounds appears two days after exposure and subsides by the sixth day.

DOSAGE AND ADMINISTRATION

The recommended dose is calculated on the basis of 2 ml. of Hypaque sodium 25 per cent per pound of body weight. The average dose of the solution for adults is 300 ml.; for optimum results, a minimum dose of 250 ml. should be used. A maximum dose of 400 ml. is generally sufficient for the largest of subjects.

The solution is administered intravenously through an 18-gauge needle over a period of three to ten minutes. Pyelographic films are taken at 10, 20, and 30 minutes from the beginning of the infusion. Early 2, 3, 4, and 5 minute films are obtained when indicated for the evaluation of hypertension. Nephrotomographic sections are best taken just at the end of the infusion, and voiding cystourethrograms when desired are usually made at 30 minutes.

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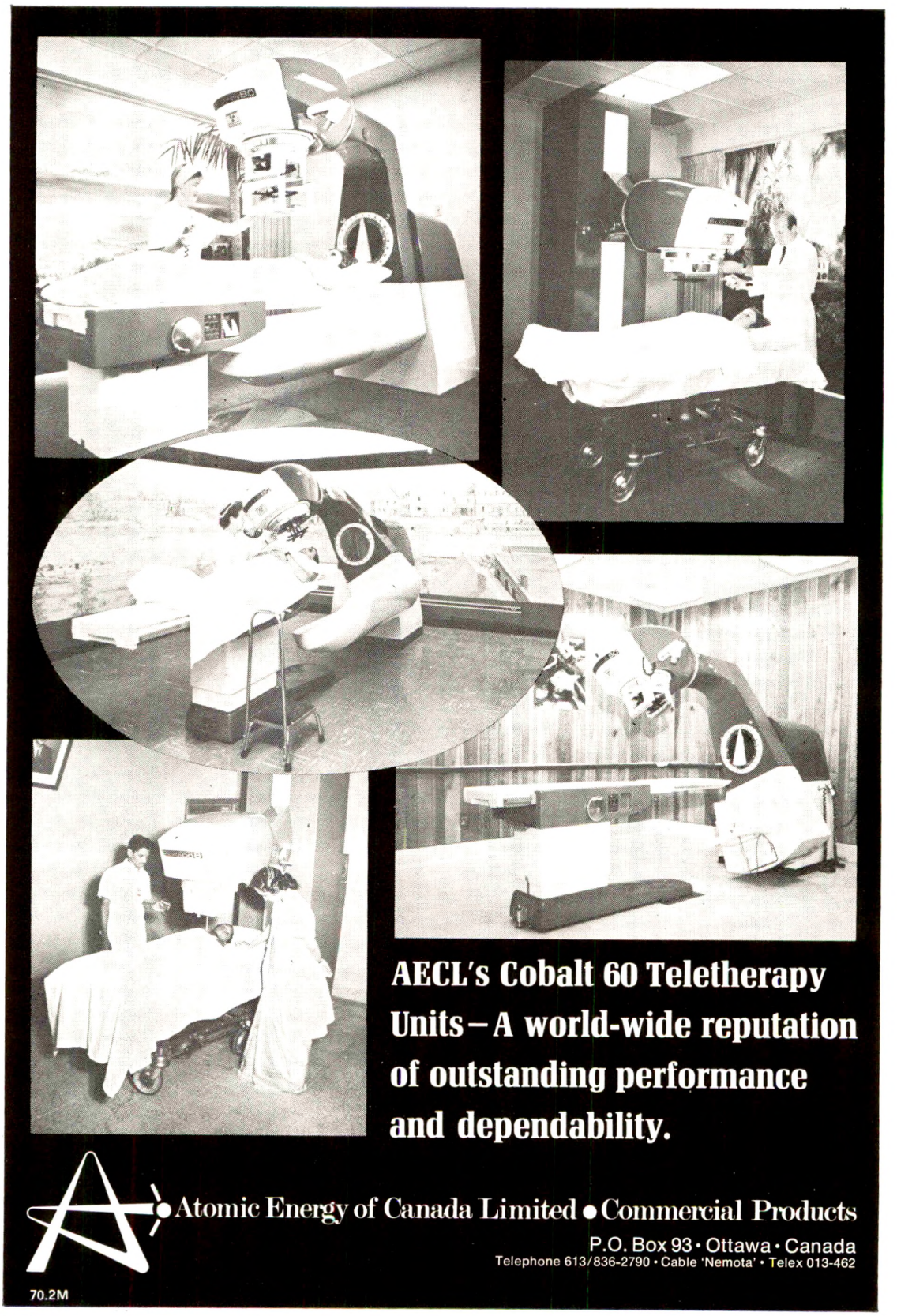
PRECAUTIONS, ADVERSE REACTIONS: Care should be taken to administer the minimum dose consistent with patient safety and validity of data. The thyroid gland should be protected by prophylactic administration of concentrated iodide solution. Urticaria and acute cor pulmonate, possibly related to the drug, have occurred.

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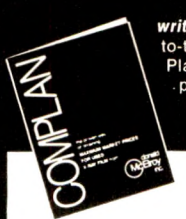
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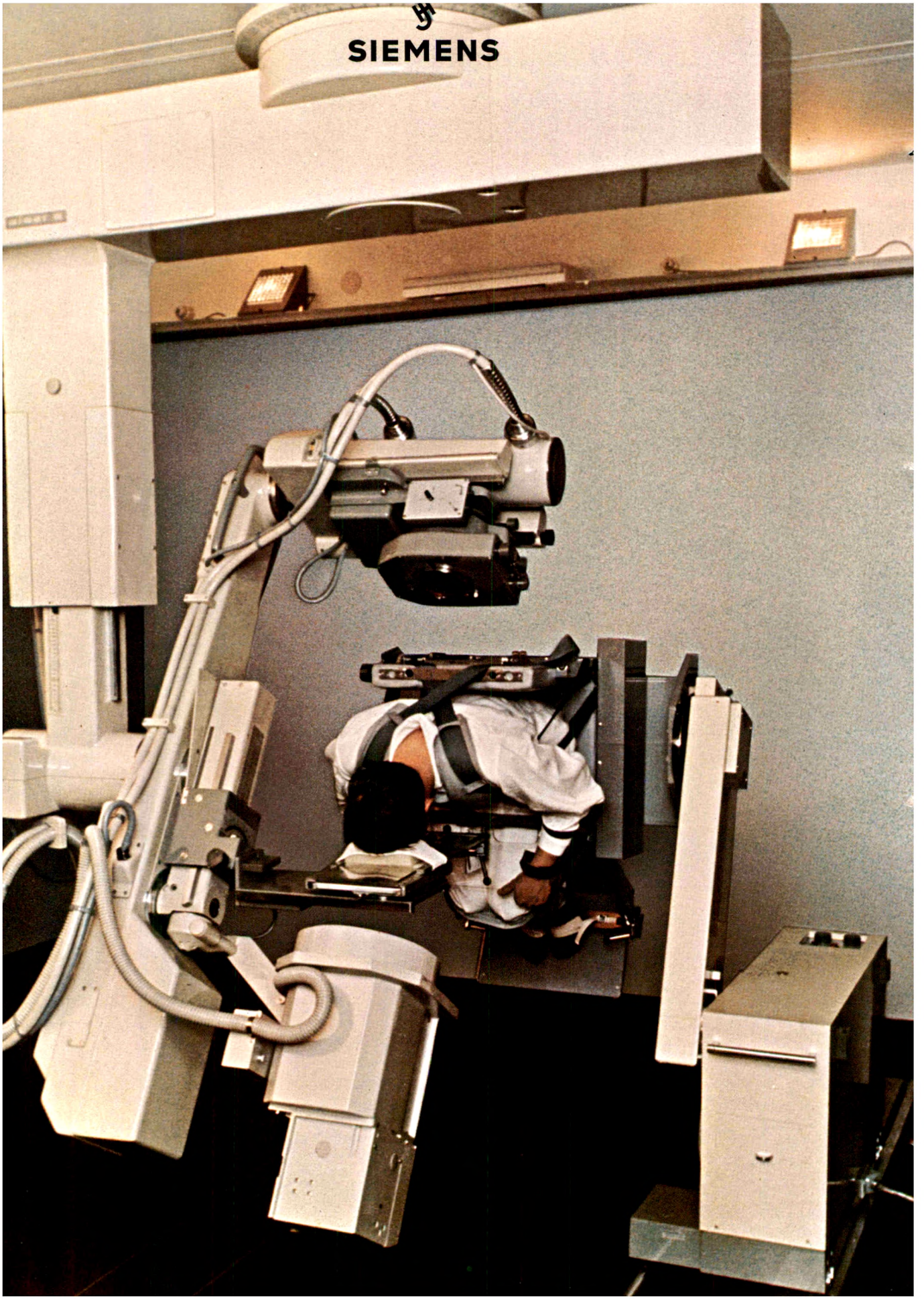
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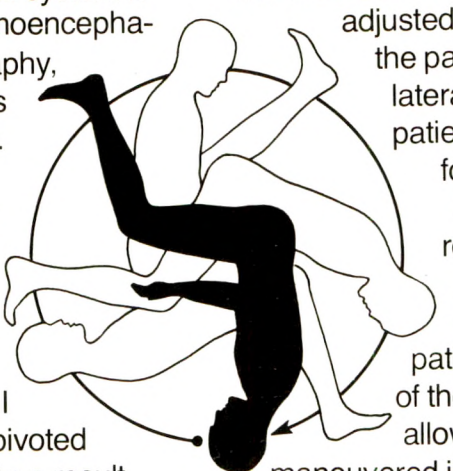
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pathology of irradiation

edited by

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Colonel, Medical Corps, U.S. Army;
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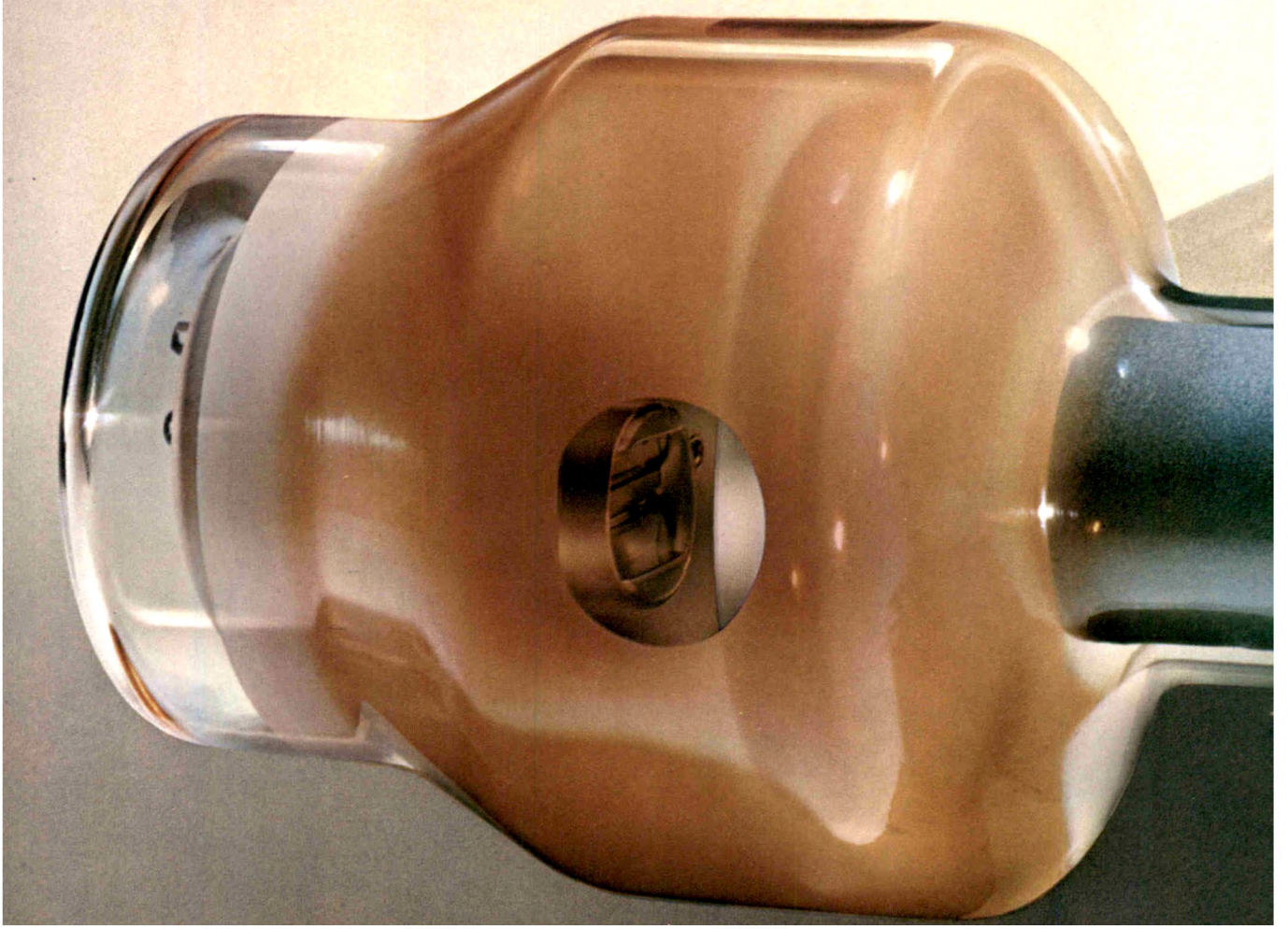
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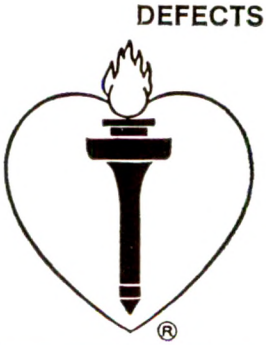
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Advanced hepatorenal disease or severe impairment of renal function, severe gastrointestinal disorders that prevent absorption, and in some patients sensitive to iodine compounds.

PRECAUTIONS

Severe, advanced liver disease may interfere with metabolism of the radiopaque medium, and therefore, a greater amount of unchanged Telepaque will be diverted for renal excretion, increasing the load on the kidneys. Although renal difficulty has rarely been attributed to Telepaque, renal function in patients with severe, advanced liver disease should be assessed before cholecystography, and renal output and hepatic function should be observed for a few days after the procedure.

Patients with preexisting renal disease should not receive high doses of cholecystographic media. Possible renal irritation in susceptible individuals could result in reflex vascular spasm with partial or complete renal shutdown.

Caution is advised in patients with coronary disorders, especially those with recent symptoms of coronary artery disease. Blood pressure should be observed after administration of cholecystographic media to these patients.

Elevation of protein-bound iodine for several months and false positive urine albumin tests (for three days) may occur after ingestion of iodine-containing cholecystographic media.

ADVERSE REACTIONS

Most reactions are mild and transitory; a very low incidence of serious side effects has been attributed to Telepaque. Most frequently reported have been mild and transitory nausea, vomiting, cramps, and diarrhea (usually occurring as a few loose stools). Frank diarrhea and cramps have been noted in only a small number of patients, and severe diarrhea with prostration is rare.

A mild stinging sensation during urination may occur, and rarely, skin and mucous membrane reactions (e.g., skin rash, urticaria, pruritus, and flushing). One case of thrombocytopenia has been reported in a patient with a history of conjunctival hemorrhages. Miscellaneous subjective complaints are dryness of the throat, burning on swallowing, heartburn, sore throat, dizziness, and headache.

DOSAGE AND ADMINISTRATION

Adult Dosage

The standard adult dose of 3 Gm. (six tablets) is usually ingested with water in the evening after a fat-free dinner, about 14 hours before the cholecystography. In some instances, double doses of 6 Gm. (12 tablets) may be ingested, e.g., for duct visualization or for some repeat examinations made over 7 days later. Many radiologists prefer that the patient take a laxative (e.g., castor oil) about four to six hours before ingesting Telepaque. (See also Repeat Examination.)

HOW SUPPLIED

Tablets of 500 mg., envelopes of 6 tablets (3 Gm., the standard adult dose) in boxes of 5 and 25 envelopes; also bottles of 500 tablets with dispensing envelopes.

Write for illustrated booklet on Telepaque.

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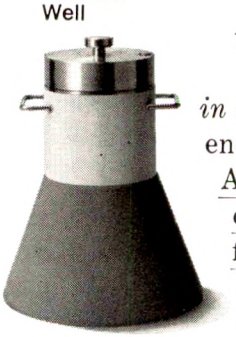
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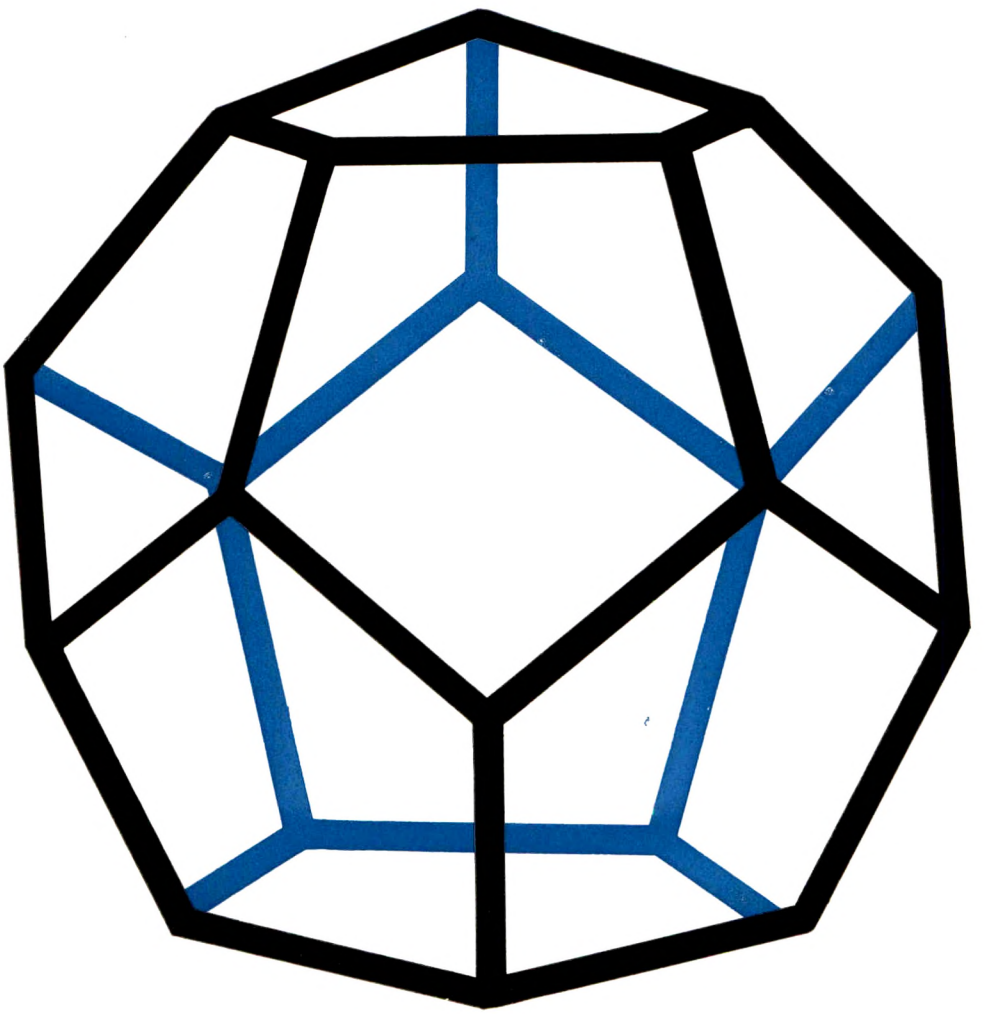
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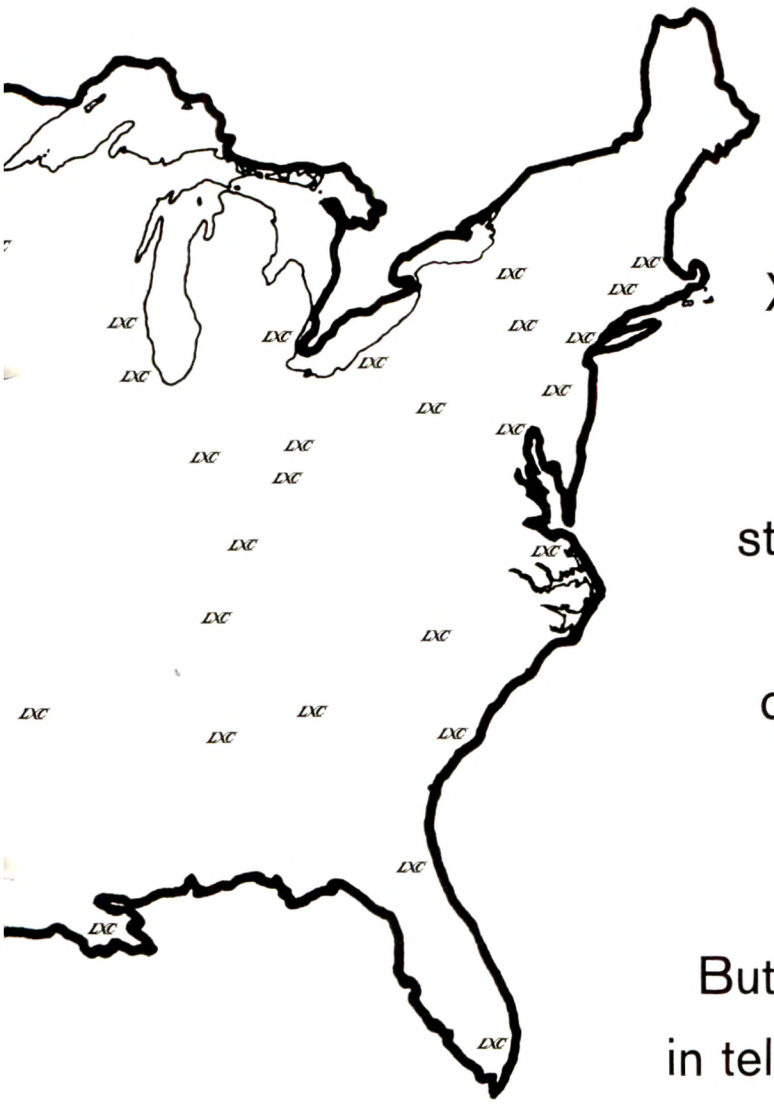
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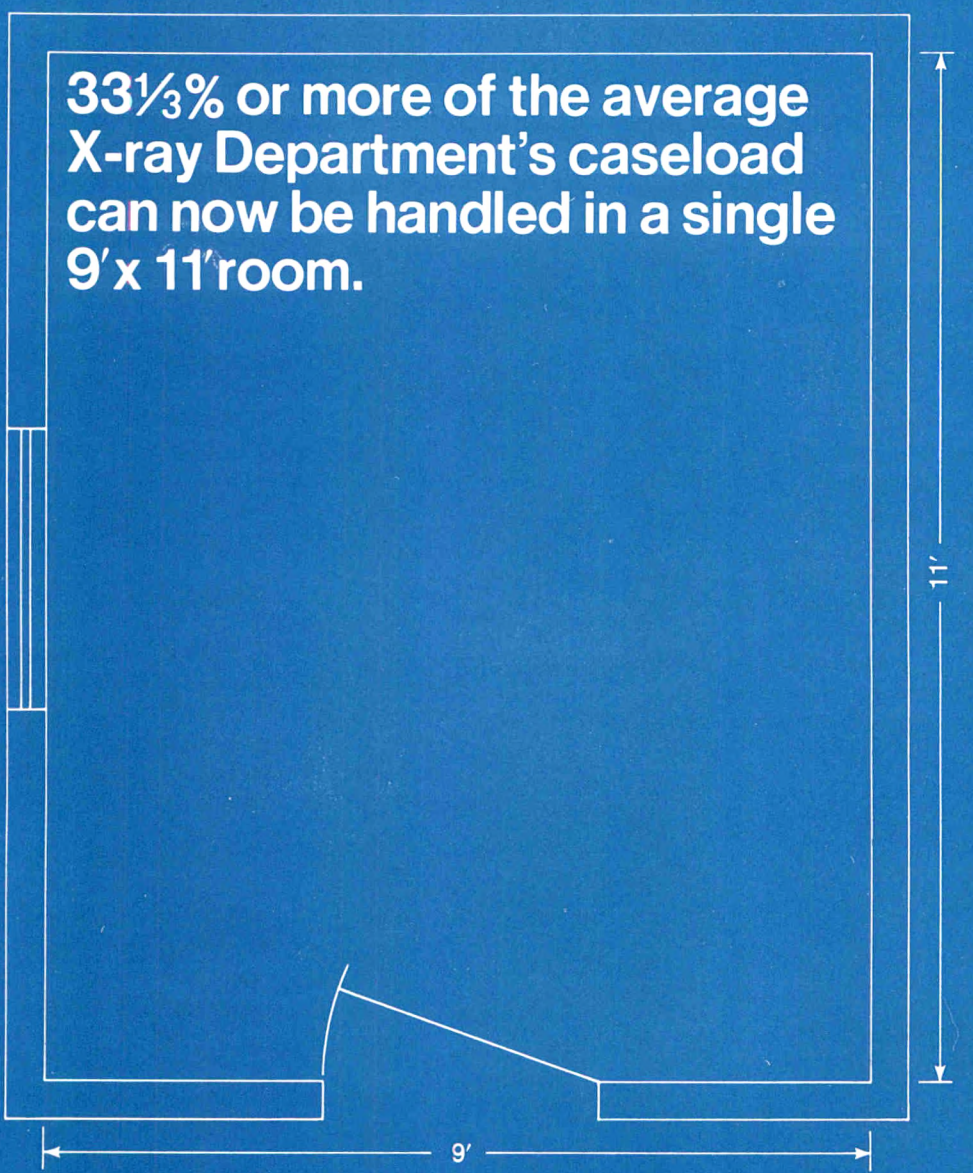
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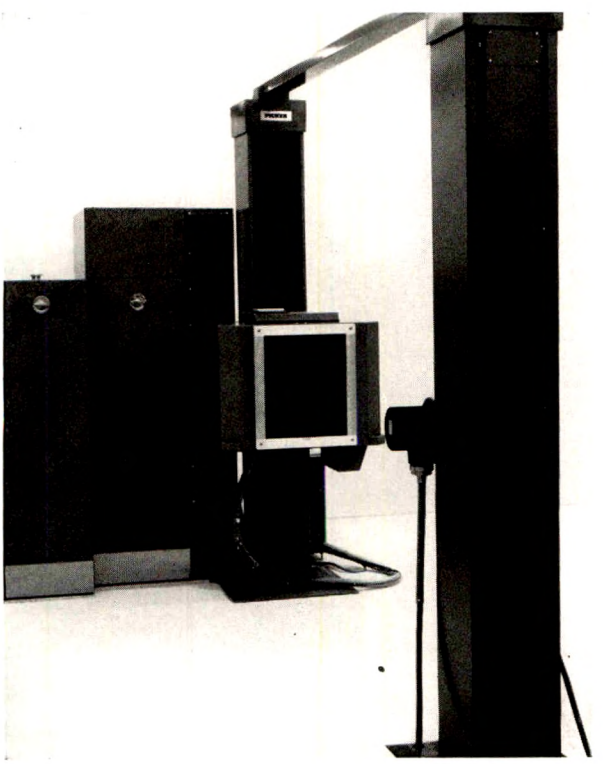
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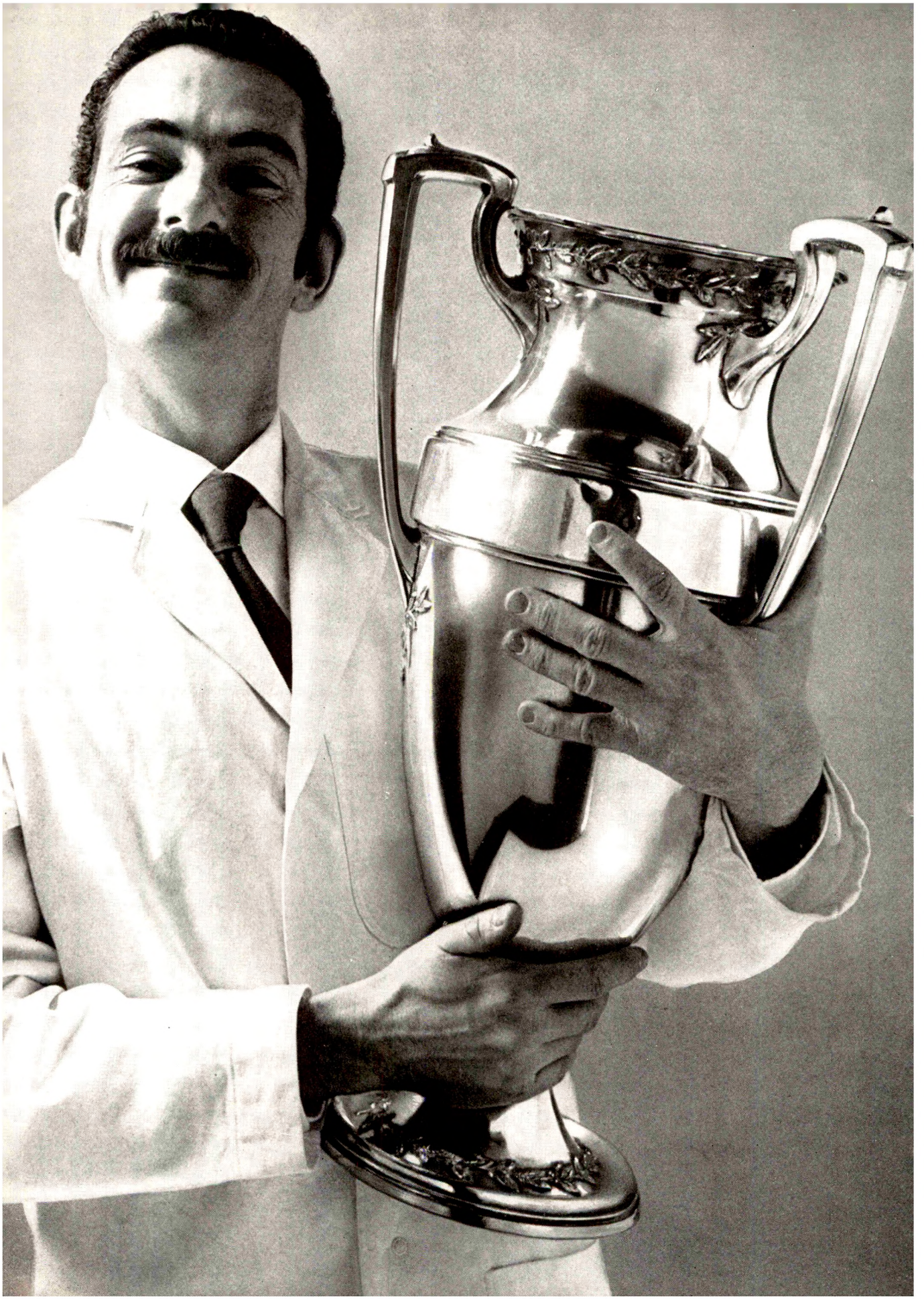
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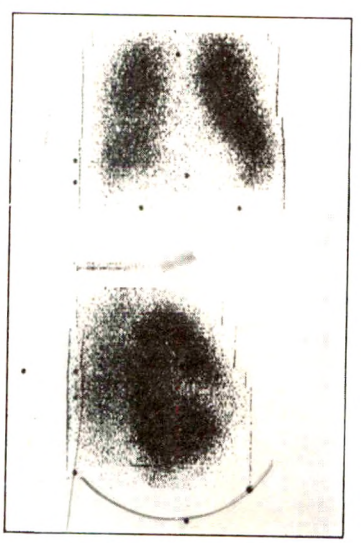
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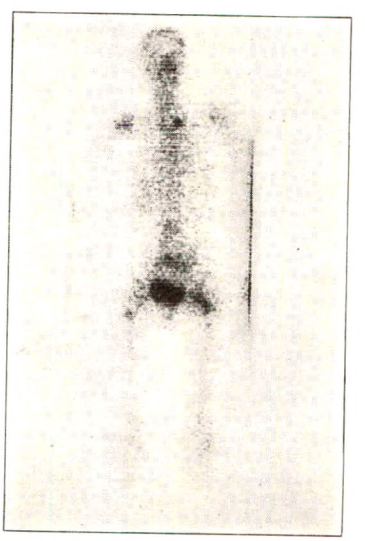
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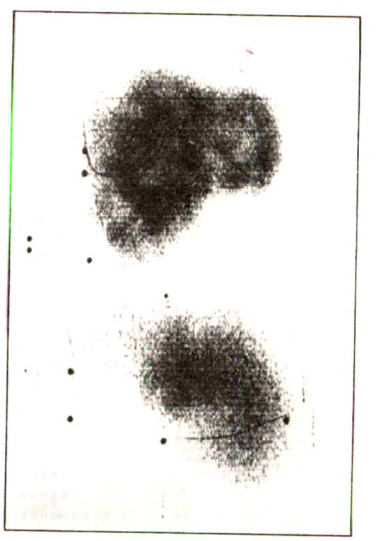
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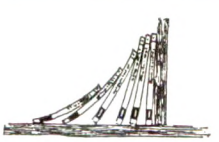
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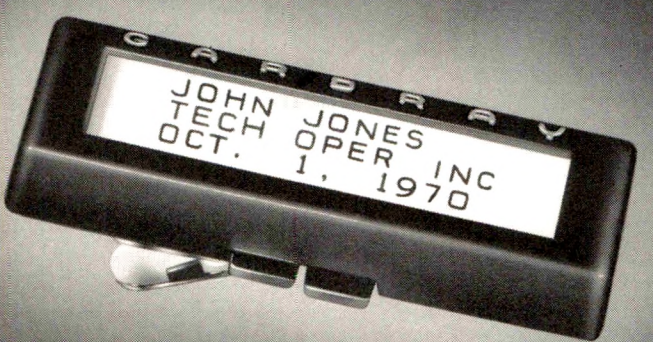


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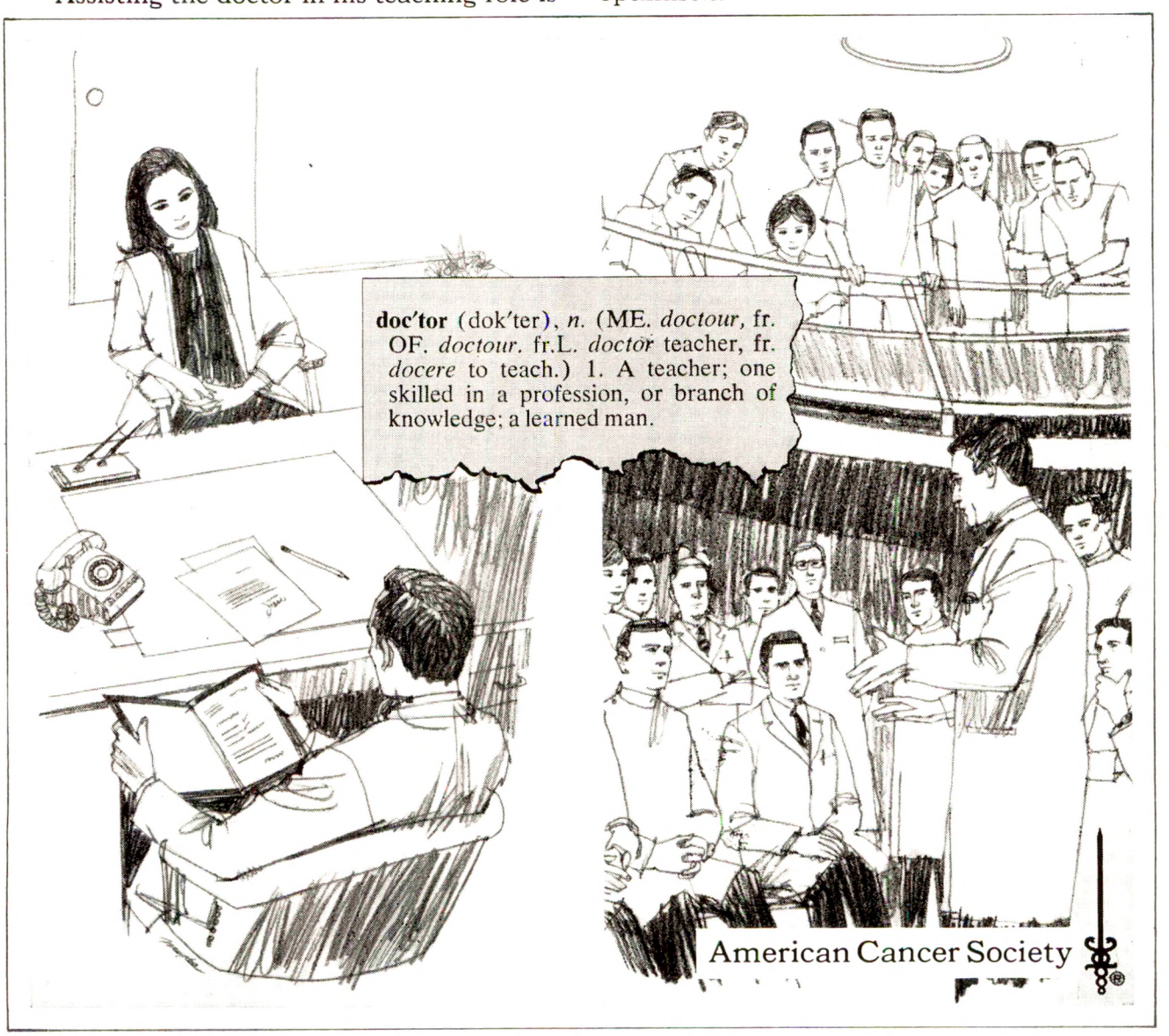
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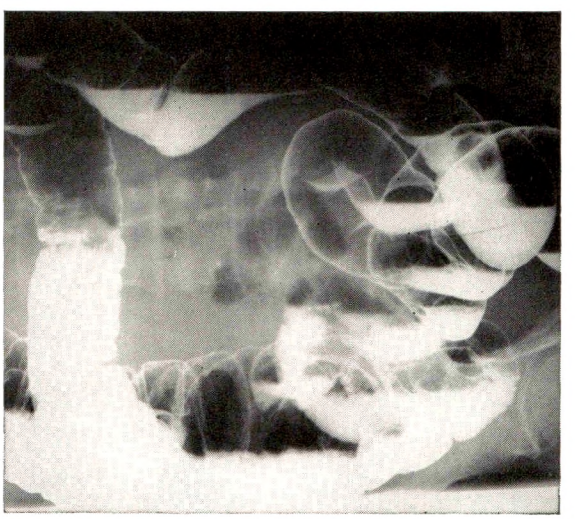
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*Bryk, D. and Roska, J. C.: Radiology 92:832, Mar., 1969.



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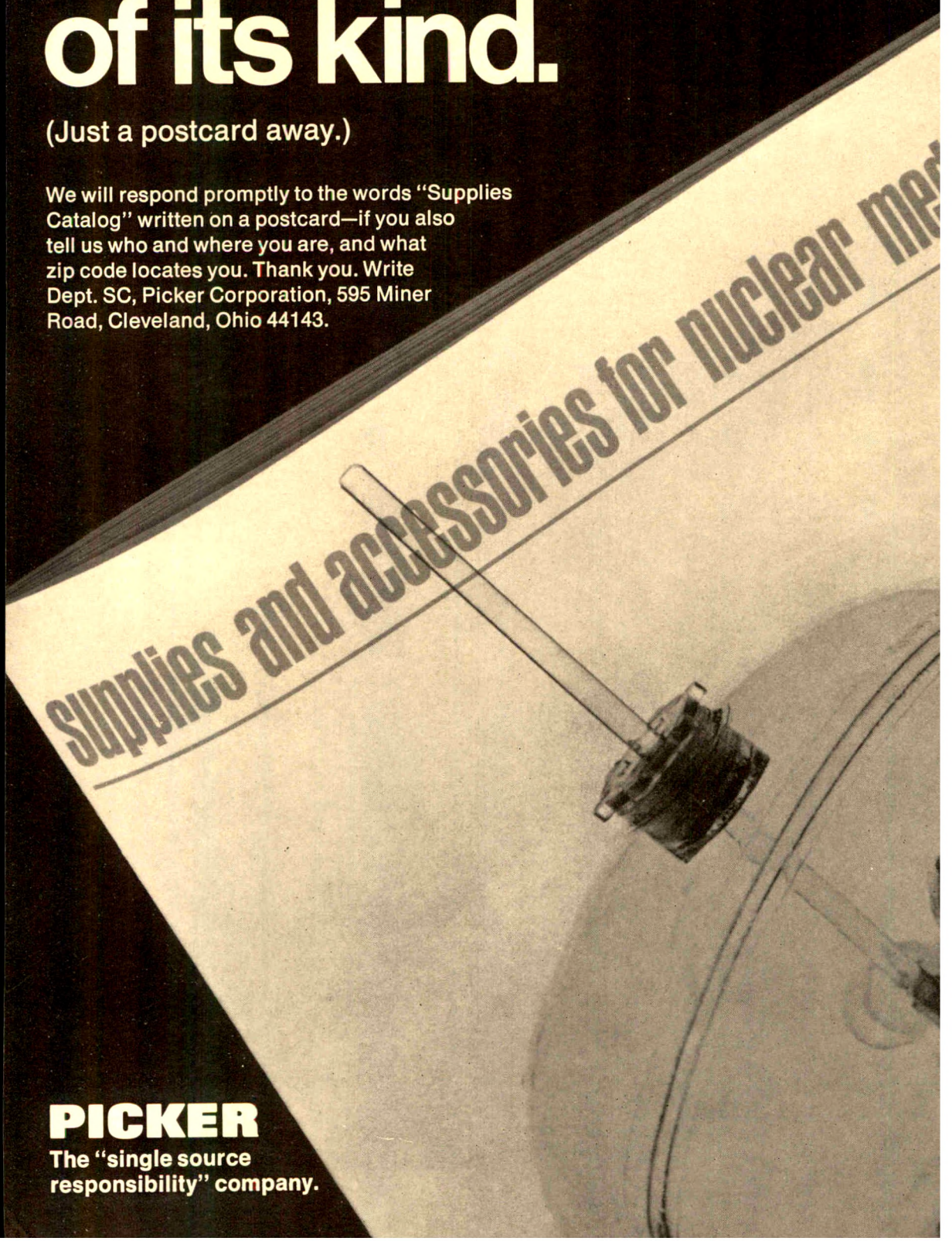
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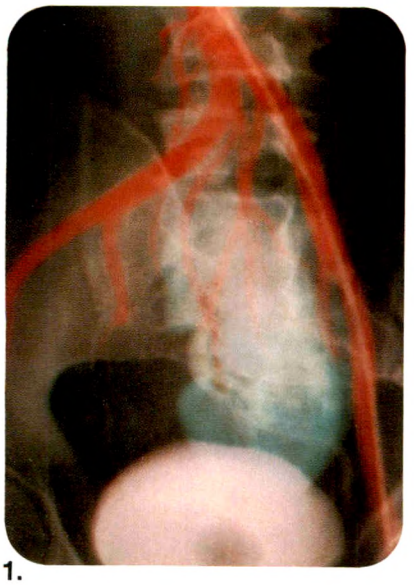
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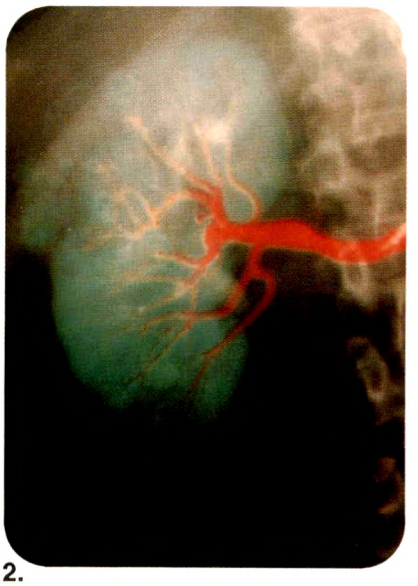
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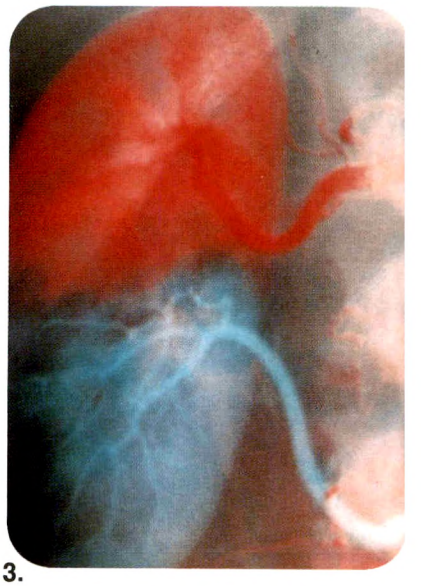
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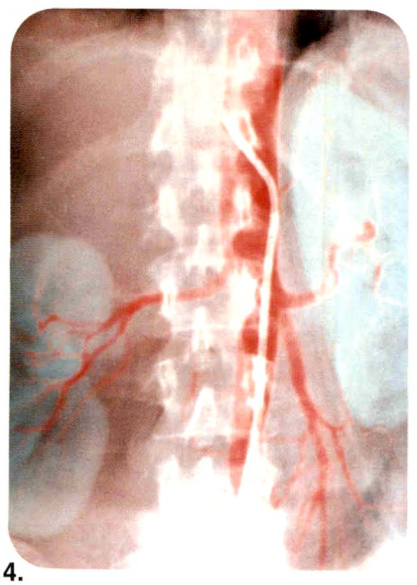
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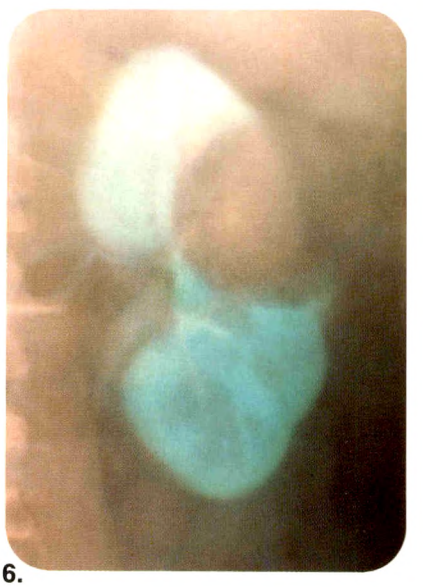
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Warning: Do not use Hypaque sodium for myelography. Injection of even a small amount into the subarachnoid space may produce convulsions and result in fatality.

General and Specific Precautions: Caution is advised in patients with a history of bronchial asthma, other allergic manifestations, or of sensitivity to iodine; these conditions represent a special risk, though not an absolute contraindication. To avoid or minimize possible allergic reactions, premedication with antihistamines may be considered. **Benadryl® (brand of diphenhydramine hydrochloride) in the same syringe with Hypaque may result in precipitation.** A test dose of contrast medium is not entirely reliable. Severe reactions, including fatalities, have occurred with a test dose as well as with the full diagnostic dose.

Caution is advised in patients with severe cardiovascular disease. In patients with impaired renal function and cardiovascular disease, drip infusion pyelography imposes a sudden osmotic and sodium load which can precipitate congestive heart failure. Contrast media injected into arteries or veins can promote sickling of red cells in susceptible individuals. In patients with reduced renal function, repeat of excretory or retrograde pyelography should be delayed at least 48 hours to avoid temporary suppression of urine. In preparation for excretory urography of patients with multiple myeloma, partial dehydration is not recommended, since this may predispose to the precipitation of myeloma protein in the kidney tubules. When thyroid function studies (PBI and 24-hour radioiodine uptake levels) are indicated, they should be performed *prior* to radiographic studies or several days afterwards, to avoid inaccurate results.

General and Specific Adverse Reactions: Adverse reactions, usually of a minor nature, have occurred in 10-14 per cent of patients who have received Hypaque intravenously. Reactions due to *faulty technique* include hematomas and ecchymoses, following extravasation from the vein, and pyrogenic reactions. *Hemodynamic reactions* include vasodilatation with flushing, hypotension and, rarely, vein cramp or thrombophlebitis. Serious *cardiovascular reactions* include rare cases of cardiac arrhythmias (e.g., ventricular fibrillation), shock, and cardiac arrest. Transient proteinuria may occur occasionally following the injection of radiopaques and, rarely, oliguria and anuria have been reported secondary to a hypotensive reaction. *Allergic reactions* include asthmatic attacks, nasal and conjunctival symptoms, cutaneous reactions such as urticaria and, rarely, anaphylactic shock, sometimes with fatal outcome. Severe reactions may also be manifested by signs and symptoms relating to the *respiratory system* (dyspnea, cyanosis, pulmonary or laryngeal edema), or to the *nervous system* (restlessness, confusion, or convulsions). *Other reactions* include nausea, vomiting, excessive salivation, anxiety, headache, and dizziness. Infrequently, "iodism" (salivary gland swelling) from organic compounds appears two days after exposure and subsides by the sixth day. Reactions to drip infusion pyelography may not appear until some hours after the examination.

ANGIOGRAPHY (PERIPHERAL ARTERIOGRAPHY AND VENOGRAPHY)

Precautions *(See also General Precautions):* Extreme caution is advised in considering peripheral arteriography in patients suspected of having thromboangiitis obliterans (Buerger's disease). Any procedure (even needling or insertion of a catheter) may induce a severe arterial and/or venous spasm. Caution is also advisable in patients with severe ischemia associated with ascending infection.

Adverse Reactions *(See also General Adverse Reactions):* In addition to those listed under General Adverse Reactions, are those due to arterial trauma during the procedure (i.e., needling, insertion of catheter, subintimal injection, perforation, etc.) as well as to the hypertonicity or effect of the medium; also reported are transient arterial spasm, extravasation, hemorrhage, hematoma with tamponade, injury to nerves proximal to artery, thrombosis (rare in venography, if vein is irrigated following injection), dissecting aneurysm, arteriovenous fistula, (e.g., with accidental perforation of femoral artery and vein during needling), transient leg pain from contraction of calf muscles in femoral arteriography; transient hypotension after intraarterial (brachial) injection, and brachial plexus injury with axillary artery injections.

CEREBRAL ANGIOGRAPHY

Inasmuch as cerebral angiography is a highly specialized procedure requiring the use of special techniques, it is recommended that Hypaque sodium (brand of sodium diatrizoate) be used for this purpose only by persons skilled and experienced in carrying out the procedure.

Contraindication: Carotid angiography should be avoided during the progressive period of a stroke because of the increased risk of cerebral complications.

Precautions *(See also General Precautions):* Select patients for this procedure with care. Use the 50 per cent solution with caution in extreme senility (but not old age *per se*), advanced arteriosclerosis, severe hypertension, and cardiac decompensation.

The diagnostic value of the procedure, according to many authorities, when employed early enough in locating lesions amenable to surgery, may outweigh the added risk to patients who have recently experienced cerebral embolism or thrombosis (stroke syndrome). A small number of postangiographic fatalities, including progressive thrombosis, have occurred in which the procedure did not appear to play a role. Patients with severe cerebrovascular disease may be examined primarily by indirect methods of angiography.

Care should be exercised to avoid contaminating catheters, syringes, needles, and contrast media with glove powder or cotton fibers.

Angiography is hazardous in subarachnoid hemorrhage. In migraine, the procedure can be hazardous because of ischemic complications, particularly if performed during or soon after an attack.

Adverse Reactions *(See also General Adverse Reactions):* The major sources of complications are faulty technique, occlusive atherosclerotic vascular disease, repeated injections of the contrast media, and higher doses than those recommended.

Untoward reactions are mostly mild and transient, although permanent visual field defects and deaths have been reported.

Vascular reactions: flushing, vessel spasm, thrombophlebitis, and cutaneous petechiae.

Neurologic complications: transient cerebral blindness, neuromuscular disorders, convulsions, coma, hemiparesis, unilateral dysesthesias, visual field defects, aphasia, and respiratory difficulties.

AORTOGRAPHY

Precautions *(See also General Precautions):* Special caution is advised to avoid inadvertent intrathecal injection when using the translumbar technique. The incidence and severity of reactions or complications that may occur are influenced by: the care and experience with which the procedure is performed; the amount and type of medium used; the age and condition of the patient; and the premedication and anesthesia used. Since aortography is not without some danger, it should be performed only by those experienced in the technique. Wherever possible, repeated injections of the solution during a single study should be avoided.

Adverse Reactions *(See also General Adverse Reactions):* Renal damage and shutdown; neurologic complications such as transverse myelitis or paraplegia; cardiovascular complications such as shock, cardiac arrest or failure, coronary occlusion, arterial thrombosis, embolism, and perforation of vessels; extravasation or hemorrhage from the puncture site or retroperitoneal catheter perforation; necrosis of the intestinal wall; acute pancreatitis; deaths; diffuse cutaneous petechiae; subintimal injection and aortic dissection.

INTRAOSSEOUS VENOGRAPHY

See General Precautions and Adverse Reactions. A general anesthetic is sometimes necessary since the method is painful. Occasionally, extravasation of the contrast medium from the needle into the soft tissue may occur.

DIRECT CHOLANGIOGRAPHY

Precaution *(See also General Precautions):* This procedure should be used with caution in the presence of acute pancreatitis, injecting no more than 5 to 10 ml. without undue pressure.

Adverse Reactions *(See also General Adverse Reactions):* These may be attributed to injection pressure or excessive volume of the medium, resulting in overdistention. Such pressure may produce a sensation of epigastric fullness, followed by moderate pain in the back or right upper abdominal quadrant, which will subside when injection is stopped.

Some of the medium may enter the pancreatic duct and cause a transient serum amylase elevation 6 to 18 hours later, without apparent ill effects. Pancholangitis resulting in liver abscess has been reported. Occasionally, nausea, vomiting, fever, and tachycardia have been observed.

HYSTEROSALPINGOGRAPHY

Contraindications: Menstruation, active or imminent; infection in any part of the genital tract, including the external genitalia; pregnancy, known or suspected. Use not advised for six months after end of pregnancy, or 30 days after conization or curettement.

Precaution *(See also General Precautions):* Caution should be exercised in patients with known or suspected carcinoma to avoid possible spread of the lesion by the procedure.

SPLENOPORTOGRAPHY

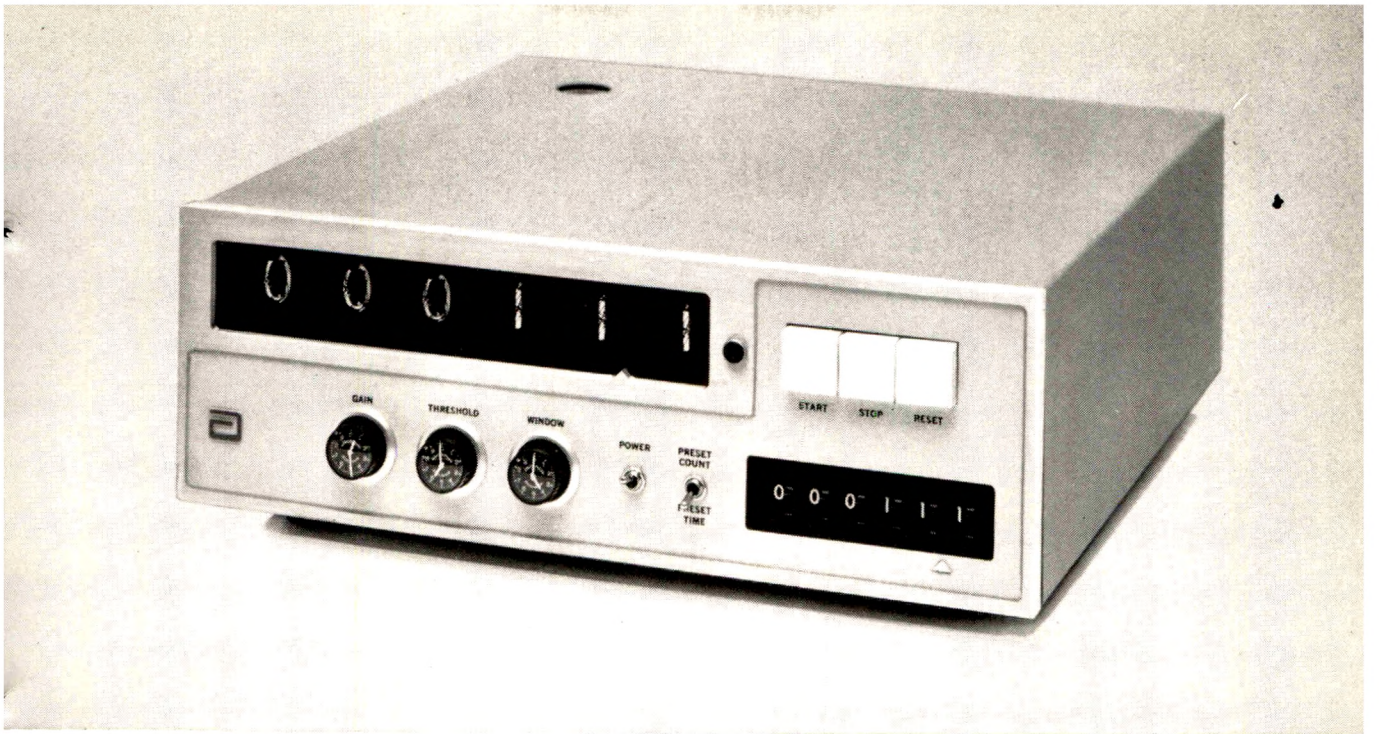
Precautions *(See also General Precautions):* Procedure should be performed with caution on patients with blood dyscrasias, a tendency to severe bleeding, or a spleen recently become tender and palpable.

Adverse Reactions *(See also General Adverse Reactions):* Persistent hemorrhage with hemoperitoneum has been reported, and, in some instances, hemorrhage has required splenectomy.

How Supplied: Ampuls of 30 ml., boxes of 10 and 25. Boxes of 25—1 ml. ampuls for testing sensitivity are available upon request. Vials of 20 and 30 ml., rubber stoppered, boxes of 10 and 25. Vials of 50 ml., rubber stoppered, boxes of 10. Each vial contains sufficient excess to permit withdrawal of 1 ml. for testing sensitivity.

If the solution is chilled, crystals may form but will readily dissolve if the ampul or vial is placed in moderately hot water before use. Cool to body temperature before injecting.

Winthrop PIONEERS IN PRODUCTS FOR RADIOLOGIC DIAGNOSIS
Winthrop Laboratories, New York, N.Y. 10016



Don't buy Nuclear Instruments—invest in them! The Abbott Modular Concept lets you do just that.

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When you're ready to do *in vivo* studies such as thyroid uptakes, you can add an External Probe, Collimators, and the Mechanical Medical Stand. What's more, this total system actually costs you thousands of dollars less than any competitive hardware.


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Nuclear Instruments You Can Count On

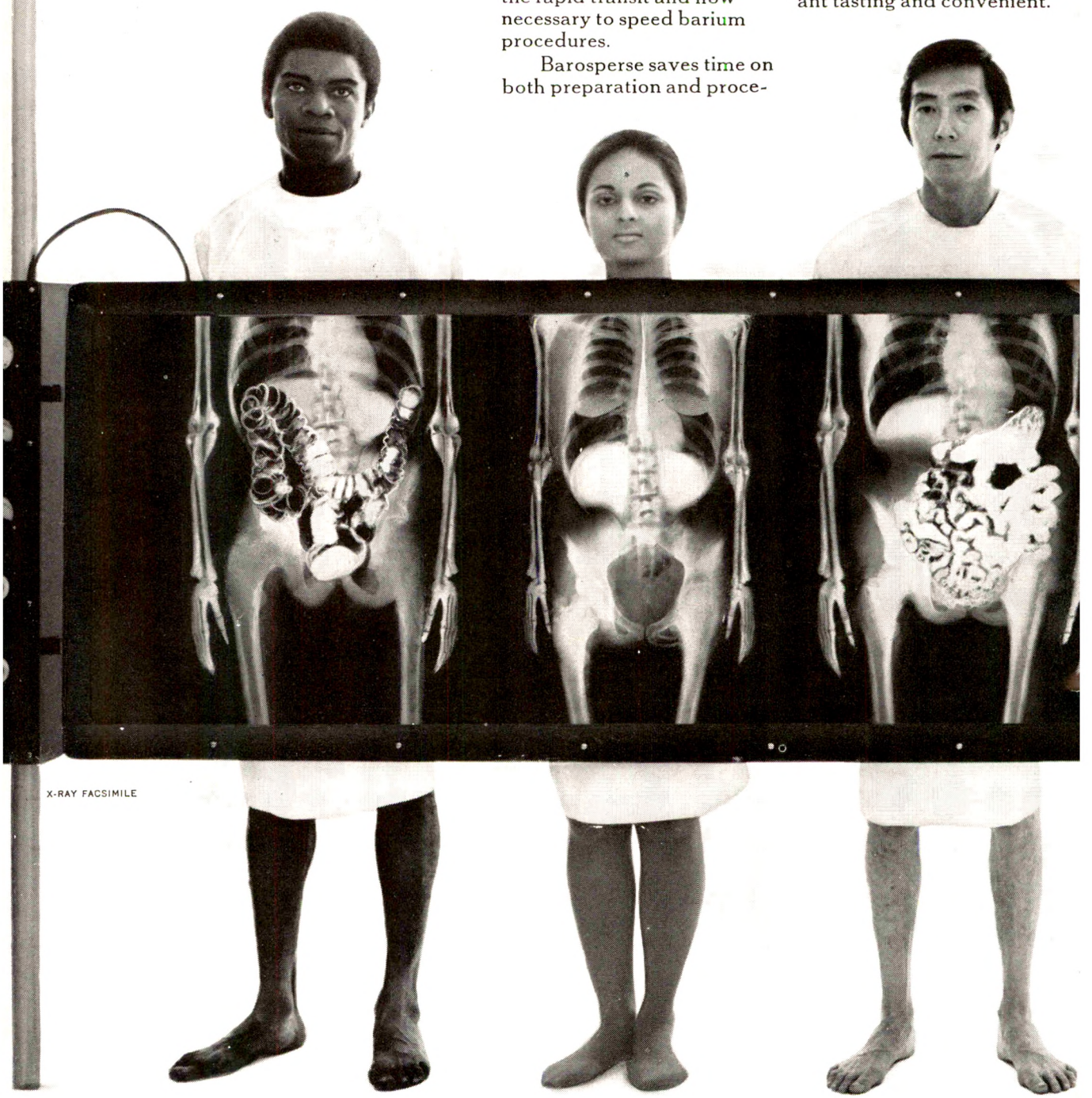
Vertretung für Europa: Labor-Service GmbH, Abt. Radiopharmazeutika, 6236 Eschborn/Ts, Germany, Postfach 1245

To be more exact, Barosperse has been used in millions of G.I. examinations in more than 30 countries around the world. Which makes Barosperse the most widely accepted of all barium sulfate formulations by far. And with good reason.

With Barosperse, you get films of unexcelled diagnostic quality and definition of detail. Used for upper or lower G.I. series, this formulation has the fluidity and coatability to fill in the folds and convolutions of the entire G.I. tract. The opacity to provide sharp, accurate, readable visualizations. And the rapid transit and flow necessary to speed barium procedures.

Barosperse saves time on both preparation and procedure. It mixes with water and is dispersed in 20 to 30 seconds. Barosperse requires minimum fluoroscopic time resulting in minimum radiation. Superb small bowel delineation is normally accomplished in 30 to 90 minutes.

Barosperse is both pleasant tasting and convenient.



X-RAY FACSIMILE

Barosperse® gets into more G.I. tracts in more countries

(barium sulfate formulation)

Most patients readily accept the smooth, pleasantly flavored mixture; and both single dose and bulk packaging permit greater flexibility and convenience in the lab.

So when you think of barium sulfate, think of the formulation more radiologists and gastroenterologists in more than 30 countries use. Barosperse.

Barosperse® (Barium Sulfate Formulation) is available in the following convenient packages.

Barosperse Esophageal Cream in 100-gm and 320-gm tubes.

The Barosperse Single Dose Oral Unit.

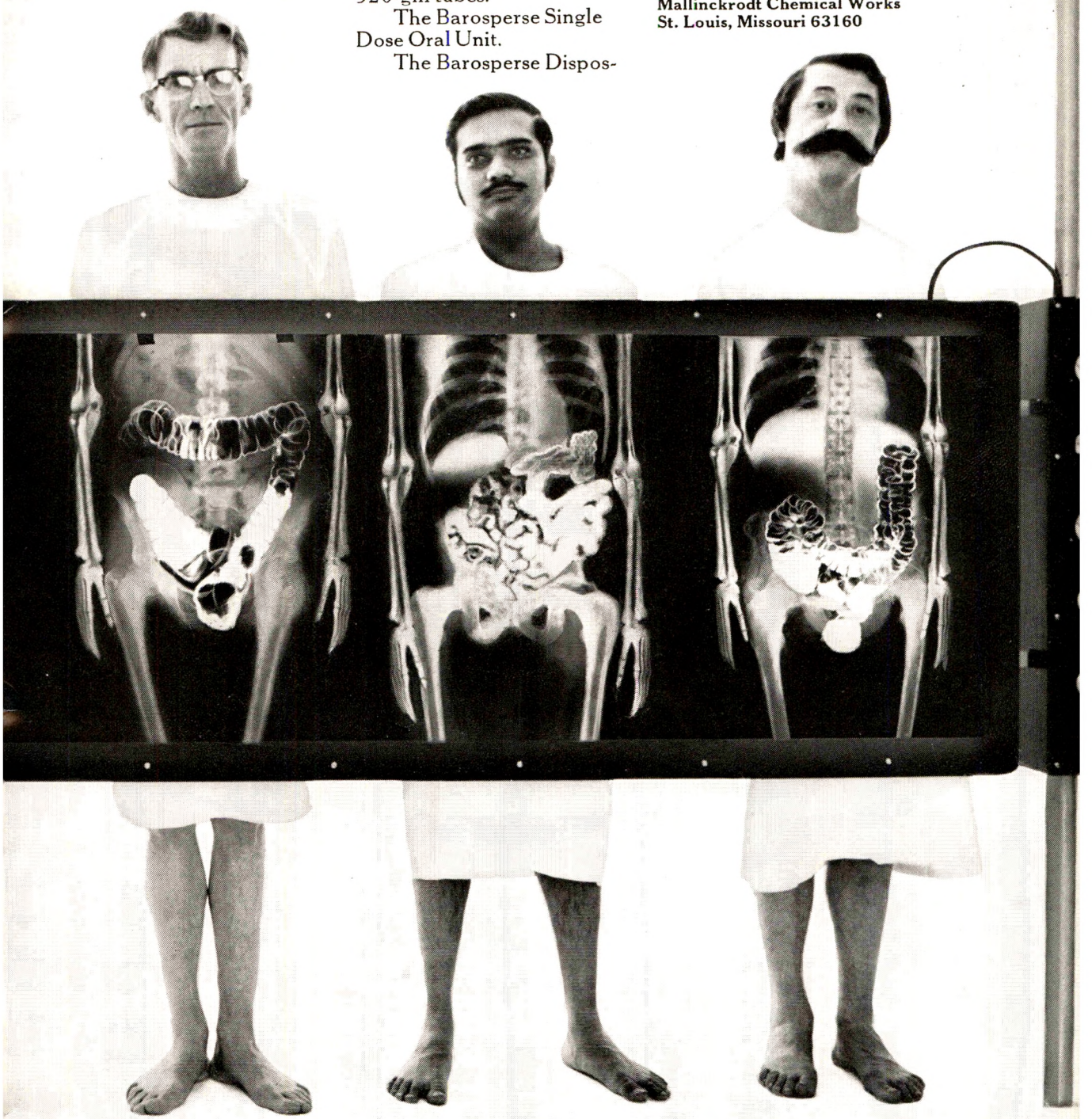
The Barosperse Disposable Barium Enema Unit.

The Barosperse Disposable Direct Air Contrast Barium Enema Unit.

Barosperse, for bulk use, in 10 lb., 25 lb., and 100 lb. drums.

Mallinckrodt Pharmaceuticals

Mallinckrodt Chemical Works
St. Louis, Missouri 63160

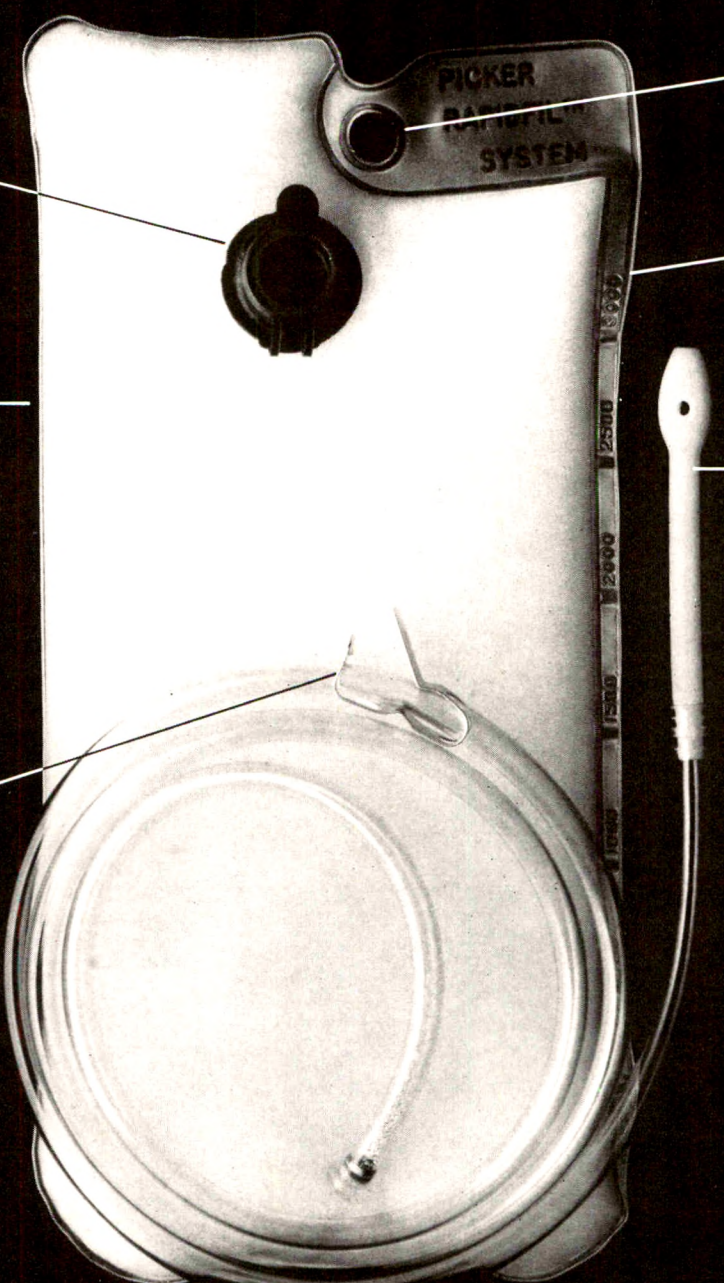


than any other contrast medium of its kind.



60 seconds gets it ready and 0 seconds gets it gone.

(The Picker Rapidfil™ disposable enema system with Micropaque®)



Snap closure: opens easily for filling, closes securely, doesn't leak.

Retrograde filling system also available.

Tough, tough heavy gauge vinyl bag: takes abuse without leaking or tearing.

Another thoughtful feature: bag impregnated with a pleasant masking scent.

Liquid level easily visible.

Spring metal positive control pinch-off.

Centered support hole: easy to hang, automatically balanced.

3000 + cc capacity with helpful 500 cc graduations.

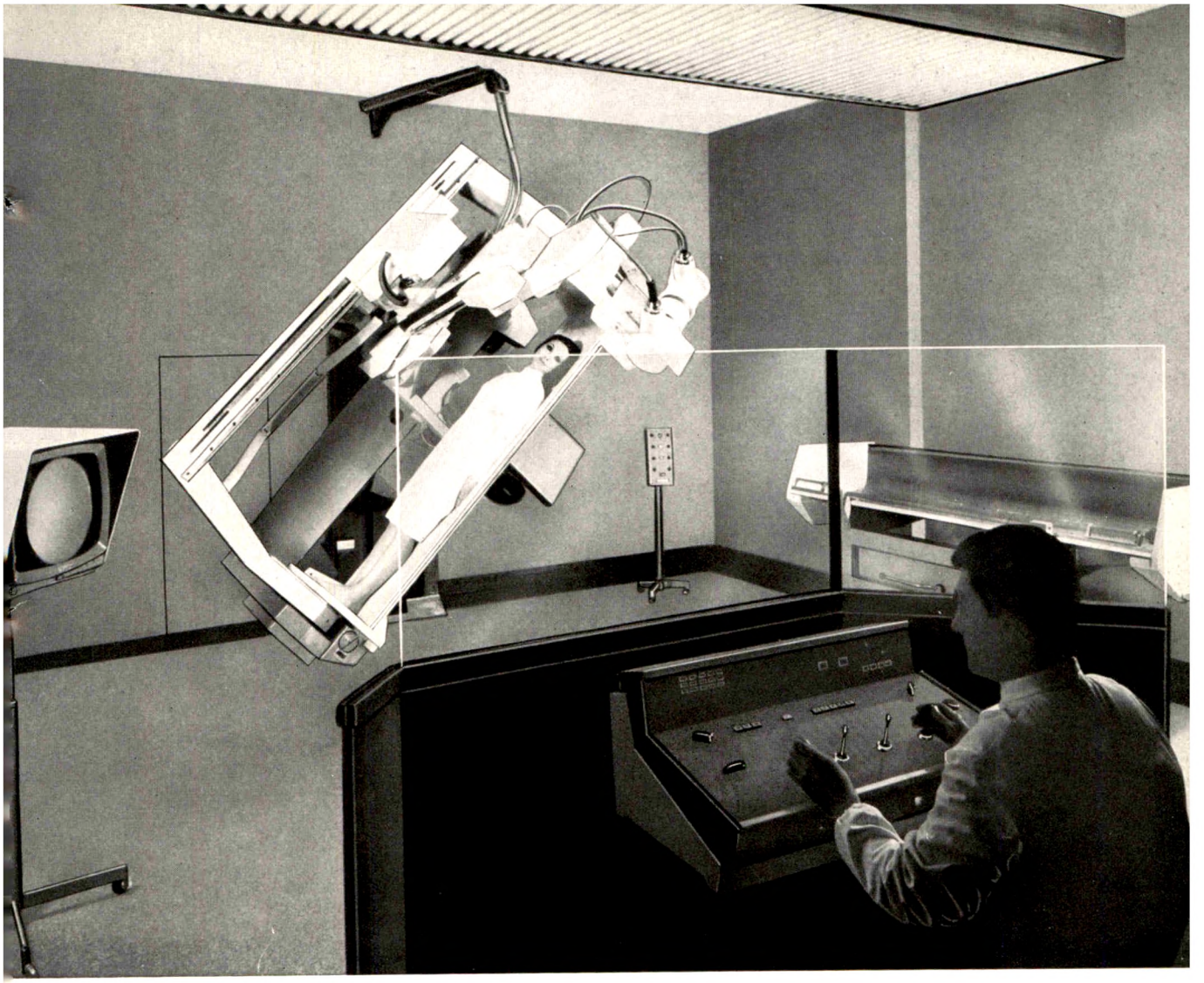
Wide choice of disposable enema tips.

Pre-filled with Micropaque® USP barium sulfate formulation. Defines mucosal patterns by coating rather than filling colon.

The Picker Rapidfil™ disposable enema system with Micropaque® is a simple-to-use, predictably dependable system which eliminates several problems: the risk of contamination and the cost and unpleasantness of cleaning. Ask your Picker representative for a sample, or write Picker Medical Products Division (Dept. L), 595 Miner Road, Cleveland, Ohio 44143.

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Universal remote controlled diagnostic table for television screening, radiography, tomography, with the patient in any position.

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RADIOIMMUNOASSAY– the long and short of it

Radioimmunoassay offers one of the most sensitive methods available for testing in medicine. This is because it can be used to measure physiological levels of protein hormones in millimicrogram to micromicrogram quantities. But obstacles in developing antibodies (an essential part of the test) have limited the use of radioimmunoassays.

Now, Abbott has helped this situation by introducing a complete radioimmunoassay kit — HGH-125 Imusay Kit.

With this kit, the quantitative determination of human growth hormone in serum becomes a practical matter. Children, whose growth rates are suspect, can be checked for a hypopituitary or an acromegalic condition. Since this is an *in vitro* test, the child receives no radioactivity.

The HGH-125 Imusay Kit introduces tomorrow's diagnostic tools today — and this is only the beginning. Abbott is now working on additional radioimmunoassay kits for other hormones.

TM—Trademark.

007225

HGH-125 IMUSAY™ Kit

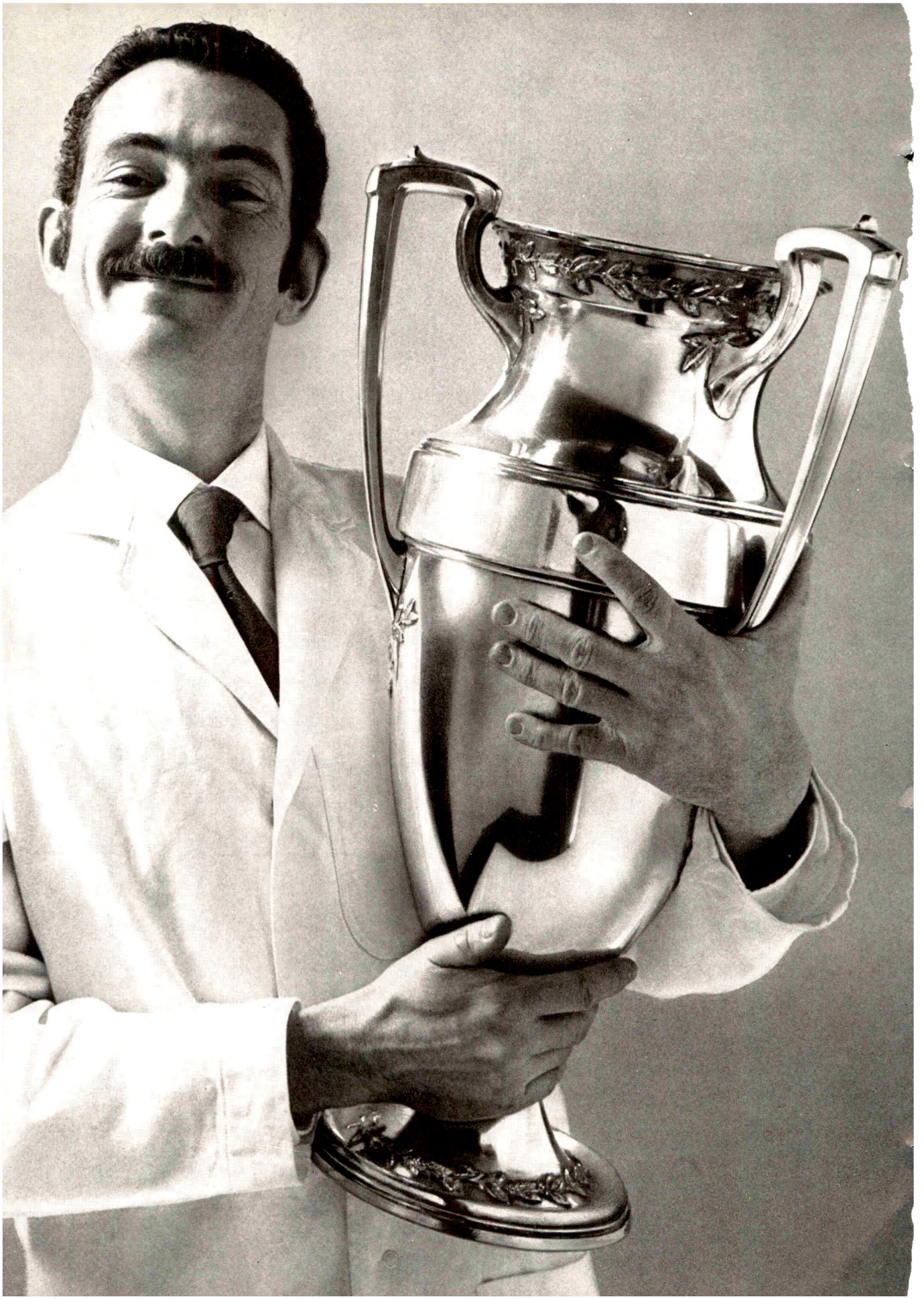
HGH IMMUNOASSAY KIT

ABBOTT LABORATORIES

North Chicago, Illinois 60064

World's Leading Supplier of Radio-Pharmaceuticals

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For prizewinning films, give FLEET® PHOSPHO®-SODA (the buffered laxative) prior to radiology. It's gentle and thorough. Really gets the colon clean—a key to great pre- and postevacuation colon x-rays. And PHOSPHO-SODA is virtually free from discomfort and irritation. *Regular* or *flavored,* it's a great diagnostic aid! **Warning:** Do not use when nausea, vomiting or abdominal pain is present. Frequent or prolonged use of this preparation may result in dependence on laxatives.

And when a large-volume enema is preferred, there's FLEET® BAGENEMA. Complete with liquid soap packet, prelubricated rectal tip, and large absorbent pad. The ultimate in convenience. Nothing to assemble. Nothing to clean. Just fill, hang, use and discard. Economical, too.

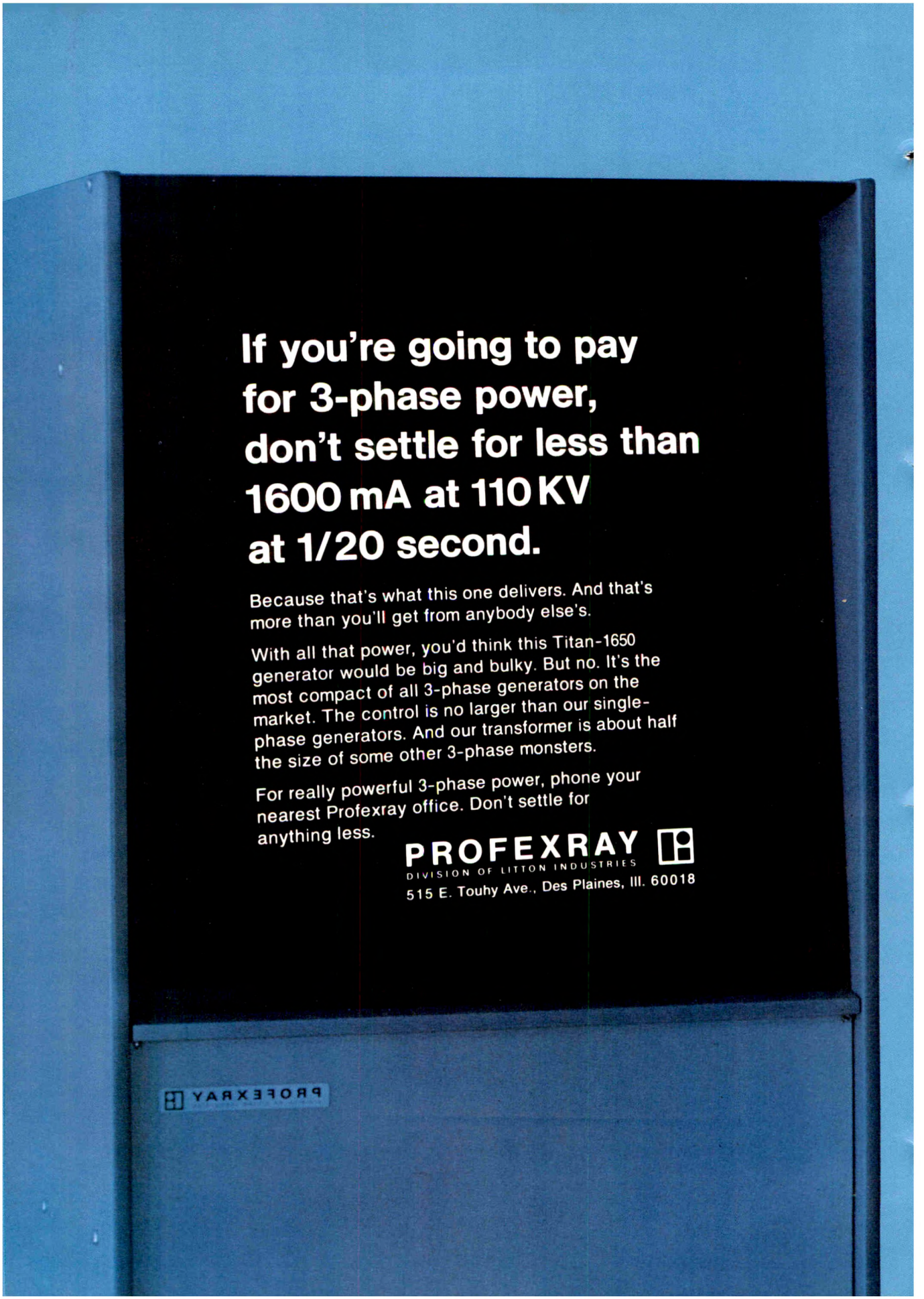
These can make the difference between good & great films



Fleet pharmaceuticals

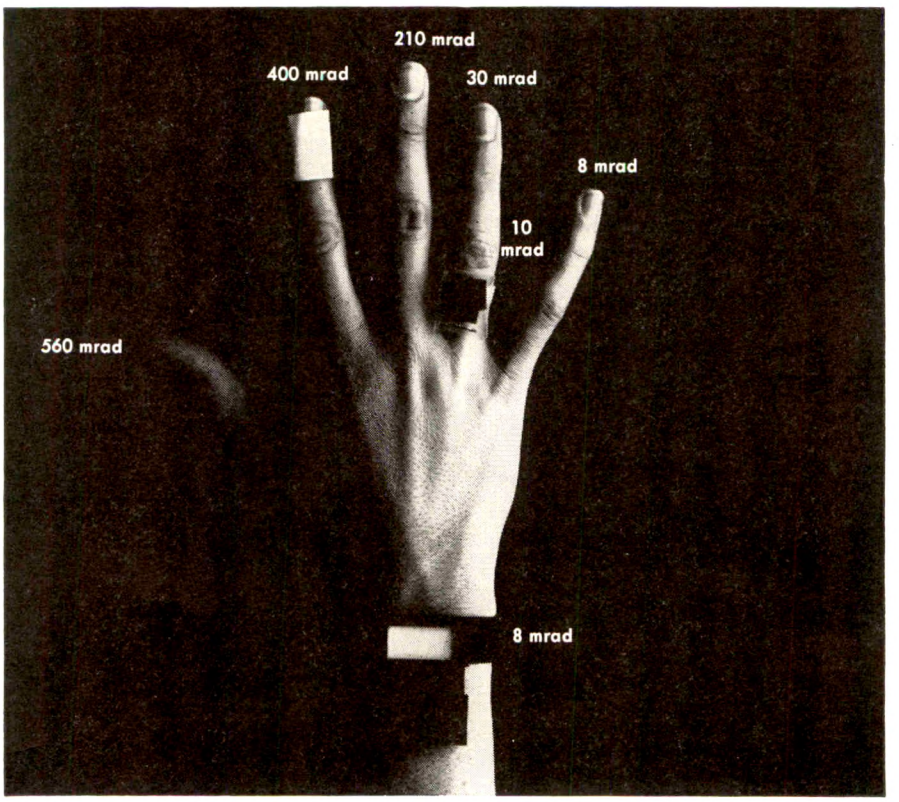
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If you're going to pay
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don't settle for less than
1600 mA at 110 KV
at 1/20 second.
Because that's what this one delivers. And that's more than you'll get from anybody else's.
With all that power, you'd think this Titan-1650 generator would be big and bulky. But no. It's the most compact of all 3-phase generators on the market. The control is no larger than our single-phase generators. And our transformer is about half the size of some other 3-phase monsters.
For really powerful 3-phase power, phone your nearest Profexray office. Don't settle for anything less.
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5 GOOD REASONS WHY YOU NEED FINGERTIP DOSIMETRY.



When you hold a 1 mg radium needle for as little as five minutes, your thumb receives a maximum dose as high as 560 mrad. A conventional photographic film dosimeter, wrist or ring, measures less than 2% of this dose. Only Isotopes LiF Teflon Finger Dosimeters can measure the dose at the points of maximum exposure — your fingertips.

What's more these rugged, thin, flexible and tissue equivalent Finger Dosimeters, with a dose range from 50 mr to 10^5 rads, measure the total dose from beta, gamma and x-rays to the sensitive layer of the skin.

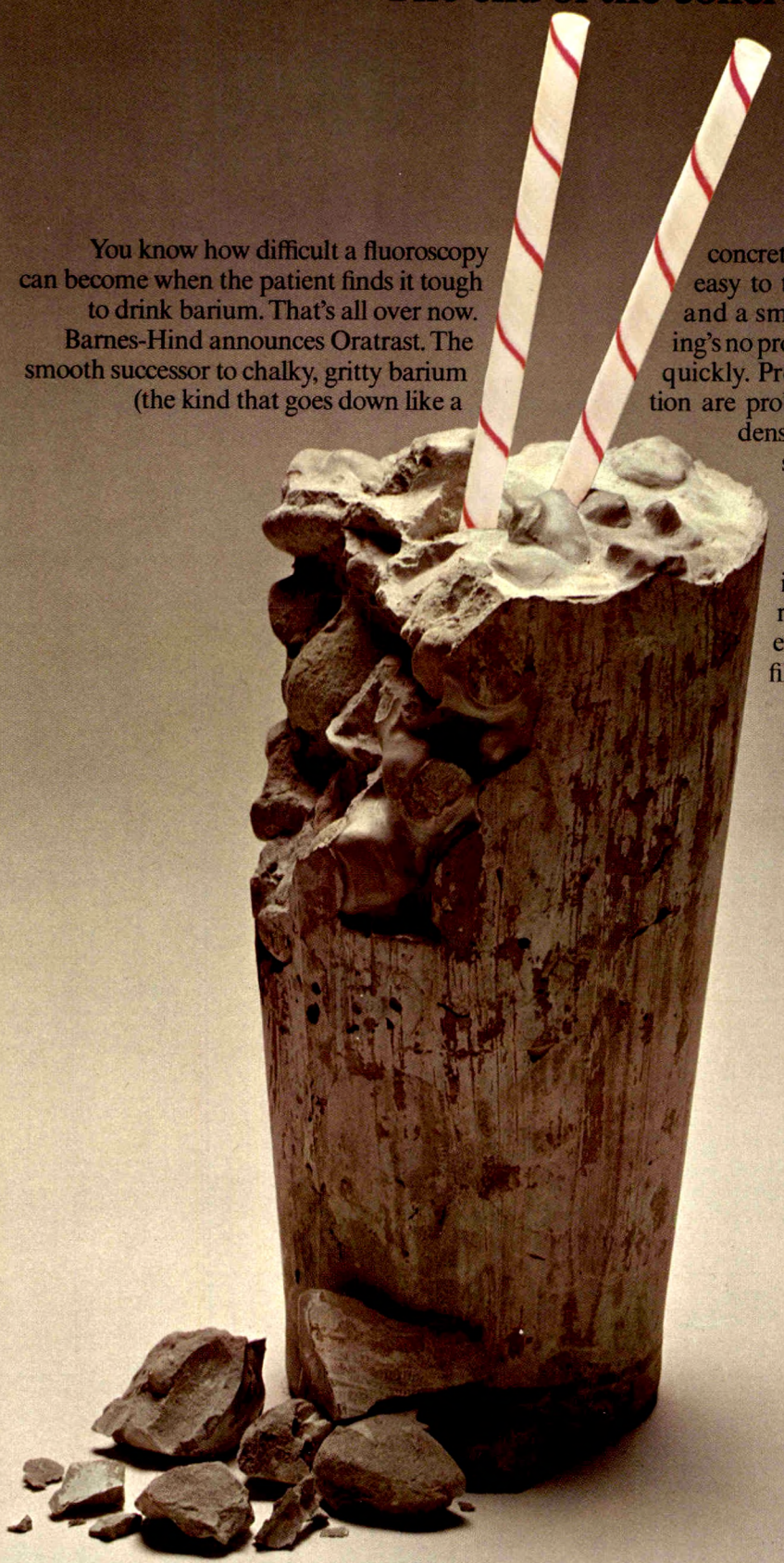
Reasons enough, we think, to write or call ISOTOPES for full information. 50 Van Buren Avenue, Westwood, N.J. 07675 (201) 664-7070.



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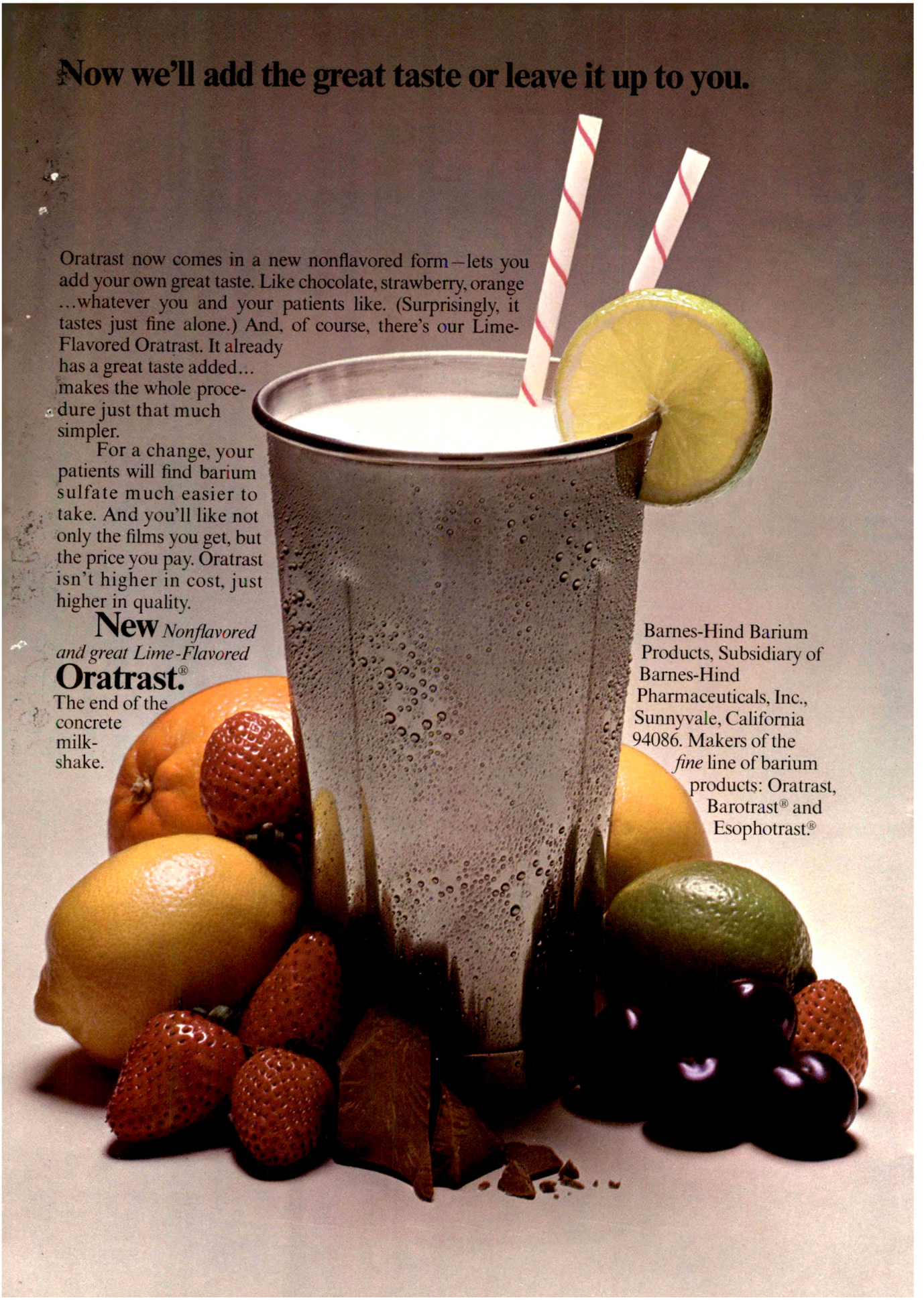
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The end of the concrete milkshake.



You know how difficult a fluoroscopy can become when the patient finds it tough to drink barium. That's all over now. Barnes-Hind announces Oratrast. The smooth successor to chalky, gritty barium (the kind that goes down like a concrete milkshake). Oratrast is easy to take...with a great taste and a smooth texture. And mixing's no problem. Oratrast disperses quickly. Precipitation and flocculation are problems of the past. High-density Oratrast has superior suspension qualities — provides good air contrast with prolonged uniform mucosal coating (yet maintains a superior radiolucent quality even when the stomach is filled). **Oratrast.®**

Now we'll add the great taste or leave it up to you.



Oratrast now comes in a new nonflavored form—lets you add your own great taste. Like chocolate, strawberry, orange ...whatever you and your patients like. (Surprisingly, it tastes just fine alone.) And, of course, there's our Lime-Flavored Oratrast. It already has a great taste added... makes the whole procedure just that much simpler.

For a change, your patients will find barium sulfate much easier to take. And you'll like not only the films you get, but the price you pay. Oratrast isn't higher in cost, just higher in quality.

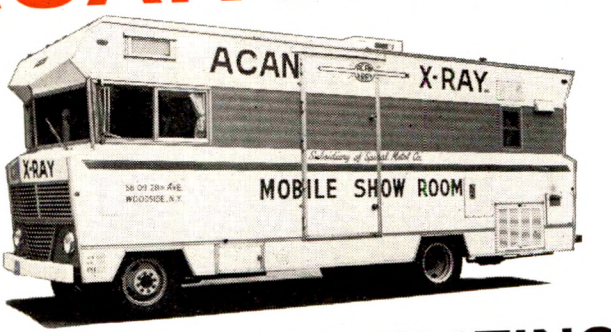
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ACAN X-RAY



IS DEMONSTRATING ACROSS THE NATION

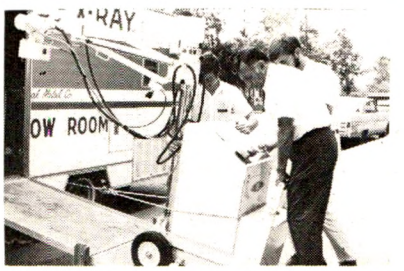
Acan's Mobile Show Room is traveling around the United States to demonstrate the Acan Model RC 1100 Mobile Type Condenser Discharge X-Ray Unit. This unique piece of equipment is designed to assure high speed, high quality radiographs.

Rather than talk about it, however, Acan is sending its Mobile Show Room on a tour of the nation to demonstrate the unit. You'll have an opportunity to see the RC 1100 in operation, and to have all your questions answered at first hand.

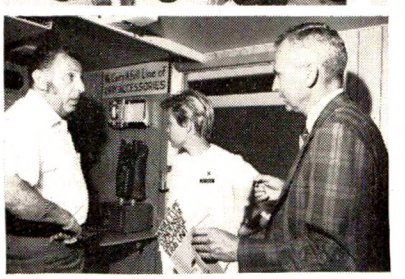
In addition to the Condenser Discharge X-Ray Unit, the Mobile Show Room will feature other Acan products including the Dri-Glov Protective X-Ray Glove Dryer, the Naca Self-Threading Compact Film Processor and the Acan Collimator.

Look for the Acan Mobile Show Room in your community. Meanwhile, if you'd like information about any Acan product or service, please let us know.

ACAN X-RAY
a subsidiary of
S.M. *Spiral Metal Company, Inc.*

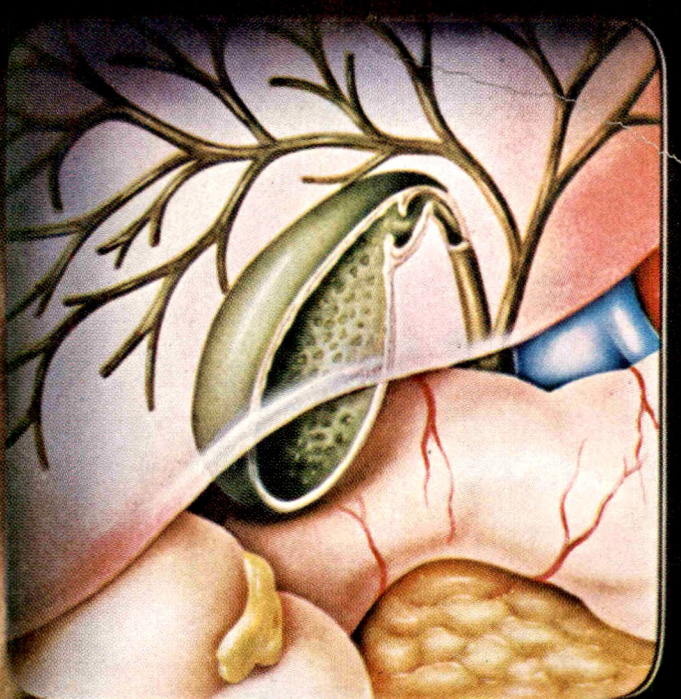






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A nonvisualized gallbladder...

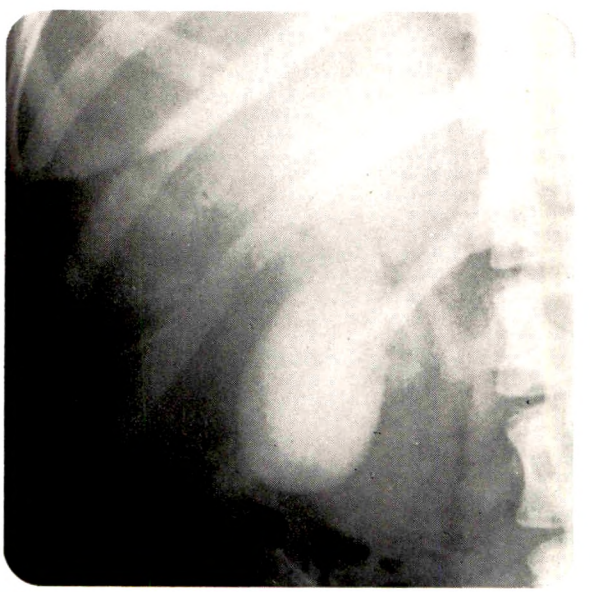


can often be visualized with Oragrafin® Calcium Granules

Calcium Ipodate



Cholecystogram 12 hours after routine dose of iopanoic acid.[1]



Cholecystogram 3 hours later after dose of 6 Gm. of calcium ipodate granules. Note filling of the common duct—from Root[1], discussion on the following page.

When surgery hangs on diagnostic films...

Calcium and sodium ipodate are rapidly absorbed from the gastrointestinal tract and begin to appear in the bile with striking rapidity. The abundant delivery to the liver and biliary system permits visualization even in a poorly functioning gallbladder. In addition, use of calcium ipodate granules permits same-day re-examination, should a second visualization be deemed necessary.

See next page for "Brief Summary."

Recent clinical study[1] demonstrated improved diagnoses

"A group of 114 patients with poorly filled gallbladders after routine administration of iopanoic acid were given 6 gm. of [calcium] ipodate granules immediately following their routine cholecystography. In 66 per cent of these patients opacification of the gallbladder was obtained within 3 hours, allowing a definite diagnosis. Of the remaining 33 per cent, one-half were ultimately proven diseased."[1] For one of the patients in this group, see x-ray pictures on previous page.

"It is apparent that many poorly visualized gallbladders can be rendered visible by the ingestion of ipodate granules. In most instances, definite diagnosis can be made within 18 hours of the original administration of opaque medium [iopanoic acid] rather than waiting 36 hours. A whole day may be saved and other diagnostic studies may be performed."[1]

plus other important advantages

Less discomfort—In a study utilizing iopanoic acid and ORAGRAFIN Sodium Capsules (Sodium Ipodate) nausea, vomiting, diarrhea, cramps, and dysuria—all were less frequent with Oragrafin Sodium Capsules.[2] *Few side reactions*—McCrory[3] reported that Oragrafin Sodium and Calcium were "...used routinely in cholecystographic studies in approximately 2000 patients with excellent diagnostic films and only rare and mild reactions." *Greater convenience*—Routine overnight procedure with Oragrafin Sodium Capsules is easy for patients to follow. If necessary, rapid, same-day re-examination procedures possible with the calcium granules reduce the need for I.V. studies.

Contraindications: Contraindicated for persons sensitive to oral iodine compounds or for patients with *combined* renal and hepatic disease or *severe* kidney impairment. Gastrointestinal disorders, which may interfere with absorption, or liver dysfunction, which may result in inadequate biliary secretion of medium, are likely to result in unsatisfactory visualization.

Precautions and Side Effects: Mild and transient nausea, vomiting, or diarrhea sometimes occur; but the incidence can be reduced by using the calcium granules and restricting the dosage to 3 Gm. Transient headache, dysuria, or abdominal pains may occur.

Hypersensitivity reactions may include urticaria, serum sickness-like reactions, and rarely anaphylactoid shock. They are more likely to occur in the individual with a history of allergy, asthma, hay fever, or urticaria and in the individual who is known to be hypersensitive to iodine compounds. Antihistamines and corticosteroids are used to control hypersensitivity reactions; but the occasional serious anaphylactoid reactions require the immediate use of epinephrine or phenylephrine, oxygen, and intravenous corticosteroids.

For full information see Package Insert.

Supply: The calcium salt (Oragrafin Calcium Granules) is available in single-dose foil packets providing 3 Gm. of calcium ipodate as granules dispersed in flavored sucrose. The sodium salt is available in capsule form (Oragrafin Sodium Capsules) providing 0.5 Gm. sodium ipodate per capsule.

References: (1) Root, H. F., Jr.: Amer. J. Roentgen. *101*:621 (Nov.) 1967. (2) White, W. W., and Fischer, H. W.: Amer. J. Roentgen. *87*:745 (April) 1962. (3) McCrory, E.: J. Tenn. Med. Ass. *58*:258 (Aug.) 1965.

Oragrafin® Calcium Granules
Calcium Ipodate

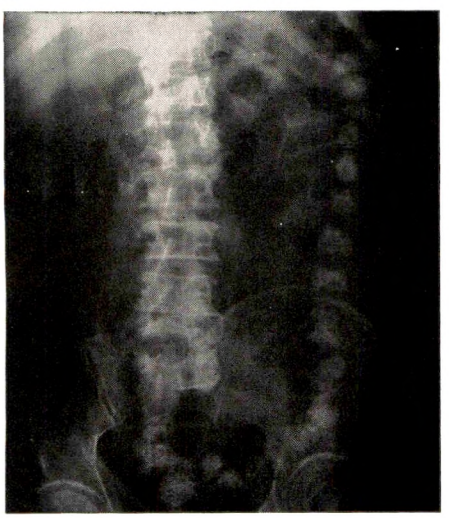
Oragrafin® Sodium Capsules
Sodium Ipodate

SQUIBB 'The Priceless Ingredient of every product is the honor and integrity of its maker.'™

Baroloid® (barium sulfate shows minimal flocculation*

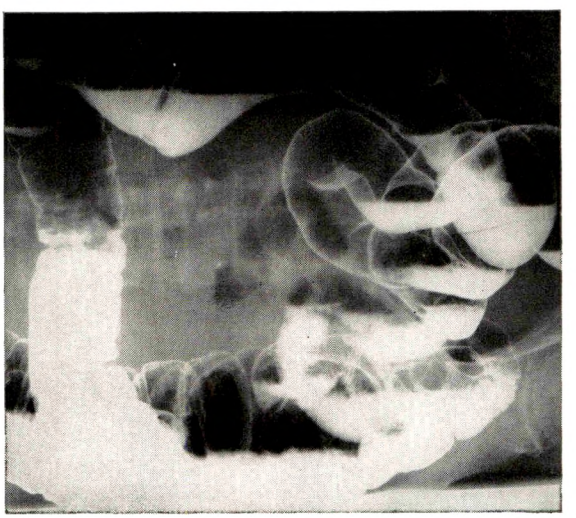
Leaves a thin, continuous coating of highest diagnostic value.

BAROLOID—exceptional nonflocculating properties, pleasant cherry flavor.



In this patient, noncolloidal barium shows heavy flocculation and precipitation in all segments of the colon. Film taken post-evacuation.

*Bryk, D. and Roska, J. C.: Radiology 92:832, Mar., 1969.



In this patient, FLEET®BAROLOID® shows no flocculation. Note thin, veillike, smooth and homogeneous pattern of BAROLOID in the colon. This air-contrast film was taken with the patient in the lateral decubitus position.

Fleet® pharmaceuticals **Makers of the Widest Barium Line**
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Introducing new HALSEY®

"Chron-I-System"*

(PAT. APPL. FOR)

A revolutionary new technique that takes positive patient-film identification out of the dark.

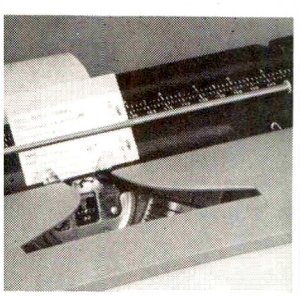
One of the most important new developments in radiographic identification in recent years, New Halsey Chron-I-System provides simple, rapid, positive patient-film identification in ordinary daylight.

Consisting of four basic components—Chron-I-Settes (cassettes), Chron-I-Screens (intensifying screens), Chron-I-Serts (cassette insertion strip and patient-identifying labels) and a specially designed Exciter unit—new Halsey Chron-I-System is independent of x-radiation (hence no "burn-out") and permits positive identification to be performed prior to, or after the x-ray examination. The need for identification printers is also eliminated.

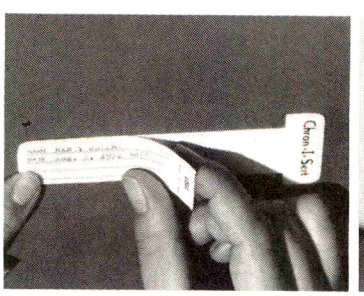
Ask your dealer for a demonstration today!

*TM

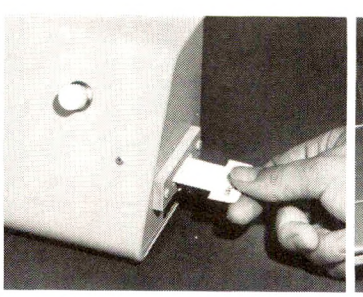
4 simple steps... in broad daylight!



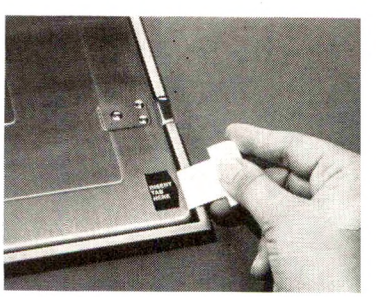
ep 1. Type or write ng pen or pencil) pa- it data on special label.



Step 2. Mount label on Chron-I-Sert insertion strip, and send into x-ray room with patient.



Step 3. Insert Chron-I-Sert (label on strip) into Exciter for a few moments and then withdraw.



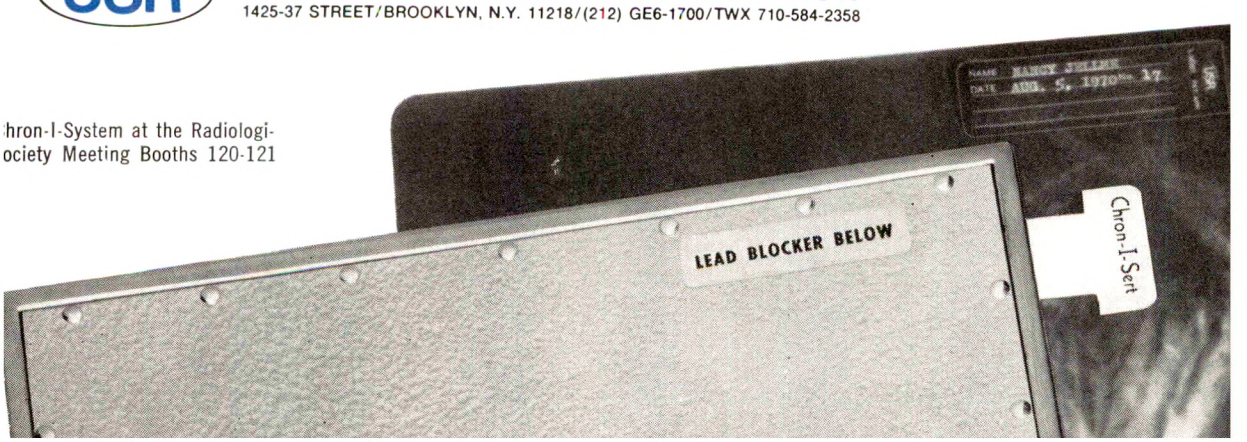
Step 4. Insert Chron-I-Sert into Chron-I-Sette slot. Remove in 3 seconds. Patient-film identification is complete—proceed with radiographic examination.

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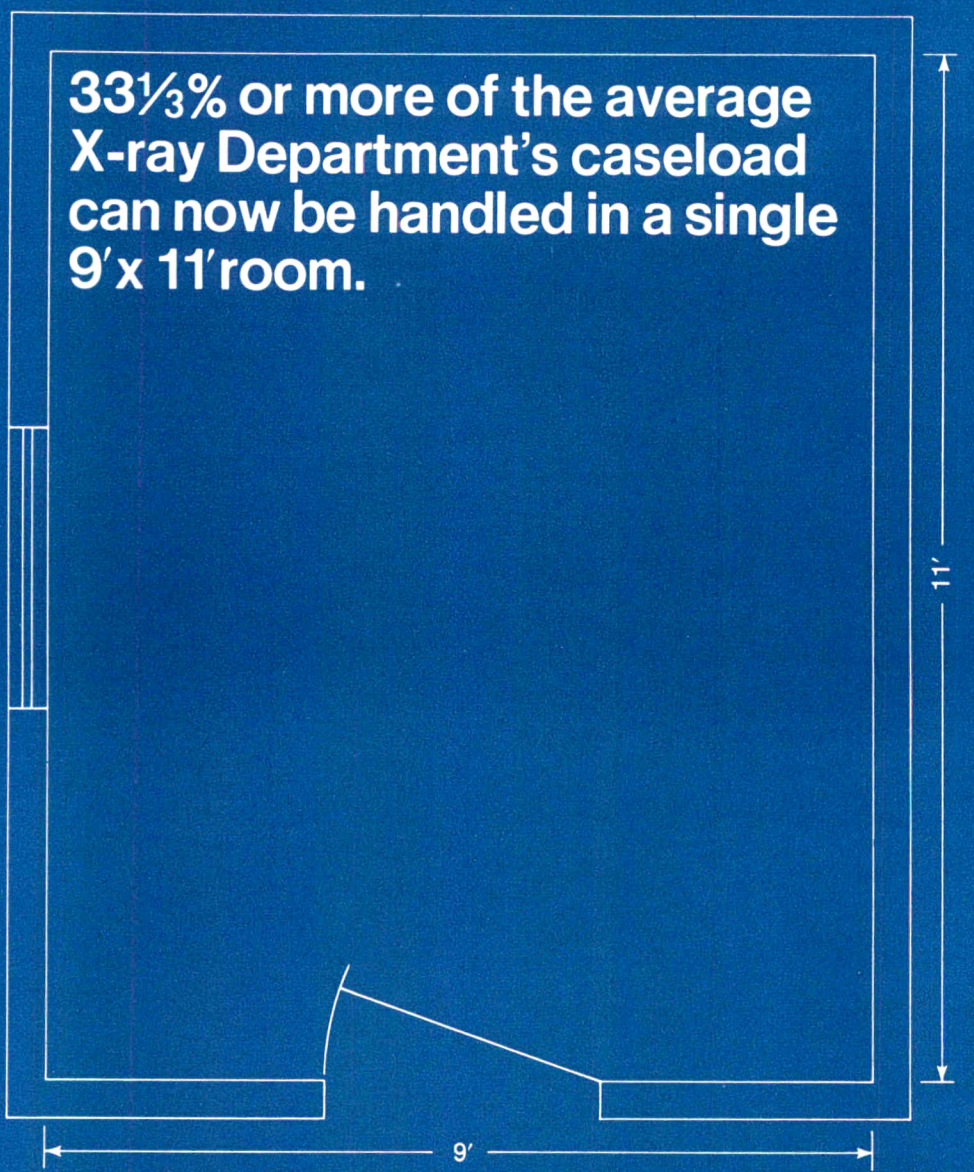
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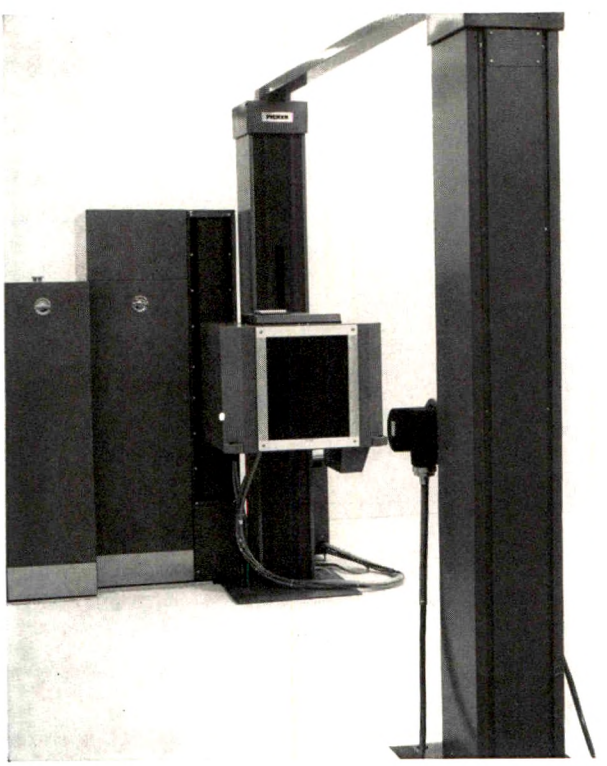
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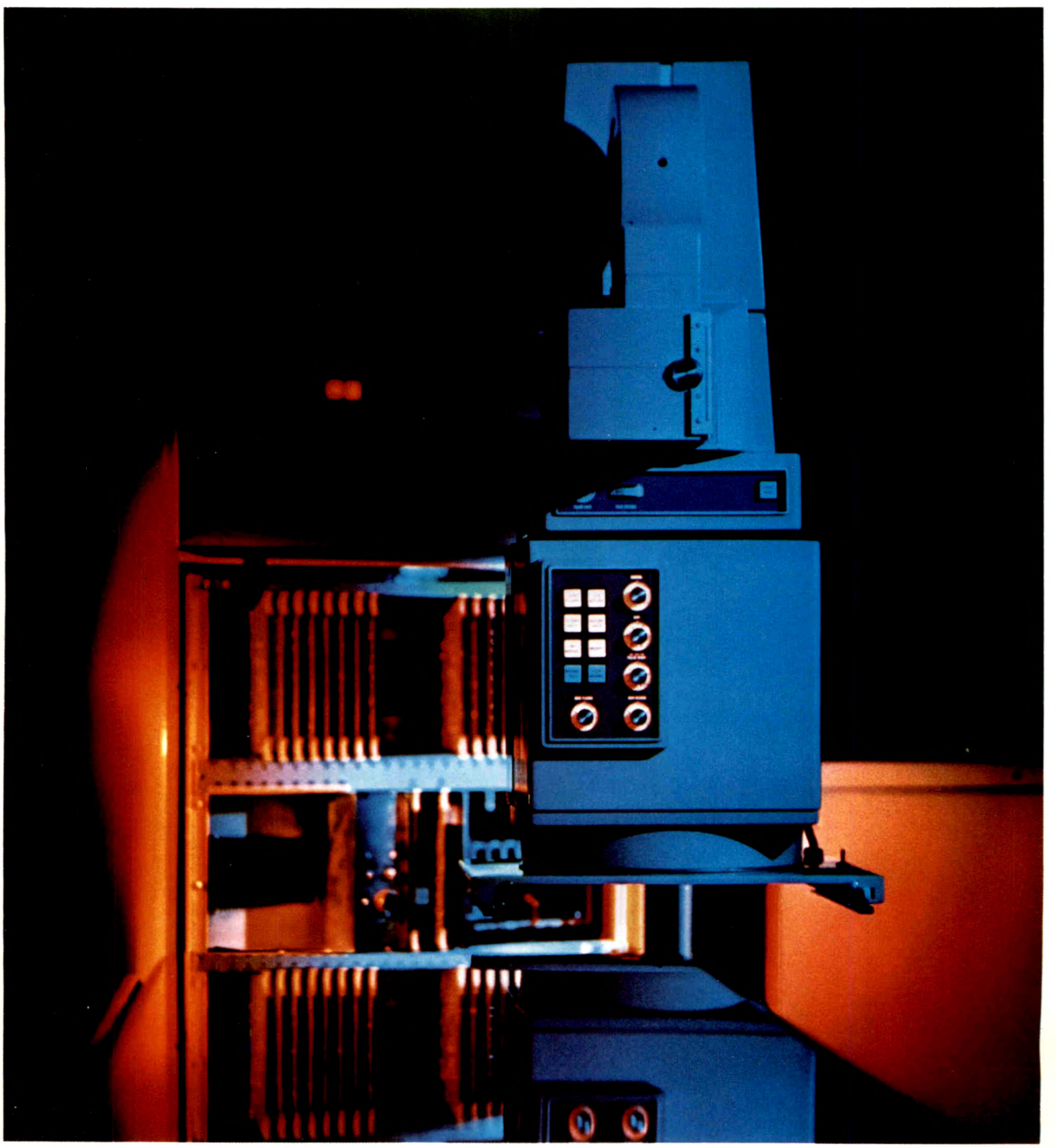
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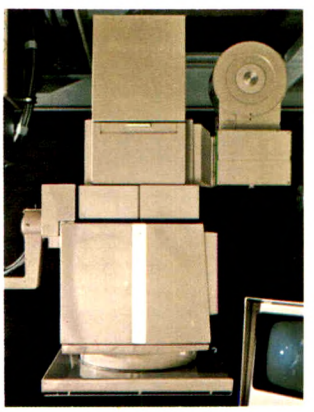
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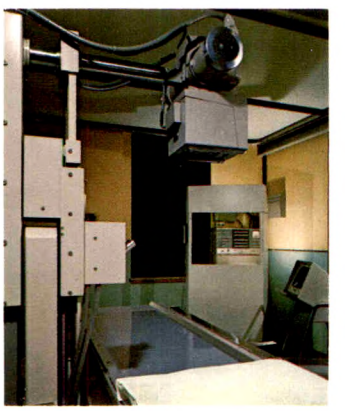
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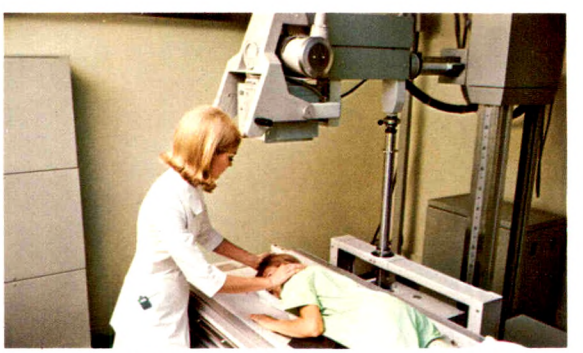
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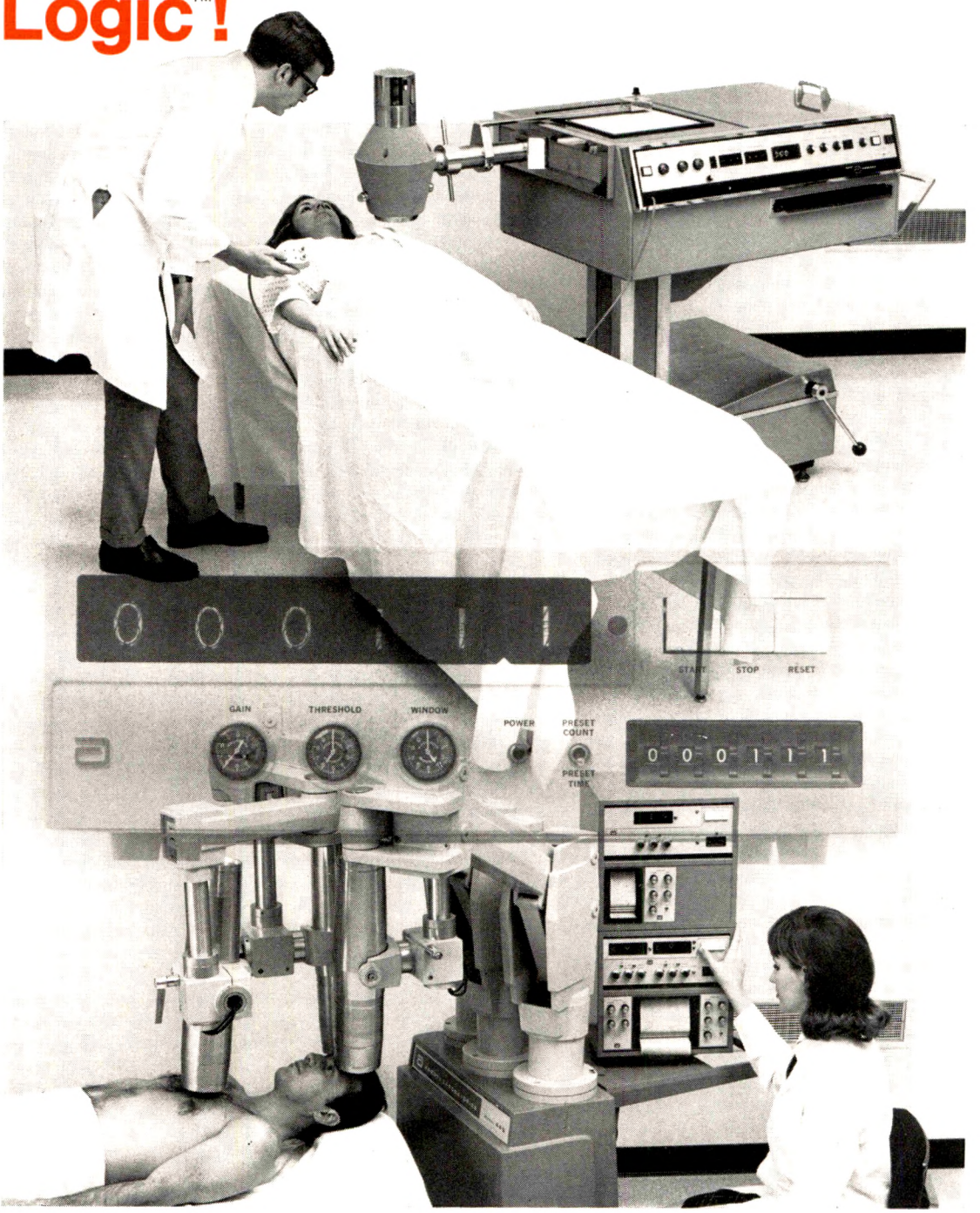
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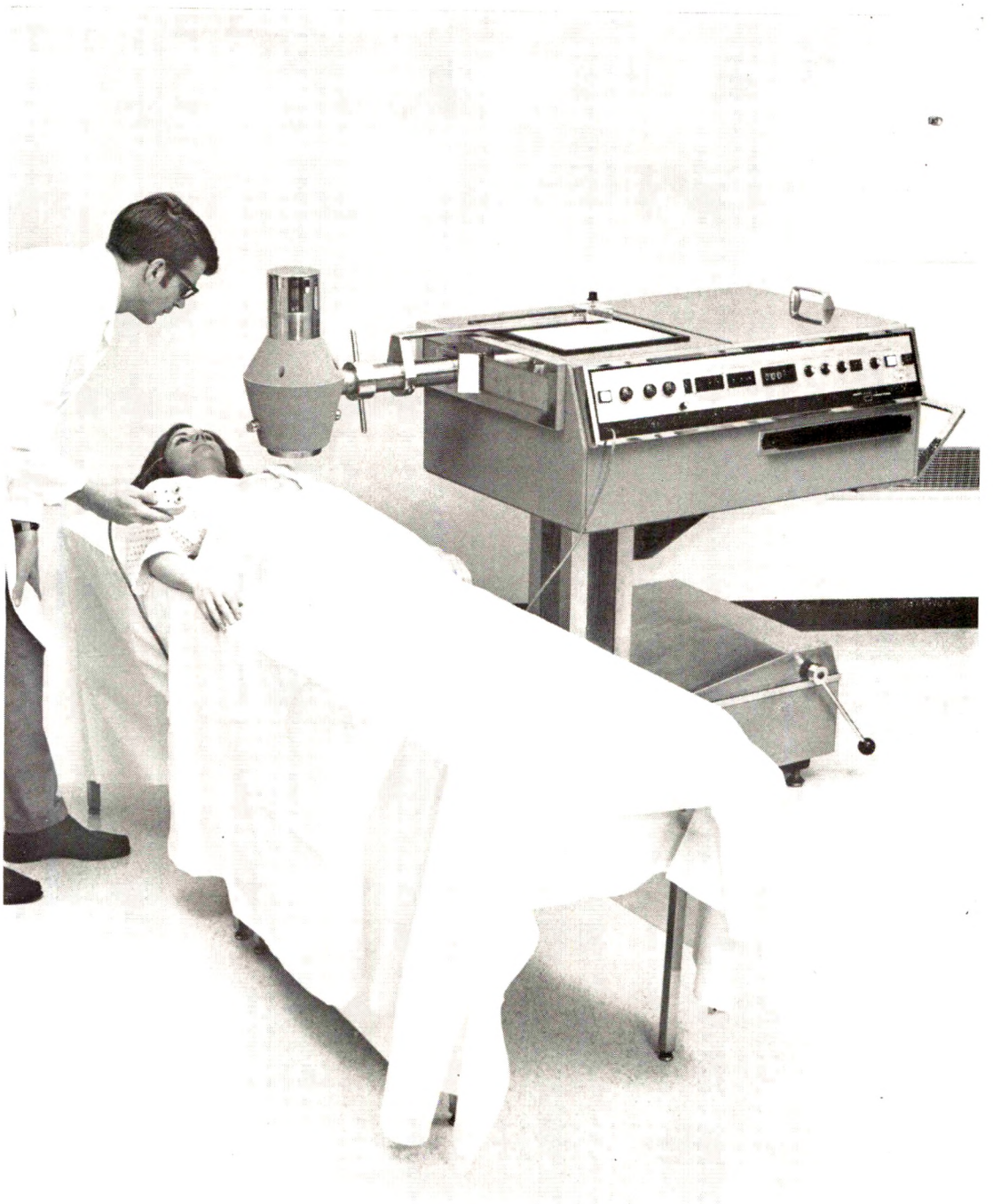
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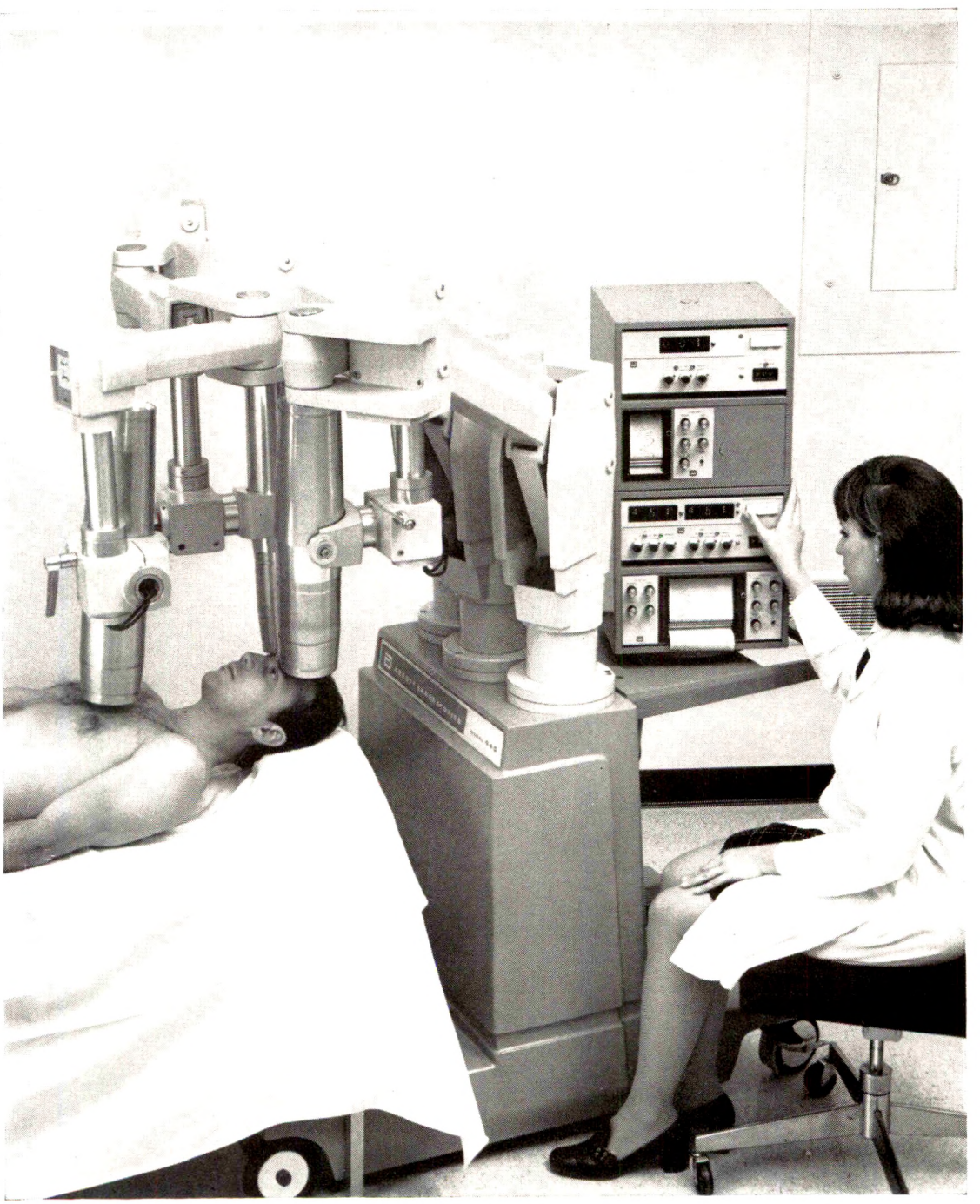
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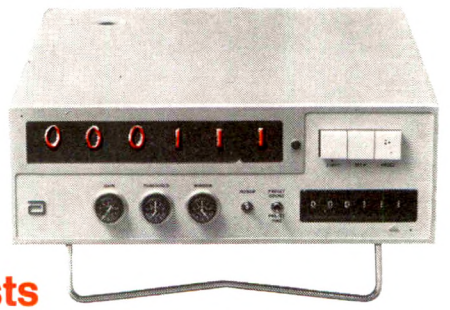
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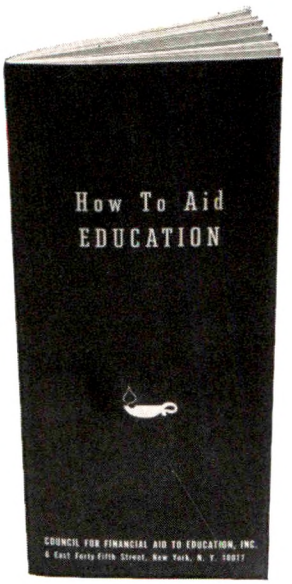
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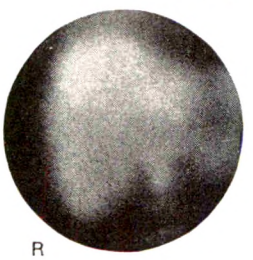
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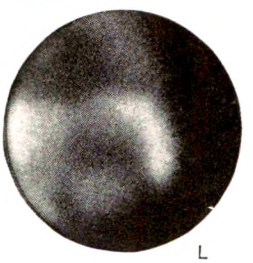
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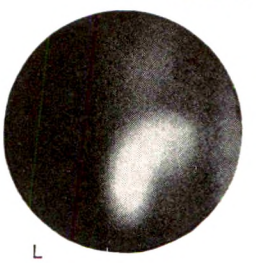
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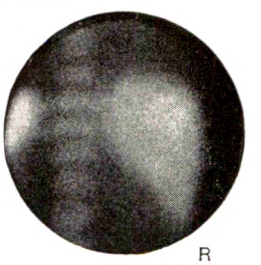
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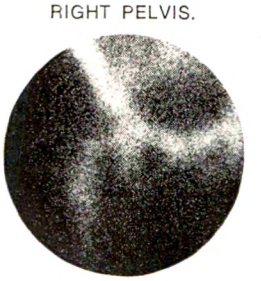


RIGHT



LEFT

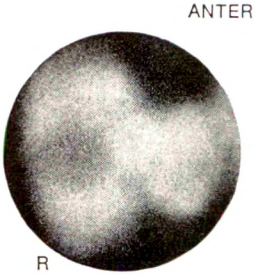
RIGHT PELVIS.

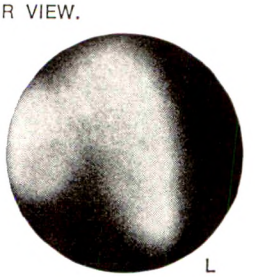


CASE STUDY NO. 2. LEIOMYOSARCOMA METASTATIC TO LIVER.

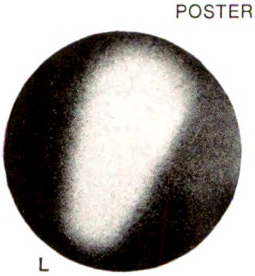
STATIC SCINTIPHOTOS.

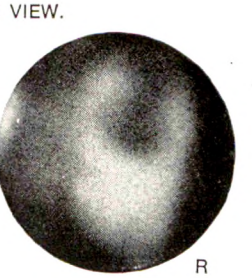
ANTERIOR VIEW.



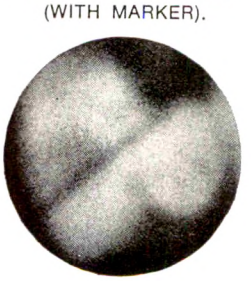


POSTERIOR VIEW.

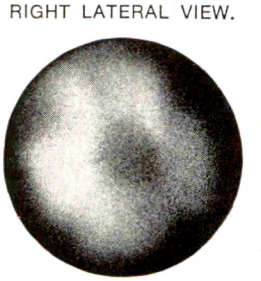




RIGHT ANTERIOR VIEW
(WITH MARKER).



RIGHT LATERAL VIEW.



Th Liv r Study

Evaluation of Reticuloendothelial System Labelling in the Liver with the Nuclear-Chicago Pho/Gamma® Scintillation Camera

Liver scintiphotography employing ^{99m}technetium sulfur colloid and the Pho/Gamma Scintillation Camera offers extremely high resolution images of reticuloendothelial-system distribution in the liver, spleen and bone marrow.

PRELIMINARY DISCUSSION. In the normal liver, the reticuloendothelial system is uniformly distributed, with areas of decreased labelling showing only in the region of the porta hepatis, gall bladder fossa, and in intersegmental fissures.

Abnormal regional decreases of liver labelling may be recognized as either (1) *irregular decrease of labelling* in the whole liver or an area of it or (2) *focal decreases of labelling* with discrete margins and clear definition in comparable scintiphoto views.

SETTING-UP. Liver scintiphotography is usually best performed with the high-resolution, low-energy Pho/Gamma collimator appropriate for ^{99m}Tc. The patient is positioned touching the collimator, and is examined in the recumbent position to reduce respiratory and other motions. In circumstances where the entire liver and spleen area are to be visualized in one view, the diverging collimator may be used.

ISOTOPE AND DOSE. An intravenous injection of 3 or 4 mCi of ^{99m}Tc sulfur colloid is administered.

DATA ACCUMULATION. Twenty minutes after injection, a series of static scintiphotos of the liver, spleen and bone marrow is obtained. A non-enlarged spleen is best imaged in left posterior and oblique views. Useful marrow views include upper sternal area, and left pelvis, hip and femur.

Data densities of 500,000 counts for an anterior view of the liver are desirable. Preset exposure time is kept constant throughout examination of the liver and spleen so that exposure intensity will be comparable in all the scintiphotos of these organs. For marrow scintiphotos, increased dot density and 2-minute exposures are normally used.

CASE HISTORIES. Case Study No. 1: Male, 60 years old. Known cirrohosis probably due to chronic alcoholism. Admitted for evaluation of low-grade fever.

Case Study No. 2: Female, 62 years old. Admitted for evaluation of abdominal cramping and liver enlargement. Seven years earlier, partial gastrectomy yielded the diagnosis of "leiomyoma, ulcerated stomach." Two years prior to this admission, laparotomy had revealed leiomyosarcoma in the left lobe of the liver.

EVALUATION. The purpose of these Pho/Gamma liver studies is to evaluate (1) shape, position, and general outline of the liver as imaged on the scintiphotos and (2) the nature of any labelling decrease, whether uniform, irregular or focal. Labelling in the spleen and marrow is compared with liver labelling to assess the possibility of portal-systemic shunting (indicated by greater spleen and marrow labelling, relative to the liver) or hypertrophy of the bone marrow.

In the clinical scintiphotos shown at left, examples of uniform decreased labelling, irregular labelling, and focal defects of labelling are evident.

The patient with cirrhosis (Case Study No. 1) has generalized decrease and irregularity of labelling consistent with that disease. Furthermore, a focal defect of labelling exists in the left lobe of the liver and is best seen in the left lateral view. (This defect was subsequently found by local surgical biopsy to be the site of focal necrosis which had been responsible for the patient's low-grade fever of unknown origin.) Also typical of a cirrhotic are the bright labelling of the slightly enlarged spleen and bone marrow (with marrow extension into the right femur).

The patient with leiomyosarcoma (Case Study No. 2) is an excellent example of focal metastatic lesions causing some decrease of liver labelling, as well as enlargement of the liver that is so common with metastatic disease of the liver. Giant splenomegaly also exists on a congestive basis.

CONCLUSIONS. Liver scintiphotography with the Pho/Gamma Scintillation Camera and ^{99m}Tc sulfur colloid appears to be a markedly improved liver-imaging technique and sensitive diagnostic test for liver disease.

This form of scintiphotography provides a large amount of specific information about liver structure and hemodynamics and is an accurate guide for the selection of biopsy sites. When combined with other special procedures, such as liver scintiphotography during rose-bengal excretion or liver-blood-flow evaluation, the Pho/Gamma liver study with ^{99m}Tc sulfur colloid offers many other diagnostic possibilities.

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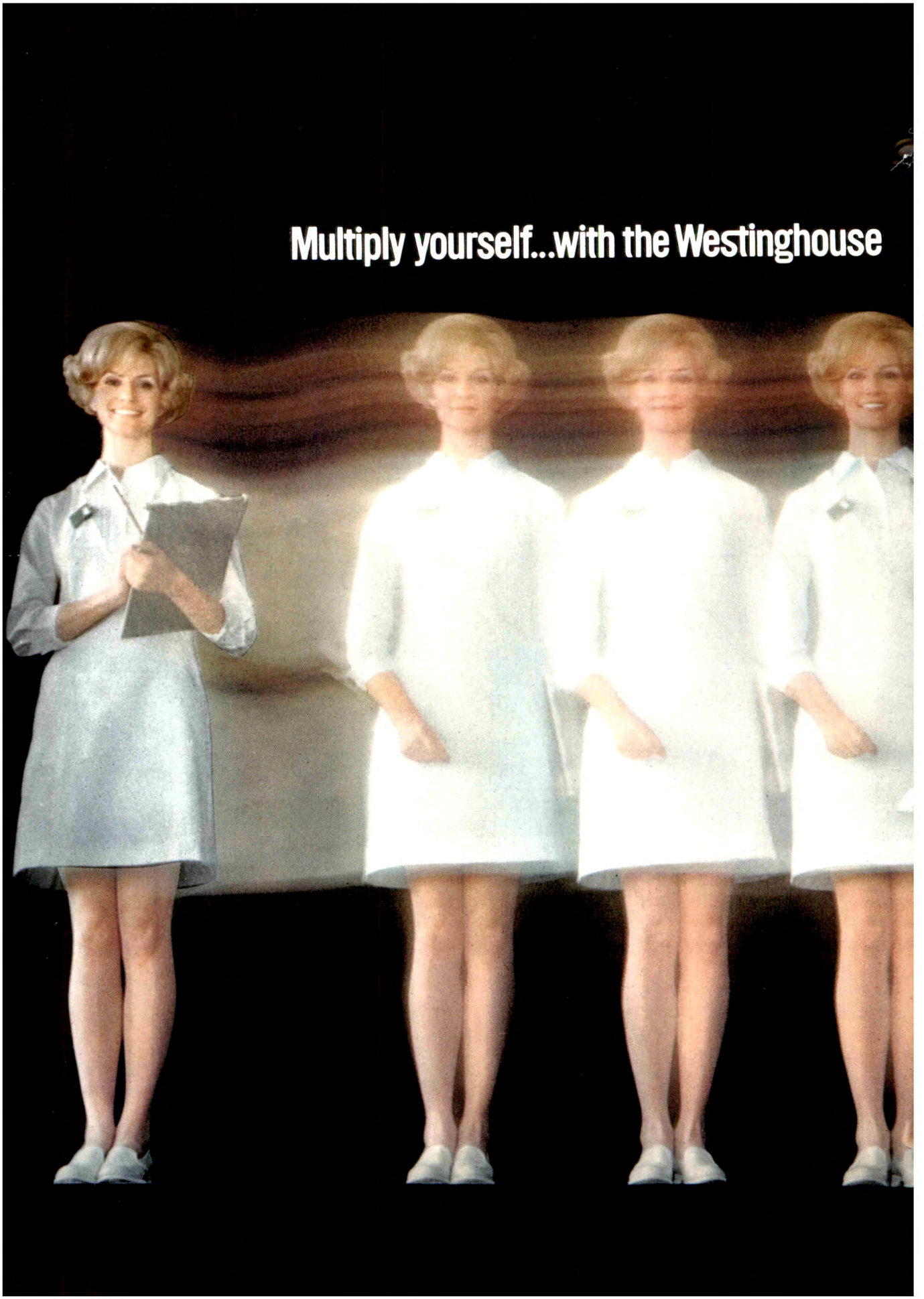
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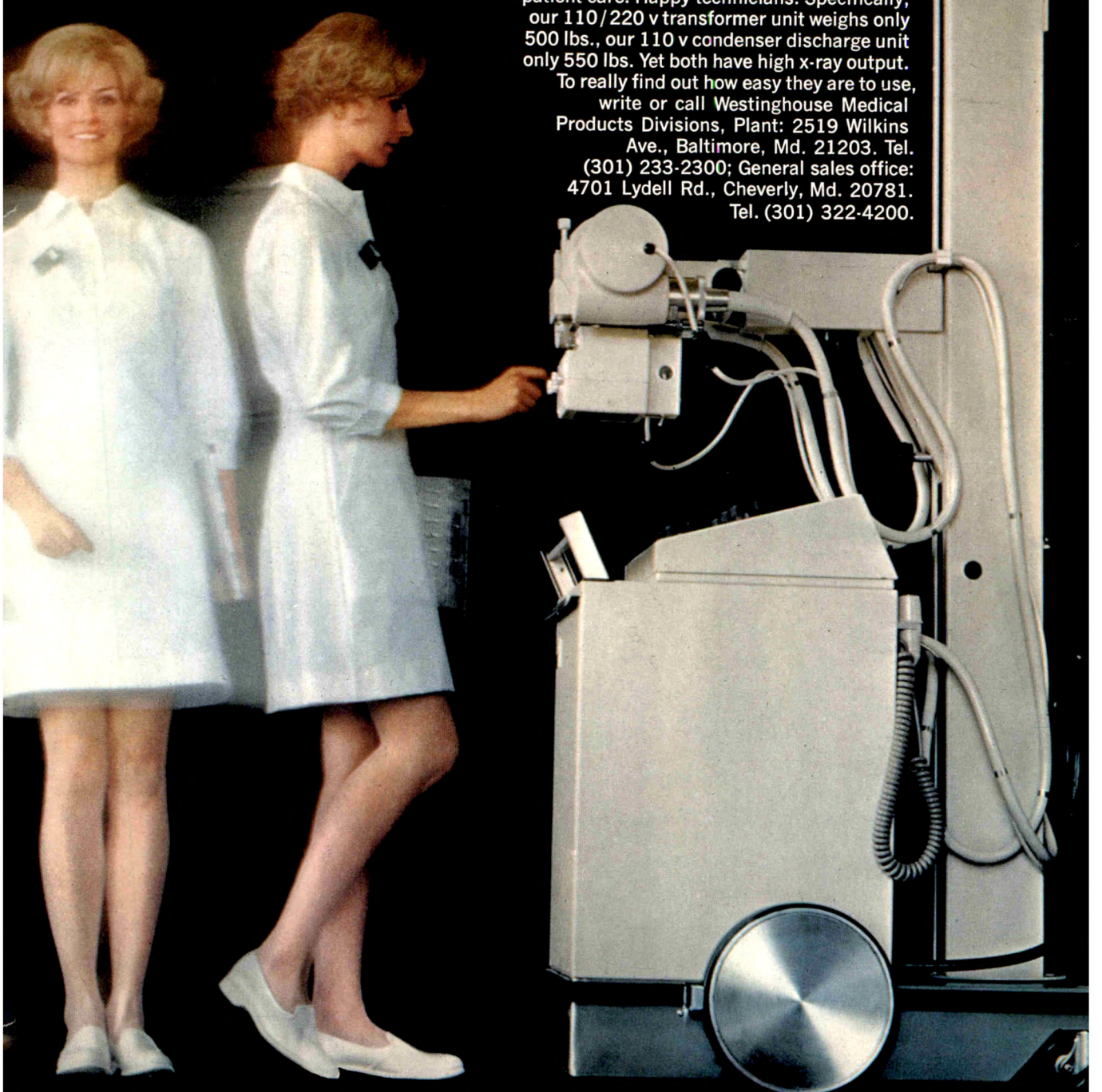
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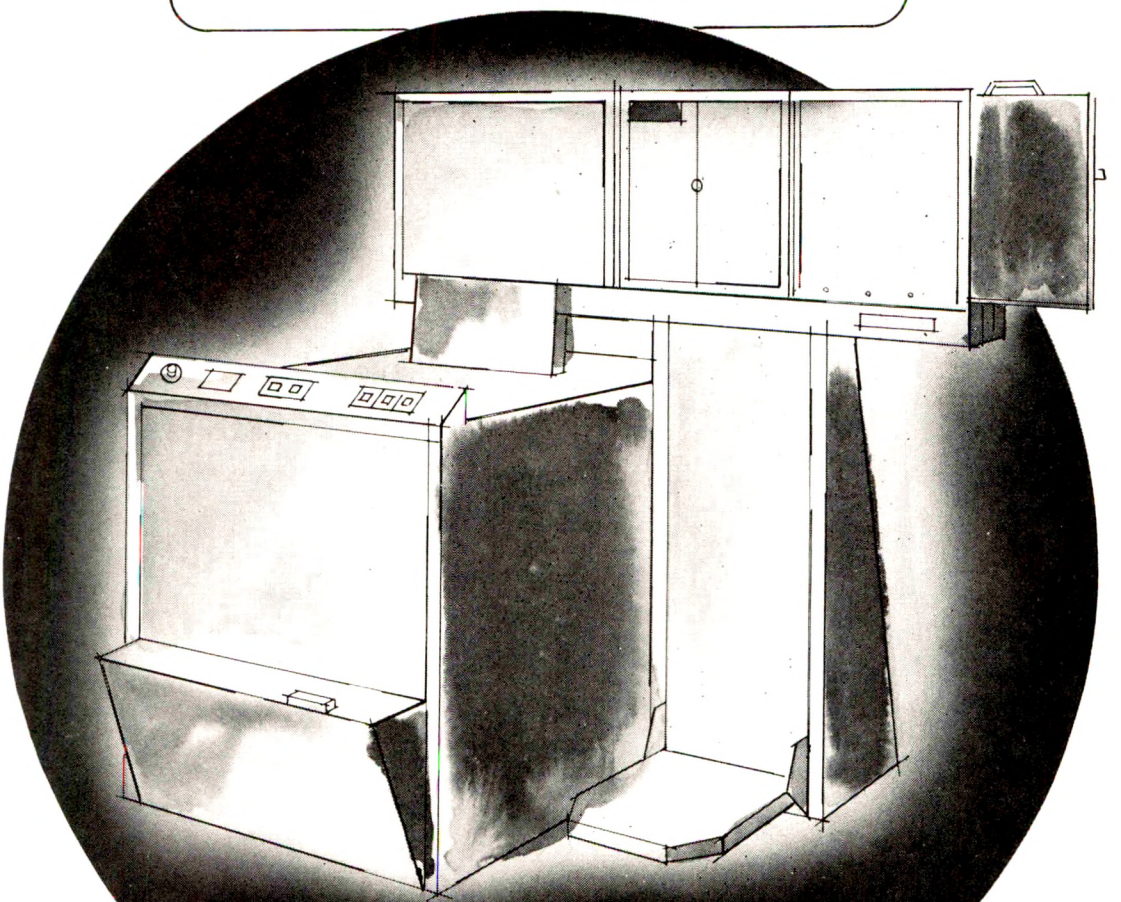
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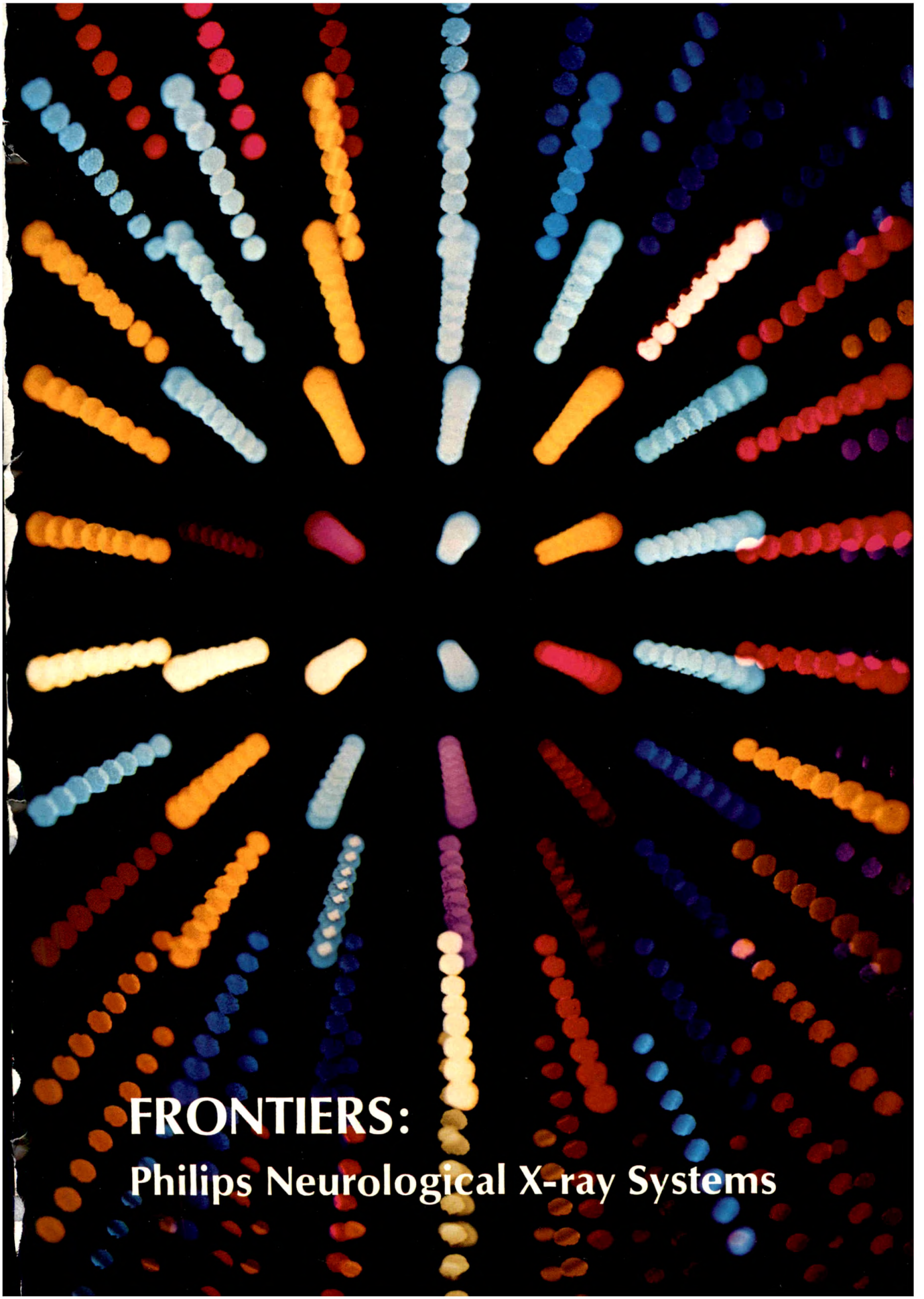
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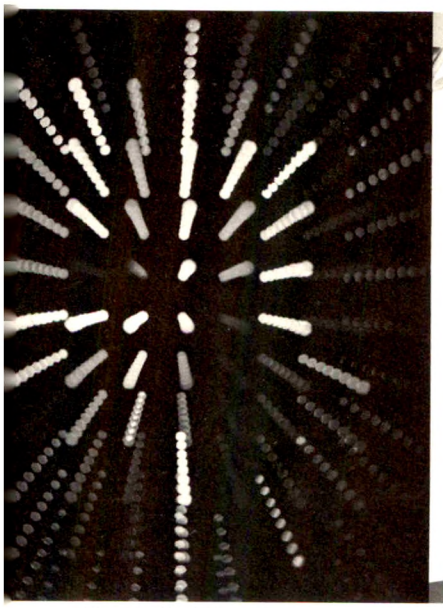
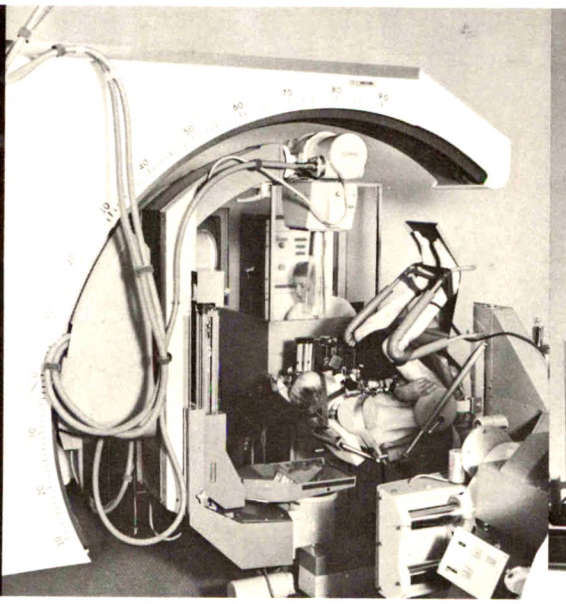
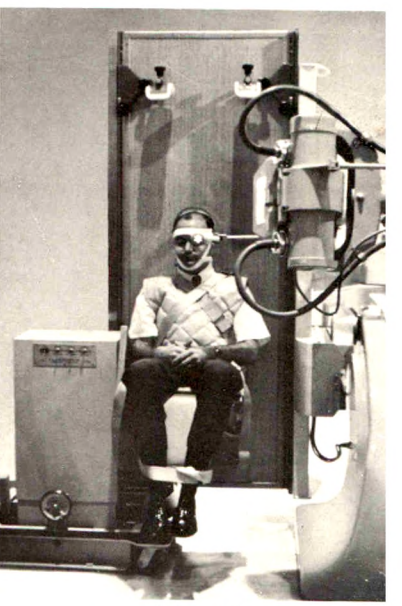


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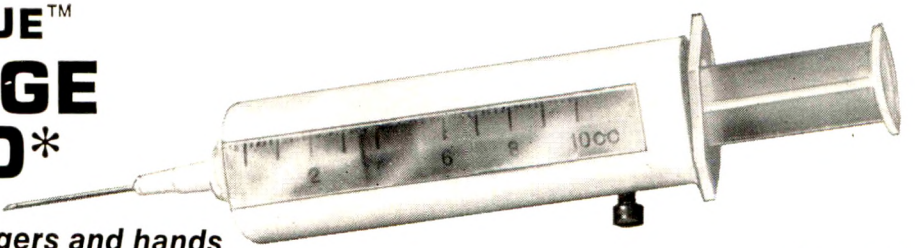
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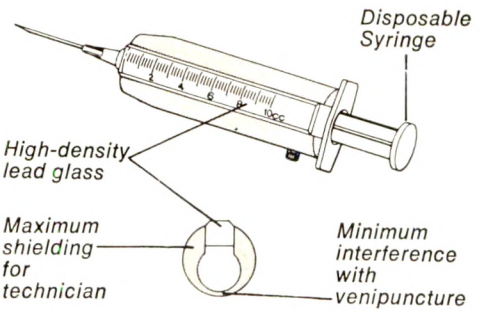
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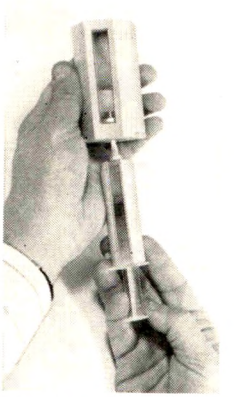
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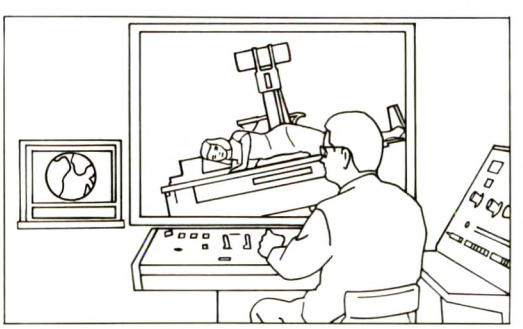
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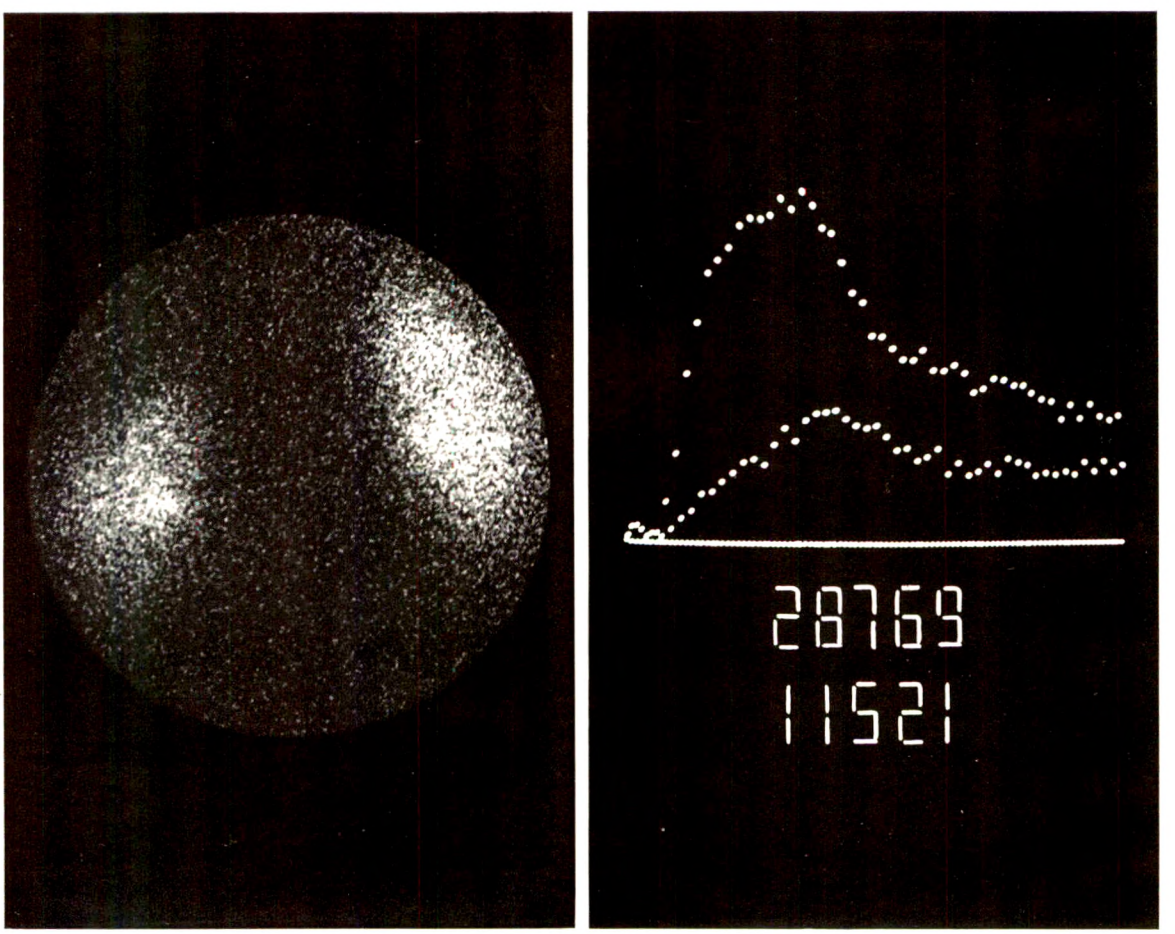
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Vol. 110, No. 4

THE SYNDROME OF BRONCHIAL MUCOCELE AND REGIONAL HYPERINFLATION OF THE LUNG*

By LEE B. TALNER, M.D., JOHN T. GMELICH, M.D.,† AVERILL A. LIEBOW, M.D., *and* RICHARD H. GREENSPAN, M.D.

NEW HAVEN, CONNECTICUT AND SAN DIEGO, CALIFORNIA

WHEN the lumen of a bronchus becomes occluded, there may develop dilatation of the distal portion with gradual accumulation of mucus. The term bronchial mucocele (bronchocele) has been previously coined to describe this dilated, bronchial structure which retains its normal connections to the distal lung parenchyma.[18] Such a sequence of events may follow either congenital or acquired bronchial obstruction. The former is exemplified by bronchial atresia while the most common examples of the latter are postinflammatory stricture and neoplasm.

An intriguing aspect of segmental bronchial obstruction is the resulting disturbance in ventilation of the involved lung segment. In contrast to occlusion of a lobar bronchus which invariably produces atelectasis, occlusion of a segmental bronchus rarely does so because of the presence of collateral ventilation. Although segmental bronchial obstruction may not produce any change in volume of the involved lung segment, most of the reported cases of mucocele due to bronchial atresia have shown a striking degree of focal hyperinflation in the involved segment. This complex of bronchial atresia and focal emphysema was recently reviewed by Curry and Curry,[7] who added 2 cases to the 6 they found previously reported.[1,24,28] Further search has revealed 11 additional examples,[4,5,6,10,12,13,18,26] most of which were presented with emphasis on the bronchocele, but which also demonstrated the features of bronchial atresia and focal emphysema.

In the present report 2 patients are added to the 19 with this syndrome. In both the findings are consistent with bronchial atresia, but the presence of evidence of chronic infection makes it impossible to exclude postinflammatory bronchostenosis as a contributing factor in the development of bronchial obstruction.

REPORT OF CASES

CASE 1. S.S., an asymptomatic 28 month old girl was admitted in September 1965 for investigation of a gradually enlarging mass in the right chest. She was in excellent health until 10 months of age when she developed a cough, fever and wheezing. Roentgenograms of the chest showed bilateral pulmonary infiltrates and hyperexpansion of the right lower lung field (Fig. 1*A*). After antibiotic therapy, she improved and remained asymptomatic. There was no history of foreign body aspiration. In serial roentgenograms, there was a persistent hyperlucency of the right lower lung field and a gradually enlarging ovoid density below and lateral to the right hilum (Fig. 1*B*).

The admitting physical examination disclosed no abnormalities other than decreased breath sounds over the right lower chest. Investigative procedures for cystic fibrosis, agammaglobulinemia and infectious processes were negative. The findings at bronchoscopy and right heart catheterization were normal. Air trapping in the right lower lobe was confirmed during fluoroscopy. On bronchographic examination (Fig. 1*C*), the upper and middle lobe bronchi were displaced superiorly. Several foci of overdistention with adjacent bronchial crowding were noted within the superior segment (B_6) and in several basilar regions, producing an unusual bronchographic pattern. A subapical (Bx_6 according to Boyden) segmental bronchus ended blindly 1 cm. proximal to the ovoid lesion. The vascular rearrangement demonstrated in the pulmonary arteriogram was consistent with the bronchographic anatomic findings. The ovoid lesion did not opacify. In a

* From the Departments of Radiology and Pathology, Yale University School of Medicine, New Haven, Connecticut, and the University of California, San Diego, California.

Supported by grants from USPHS–HE 7277, HE 12373–G.M. 1152.

† USPHS Trainee in Pathology (HE-5153).

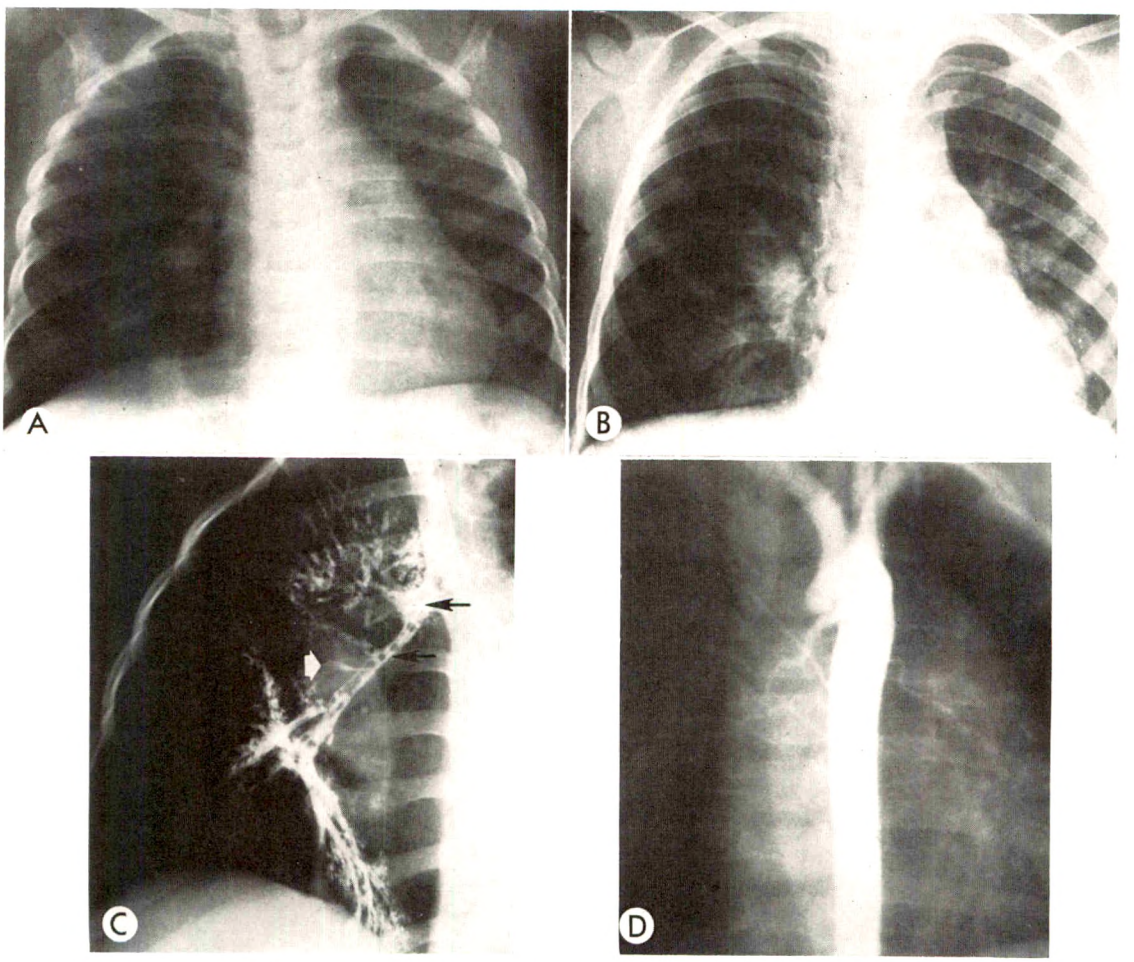


FIG. 1. Case 1. (*A*) Initial frontal roentgenogram at the age of 10 months shows increased radiolucency of the right lower lung field with streaky densities at the right base. (*B*) Posteroanterior chest roentgenogram at 28 months of age demonstrates large ovoid density near the right hilum with lower lobe infiltrate. Hyperlucency and overinflation are clearly demonstrated on the right side. (*C*) Right posterior oblique view o right bronchogram shows bizarre distortion of bronchial anatomy. The upper and middle lobe bronchi are displaced superiorly (dark arrows) by an emphysematous lower lobe. The basilar segmental bronchi are compressed. A subapical bronchus (white arrow) ends blindly proximal to the oval density. (*D*) Thoracic aortogram reveals enlarged right bronchial artery but no aberrant systemic arterial supply to the right lung.

thoracic aortogram (Fig. 1*D*), there was enlargement of the right bronchial artery but no abnormal systemic vessels coursed to the right lung.

At thoracotomy in October 1965, there was overinflation of the lateral portion of the right lower lobe and only a minimal ventilatory response could be demonstrated by the anesthesiologist. Normal ventilatory responses of inflation and deflation were observed in the middle and upper lobes. A right lower lobectomy was performed.

After its removal, the intact right lower lobe maintained its usual size and contour because of severe air trapping. The lobe was inflated with a modified Kaiserling fixative via a bronchial cannula. A bronchogram made on the specimen again demonstrated the blind subsuperior segmental bronchus, and no contrast material could be introduced into the ovoid lesion. In an effort to trace possible proximal connections of the latter, the B_6 and Bx_6 segmental bronchi were injected with different colored vinyl plastic, following which the lung

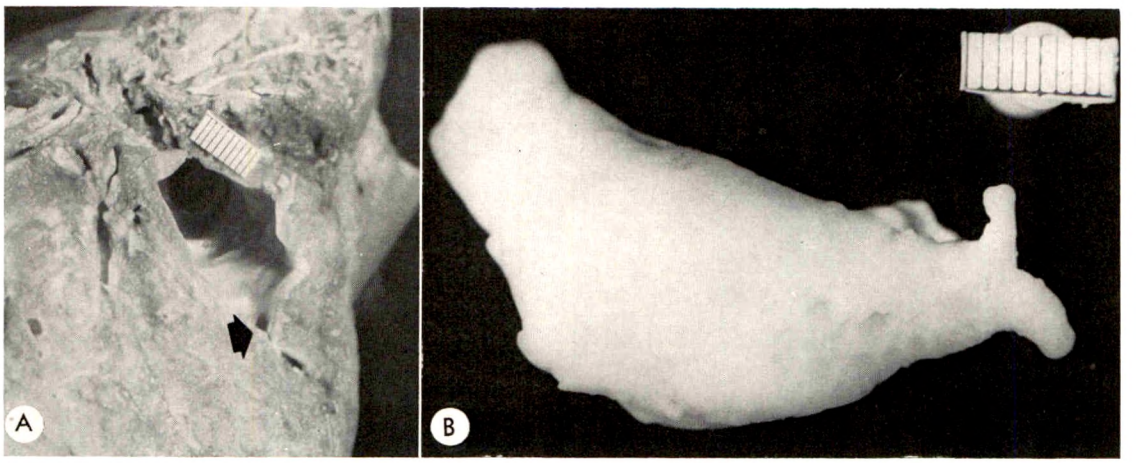


Fig. 2. Case 1. (*A*) Gross photograph of resected right lower lobe shows the fusiform cavity occupying the subapical region. No proximal bronchial connection could be demonstrated. However, distal bronchial distributions are present (arrow). (*B*) Large plug of solid mucus which completely filled the dilated bronchial cavity shown in *A*. Note the finger-like projections which extended into the distally connected bronchi.

was sectioned along the major bronchi. A 2×3×1.2 cm. fusiform sac occupied a subapical position (Fig. 2*A*). No proximal connections to the B_6 or Bx_6 bronchi could be found. This dilated bronchial segment was filled with a large, solid mucus plug (Fig. 2*B*) which had 3 small finger-like projections extending into distally connected bronchi. The wall of the sac was slightly fibrous, measured 1.5 mm. in thickness, and was lined by pseudostratified columnar ciliated epithelium. Circumferentially arranged smooth muscle and small mucous glands were present, but no islands of cartilage were identified. In the distal lung parenchyma, there was bronchiolectasis and chronic bronchiolitis with a plasmacytic and lymphocytic cellular infiltrate in the submucosa. The sections of the Bx_6 bronchus and posterior basilar bronchus contained immature cartilage. This interpretation was based on the increased pliability to palpation and microscopic demonstration of the thick perichondrium, decreased amount of matrix and hypercellularity of the chondrocytes in the small islands of cartilage. The apical (B_6) bronchus and the other basilar bronchi had more mature and well developed cartilage. Pathologic diagnosis was atresia of the subapical bronchus to the right lower lobe with bronchocele formation.

Case II. L.D., a 14 year old girl with dorsal-lumbar scoliosis first noted in March 1965, was admitted in April 1966 for corrective surgery of the increasing curvature. A left hilar mass was observed on preoperative chest roentgenograms (Fig. 3, *A* and *C*), which, retrospectively, had been present on lateral roentgenograms of the spine in September 1965, but not in the preceding March (Fig. 3*D*). The patient had neither symptoms nor history of any respiratory illness.

On physical examination, no abnormalities were noted other than the scoliosis and decreased breath sounds over the left upper lobe. On fluoroscopic examination, the lesion neither pulsated nor changed volume with the Valsalva maneuver but there was marked air trapping in the left upper lobe, confirmed on the expiration chest roentgenogram (Fig. 3*B*). In laminagrams (Fig. 3*E*), the mass was sharply defined, somewhat lobulated in contour and without calcification. Numerous unrevealing diagnostic studies included skin tests for tuberculosis and fungal infection, sputum cultures, sputum cytology, arterial oxygen saturation, scalene lymph node biopsy, bronchoscopy and right heart catheterization. In pulmonary angiograms, there was evidence neither of arteriovenous malformation nor of pulmonary venous varix, and the lesion did not opacify. There was decreased flow of contrast material to the left upper lobe with splaying of vessels in this region consistent with focal hyperinflation (Fig. 3*F*).

At thoracotomy on April 28, 1966, a soft, round, 4 cm. mass was palpated within the left

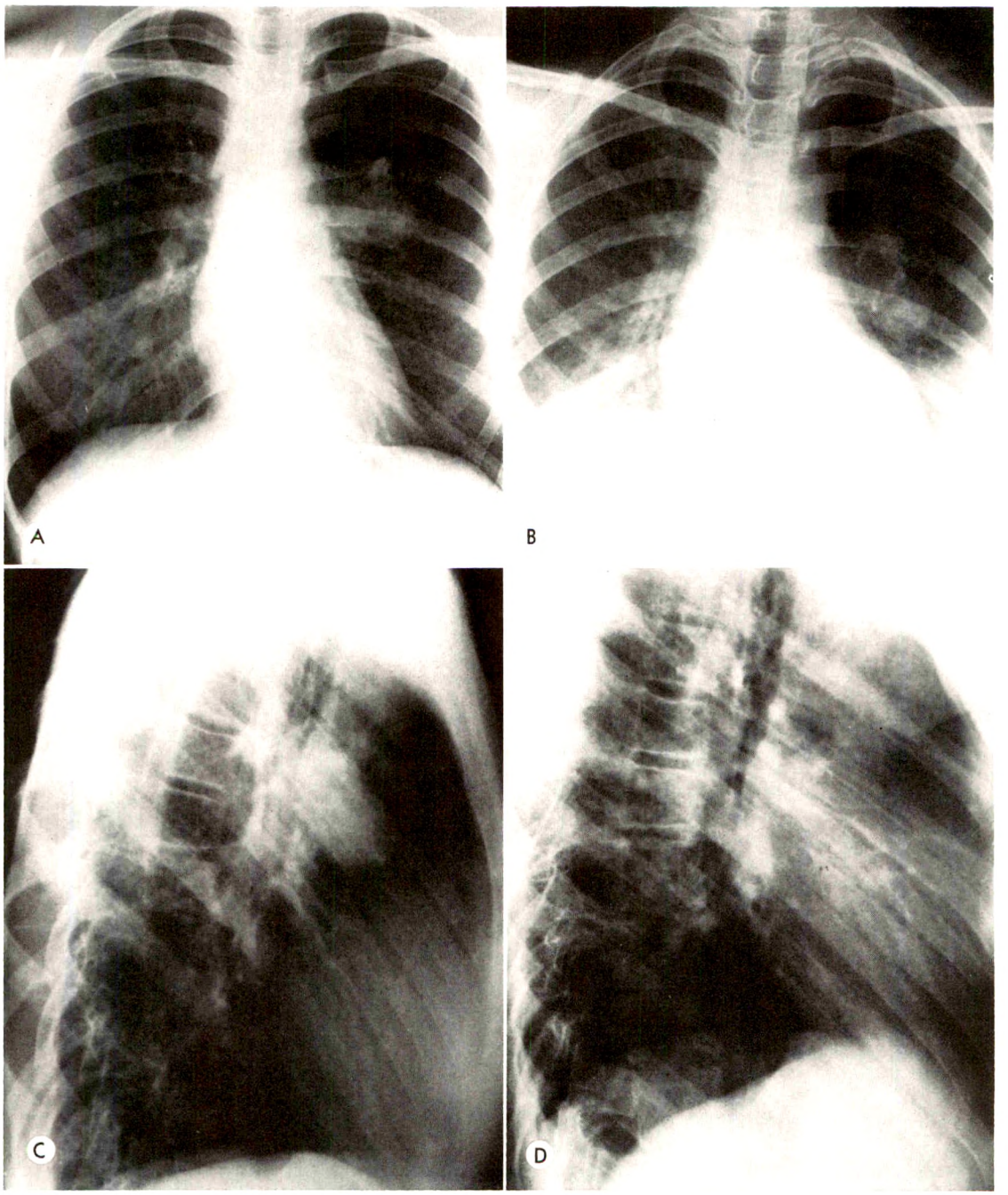


FIG. 3. Case II. (*A*) Left hilar mass with hyperlucency of the left upper lung field on a routine chest roentgenogram taken prior to surgery for correction of scoliosis in April 1966. (*B*) Air trapping in the left upper lung field is evident on the expiratory chest roentgenogram (April, 1966). (*C*) Lateral chest roentgenogram (April, 1966) reveals the smooth, rounded contour and hilar location of the lesion. (*D*) Lateral roentgenogram from the spine examination performed in March 1965. No mass is evident. Subsequent spine examination in September 1965 demonstrated the lesion, but it was not recognized at that time.

upper lobe. There were hyperinflation and air trapping in the superior portion of the left upper lobe. Multiple hyperplastic lymph nodes were present in the left hilum. A left upper lobectomy was performed.

The left upper lobe, after intrabronchial cannulation and inflation with a modified Kaiserling fixative, was sectioned along bronchi (Fig. 4*A*). The bronchial anatomy was extremely complicated as illustrated in Figure 4*B*. In essence, a diaphragm of mucous membrane produced subtotal obstruction of the superior division of the left upper lobe bronchus, just proximal to its bifurcation. Distal to the obstruction, the bronchial subdivisions showed varying degrees of cylindrical and saccular dilatation, the largest measuring 3×2 cm. As in Case 1, the sac had a lining of pseudostratified ciliated columnar epithelium. Its wall contained mucous glands and circumferentially arranged smooth muscle, but in this instance there were islands of cartilage. The obstructing membranes were redundant mucosal folds with superimposed plugs of granulation tissue. The bronchial wall contained islands of cartilage that were few in number, small in size and immature in appearance. There was no chondritis, although plasmacytic and lymphocytic infiltrates were present deep in the walls. In the distal lung parenchyma of the superior division, there were bronchiolectasis, mucus plugging, chronic bronchiolitis, foci of obstructive or "golden" pneumonia, and panacinar emphysema. In contrast, the inferior division of the left upper lobe had bronchi of normal caliber as well as undamaged lung parenchyma. The pathologic diagnosis was obstruction of the superior division of the left upper lobe bronchus due to a congenital diaphragm with subsequent bronchocele formation involving the distal bronchial subdivisions.

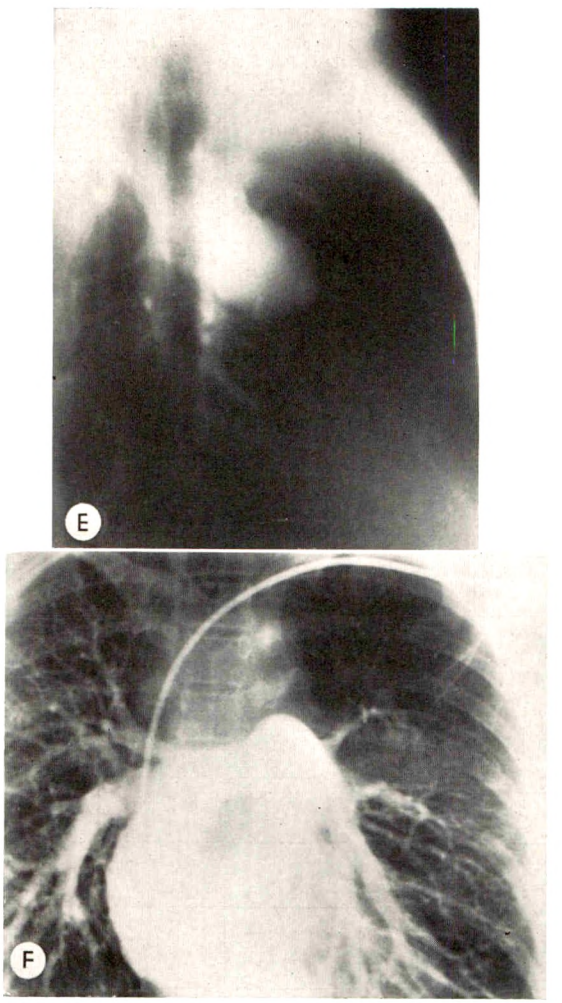


Fig. 3. (*E*) Lateral laminagram demonstrates the smooth, sharply defined margins and intimate relationship to the hilar shadow. No calcification is present. (*F*) Pulmonary arteriogram made from the right atrium. Note the poor flow of contrast to the left upper lobe, splaying of upper lobe arteries and crowding of the lower lobe vessels. These findings are consistent with hyperinflation. The hilar mass does not opacify.

DISCUSSION

The first recorded cases of mucocele of the bronchus were described by Ramsay and Byron in 1953.[18] They offered evidence which suggested that mucocele, bronchogenic cyst and "congenital bronchiectasis" are simply variants of the same pathologic process, but the writings on the subject are controversial and the relationship, if any, between bronchogenic cyst and mucocele remains unclear. Seventeen documented cases of bronchial mucocele have been reported since then. In most of these the underlying abnormality was interpreted to be bronchial atresia, usually involving the left upper lobe. A summary of the pertinent clinical, roentgenologic and pathologic data on the previously reported patients plus our own 2 patients is given in Table 1. Of particular interest is the age distribution (17 of 21 patients below 25 years of age), frequent

TABLE I

CLINICAL, ROENTGENOLOGIC AND PATHOLOGIC FINDINGS IN 21 PATIENTS WITH BRONCHIAL MUCOCELE

Author	Age (yr.)	Sex	Location	Symptoms	Roentgenologic Evidence of Overinflation	Mass on Chest Roentgenog am	Bronchial Abnormality	Surrounding Lung Tissue
Ramsay and Byron[18] 1953	17	F	LLL superior segment and LUL posterior segment	History of pulmonary tuberculosis	Not mentioned	+air-fluid level	Membrane occluding origin of subsegmental bronchus to B_6; branched mucocele beyond	Not stated
	30	F	LUL	Asymptomatic	Not mentioned	+	Complete atresia of anterior segmental bronchus with branched mucocele	Not stated
Jones[13] 1955	22	M	LUL anterior segment	Asymptomatic	Not mentioned	+	Blind end to anterior segmental bronchus; cyst lined by respiratory epithelium and contained cartilage	Not stated
Belsey[1] 1958	6	M	LUL	Cough for 1 year	+	No mass on roentgenogram	Dilated bronchus; no patent communication with left main bronchus	Emphysema, entire LUL
Simon and Reid[24] 1963	19	M	LUL apico-posterior	Increasing shortness of breath	+	+	Dilated branched bronchus (1 cm. diameter) with blind end proximally	Emphysema
	25	F	LUL apical and posterior bronchus	Pain below left shoulder	++	+air-fluid level	2 cm. cystic cavity, branched towards periphery, blind on hilar aspect	Emphysema
	18	?	LUL anterior segment	Recurrent chest infections from age 5 mo.	+	Several thin-walled cavities with fluid levels	Dilated bronchial segment with blind end proximally	Emphysema
Culiner[4] 1964	15	M	RUL apical and anterior segments	Pneumonia RUL, posterior segment	+	+air-fluid level	3 cm. cystic mass with mucoid secretions	Emphysema
	41	M	RUL posterior segment	Asymptomatic	+	+	Several mucus-filled cysts	Emphysema
	8	?	LUL anterior segment	Asymptomatic	++	No mass on roentgenogram	Atretic bronchus with branched mucocele	Emphysema
	25	M	RLL superior segment	Asymptomatic	+	+air-fluid level, comes and goes	No surgery	
Waddell *et al.*[28] 1965	25	M	LUL apical segment	Asymptomatic	++	No mass on roentgenogram	Cystic mass; blind bronchus on hilar aspect with distal branching	Emphysema
	7	F	LUL	Asymptomatic	+	+ (shadows increased in size over 9 year period)	No surgery	
Hillerdal and Nordlund[12] 1965	22	M	LUL apico-posterior	Asymptomatic	+	No mass on roentgenogram	Apico-posterior segment bronchus blind on hilar aspect; dilated to 5–6 mm. diameter	Emphysema
Curry and Curry[7] 1966	17	M	LUL apico-posterior	Asymptomatic	+	+	Occlusion apico-posterior bronchus near its origin with distention beyond, filled with mucoid material	Emphysema
	31	F	LUL apico-posterior	Asymptomatic	+	++	Obstruction of apico-posterior bronchus LUL with bronchial distention distally	Emphysema
Hanna *et al.*[10] 1969	7	M	RML	Asymptomatic	+	No mass on roentgenogram	Dilated central bronchus ending in a blind pouch proximally, branching distally.	Emphysema
Vaghei[26] 1970	13	M	LUL apico-posterior	Shortness of breath on exercise	+	+	Blind, dilated bronchocele containing clear mucus	Emphysema
	36	M	RUL anterior segment	Asymptomatic	+	+	Cystic bronchial structure, blind on hilar aspect, branched peripherally, contained clear mucus	Emphysema
Talner *et al.* 1970	2	F	RLL superior segment	RLL pneumonia at age 10 mo.	+	Gradually enlarging ovoid mass	Subsegmental atresia (B_{X6}) with branched bronchocele containing a rubbery mucus plug	Emphysema
	14	F	LUL superior division	Asymptomatic	+	+ (mass not present on roentgenogram 1 year before)	Membranous obstruction superior division LUL bronchus with varying degrees of dilatation of distal branches	Emphysema and obstructive pneumonia

LLL=left lower lobe; LUL=left upper lobe; RLL=right lower lobe; RUL=right upper lobe; RML=right middle lobe.

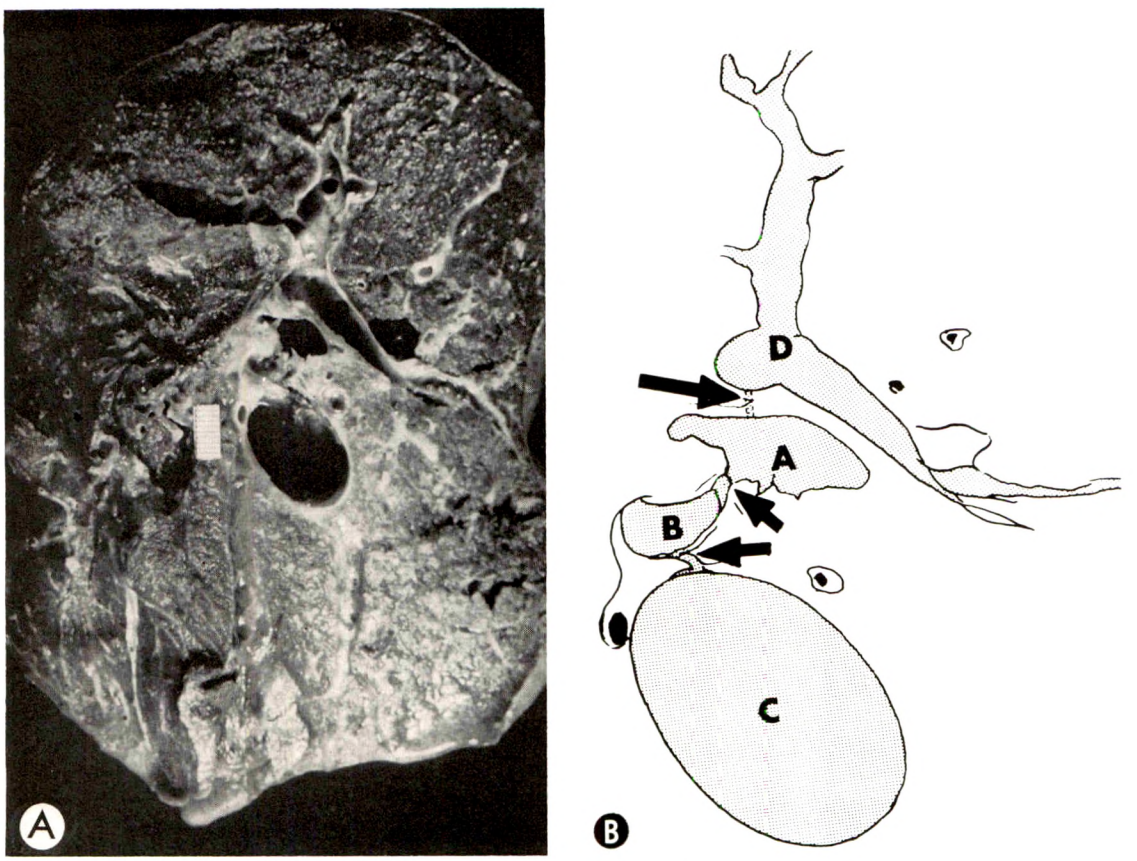


Fig. 4. Case 11. (*A*) Resected left upper lobe sectioned along the bronchi. The bulging, lighter anterior segment is emphysematous and contrasts with the atelectatic darker regions. The larger sacs in the anterior segment were filled with green, watery mucus, and an obstructive "golden" pneumonia involves the more distal lung parenchyma in the same segment. There is cylindrical dilatation of the apico-posterior bronchus and an anomalous branch of the anterior bronchus. Continuity of the bronchial tree is only suggested because the stenotic proximal portion of the bronchus has been removed. (*B*) The distorted ramifications of the bronchus to superior division are schematized. A stenosis at one point results in a dilated bronchial segment (A) just proximal to its bifurcation. The serpentine communications (arrows) from (A) to the apico-posterior and anterior bronchi resulted in cylindrical dilatation of the former (D) and large saccules in the latter (B and C).

absence of history of previous respiratory illness, and dominance of involvement of the left upper lobe (14/21 patients). A mass was apparent in the chest roentgenograms in 15 of 21 patients. In 5 an air-fluid level was demonstrated at some time during serial roentgenographic examinations.

Although the finding of regional emphysema in relation to bronchocele has received some consideration by most of the writers, this association received particular emphasis from Culiner in 1964.[4] This phenomenon was demonstrable on plain chest roentgenograms, expiratory roentgenograms and bronchograms and was confirmed at surgery when the involved segment showed air trapping as compared with normally ventilated adjacent regions.

The pathogenesis of focal air trapping associated with bronchial atresia and mucocele remains unknown, but several possibilities exist. Although interalveolar air drift through the pores of Kohn is an established physiologic phenomenon,[19,27] its clinical significance has been insufficiently appreciated. Thus, it is not common knowl-

edge that atelectasis need not develop following segmental bronchial occlusion, whether consequent to tumor, mucus, or as in the present cases, bronchial atresia with mucocele formation. It should be remembered, however, that trans-segmental interalveolar passage of air extends only to the boundaries of a lobe, so that occlusion of a lobar bronchus will always result in atelectasis if interlobar septa are intact. Recent experimental evidence suggests that collateral air drift may also occur at the respiratory bronchiole level.[16] In the light of these observations, the absence of atelectasis in the patients with bronchocele is not difficult to understand.

It has not been generally accepted that this supposedly conservative phenomenon alone could lead to air trapping and the development of emphysema. However, Culiner *et al.*[4,5,6] and Reich and Abouav,[19] on the basis of preliminary animal investigations and observations in clinical material, have concluded that complete bronchial obstruction may, in fact, lead to emphysema when the aeration of the distal parenchyma is maintained solely via collateral channels. They suggest that air trapping occurs in such regions because interalveolar drift takes place more readily during expansion of the lung than during contraction. McLean has postulated collapse of the pores of Kohn during expiration, with consequent formation of check-valve phenomena at the interalveolar channels.[17] This is an untenable explanation in the presence of normal circulation, however, unless collateral ventilation is more than a small fraction of normal ventilation —an unlikely situation.

There is an alternative mechanism which could explain the focal hyperinflation in these patients with bronchial atresia. If the patent bronchi and bronchioles which conduct the collateral air are themselves the seat of partial obstruction, then air trapping will occur distally. Since this group of patients has in common one type of bronchial anomaly, it is reasonable to ask whether or not there exist other more subtle aberrations such as immature, soft, bronchial cartilage, redundant bronchial mucosa or regions of stenosis which could result in air trapping by check-valve action. The latter two have not been observed and there is no sound evidence of the first. Although the mechanism by which air trapping develops remains unclear, its association with bronchocele is intriguing and appears to be an important diagnostic roentgenographic finding.

Atresia of a segmental bronchus is the most recently described anomaly of bronchopulmonary development.[24] In contradistinction to other bronchial anomalies (bronchogenic cyst, sequestration, displaced bronchi, supernumerary bronchi), the atretic bronchus in bronchial atresia is one segmental airway of a normal bronchial complement and has normal distal communications with the lung parenchyma.

As a result of the absence of a proximal connection, mucus accumulates resulting in dilatation of the distal bronchus and its ramifications. The bronchial dilatation may not be apparent on the initial roentgenographic examination but can be seen to develop with time, as observed in our cases and others. As the blind segment enlarges, it becomes increasingly more saccular. Its connections with the distal lung parenchyma become more distorted and appear to be too few for so large a mass. At this stage the appearance of the lesion can be misleading and misinterpretation as a bronchogenic cyst can be made.

A further consequence of bronchocele formation is the inspissation of mucus within the distal smaller air spaces. When not dissected along the plane of the bronchus, ectatic bronchioles may give an appearance that may be misinterpreted as multiple "microcysts." The distal lung parenchyma may appear golden-brown and consolidated due to an obstructive or "golden" pneumonia, the frequent result of any distal obstruction.

Total occlusion of lobar bronchi has been produced experimentally by ligation[3] and by surgical exclusion.[9] In both studies,

mucus production persisted within the ligated bronchus and caused gross bronchial dilatation and mucus plugging within 1 week. The degree of bronchial dilatation and the amount of mucus produced increased with the duration of occlusion until, at 3 months, there was a maximal dilatation with a twofold increase in bronchial volume. Some of the reported atretic bronchial segments have shown at least a fourfold increase in volume. The most rigid and inflexible component of the bronchial wall is the cartilage, the amount of which decreases corresponding to the size and more peripheral location of the bronchus. When the cartilage is present as almost complete rings, little dilatation is possible, but when cartilage is present as islands, then much greater dilatation can occur. This is supported by observations in bronchiectasis in which the second through fourth orders of segmental bronchi and not the first are ectatic. It is also possible that the cartilage may be immature, underdeveloped, or partially absent in atretic bronchial segments.

The pathogenesis of bronchial atresia remains unknown although its occurrence has been chronologically estimated. Detailed studies of bronchopulmonary development record the first appearance of segmental bronchi at the fifth week of gestation, while the adult complement of segmental bronchi is not achieved until several weeks later. Simon and Reid[24] speculated that bronchial atresia may occur as early as the fifth week of gestation, while Curry and Curry[7] suggest that it occurs after the fifteenth week; *i.e.*, after development of the segmental bronchi. The critical question unanswered is whether normal distal bronchial development can take place in the presence of abnormal proximal bronchi; if it cannot, the later time of occurrence becomes more likely.

Vascular occlusion had long been the suggested cause of intestinal atresia. In 1955, Louw and Barnard[15] experimentally produced atresia of an intestinal segment of puppies by ligation of the mesenteric arteries late in fetal development. Occlusion of the bronchial arteries which are the exclusive vascular supply of the bronchi could well produce a comparable lesion within the bronchial tree. This is questioned by Waddell *et al.*,[28] since they could not attribute with certainty any damage to the lung by occlusion of the bronchial arteries after birth. However, catheterization of bronchial arteries and subsequent occlusion by an injected toxic material has caused selective necrosis of bronchi.[8] Since branches of the bronchial arteries in part supply segments of the esophagus, their occlusion also causes focal esophageal necrosis. This supports the suggestion of Lister that vascular occlusion also produces esophageal atresia.[14] Specific evidence in the case of bronchial atresia, however, is lacking, and actual enlargement of the major bronchial artery to the lung containing the bronchocele was noted in Case 1 of the present series.

It is surprising that more of the patients with bronchial atresia did not suffer from lung infections, in view of the poor drainage of secretions from affected areas. In those patients who did come to medical attention because of pulmonary infection, it is likely that the obstructed segment provided the locus minoris resistentiae.

ROENTGENOLOGIC FEATURES OF MUCOCELE

As illustrated above, those examinations yielding the most information are inspiration-expiration chest roentgenograms, chest fluoroscopy, laminagraphy and bronchography. Localized emphysema is demonstrated by each of these techniques. Although the bronchocele in Case II was soft and contained fluid mucus, there was no observable change in size or shape with Valsalva maneuver. Bronchography demonstrates nonfilling of a bronchus, but it is often difficult to determine whether this is due to anatomic obstruction, atresia or retained secretions.

Common diseases to be considered in the differential diagnosis of oval or round midzone lesions of the type seen in Cases 1 and

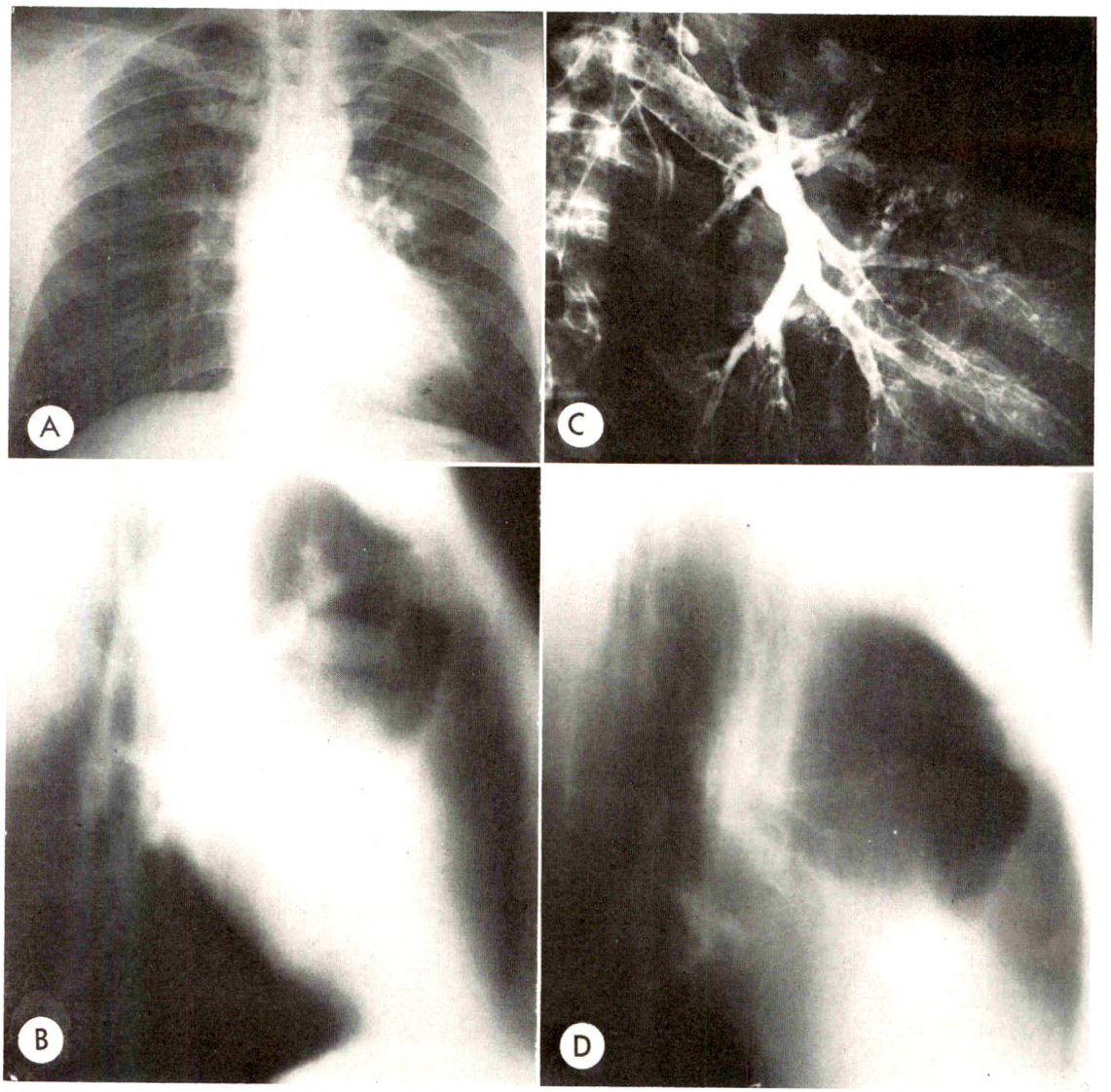


Fig. 5. Mucoid impaction. (*A*) Frontal roentgenogram of a 40 year old man who had a history of childhood tuberculosis and chronic bronchitis. He was examined at this time because of hemoptysis. A region of increased density projects over the left hilum. Hilar calcification is present. (*B*) A V-shaped, lobulated density is well demonstrated on the lateral laminagram of the left upper lobe. Proximally, there is connection to the hilar shadow, and focal hyperlucency is evident in the parenchyma about the dilated, mucus-filled bronchi. (*C*) Left posterior oblique projection of left bronchogram. Nonfilling of the distal portions of the left upper lobe segmental bronchi. Note the dilatation of the proximal 2 cm. of all 3 segmental bronchi. (*D*) The patient was treated with antibiotics after extensive hospital work-up had excluded tumor or tuberculosis. Several weeks after discharge, he coughed up a large mucus plug and repeat chest roentgenograms showed disappearance of the lobulated shadows in the left upper lobe. Lateral laminagram at this time demonstrates thin-walled, ectatic bronchi which can be superimposed on the previous radiopacities.

11 include tuberculosis and other granulomatous processes, lymph nodes enlarged because of infection or tumor, lung abscess, fluid or pus-filled bulla, bronchogenic cyst, organizing pneumonitis, and primary or metastatic tumor. The history and appropriate laboratory studies will often lead to one of these diagnoses. It is well known that interlobar encapsulated fluid may assume a rounded or oval configuration and this was

one of the diagnostic possibilities considered in Case 1. Intralobar pulmonary sequestration should be excluded with thoracic aortography.

Rarer causes of an oblong, sharply marginated pulmonary opacity include intrapulmonary lymph node, plasma cell granuloma, localized amyloid disease, limited Wegener's granulomatosis, aneurysm of a lobar pulmonary artery and pulmonary venous varix. In addition, examples of mucoid impaction of the bronchus producing similar roentgen findings have appeared recently.[2,23] Since the latter involves segmental as well as major bronchi and may be confused with bronchial mucocele, the distinguishing features are worthy of mention.

Over 100 cases of mucoid impaction of the bronchus have been reported beginning with Shaw's initial observations in 1951.[22] The syndrome is almost exclusively confined to patients with a history of asthma, chronic bronchitis or cystic fibrosis.[29] Allergy to aspergillus has been demonstrated in a high percentage of the asthmatic and bronchitic patients with mucoid impaction.[11] In some the fungus has been present within the mucus plug.[21] The essential feature of mucoid impaction is occlusion of a segmental or subsegmental bronchus by inspissated mucus leading to dilatation of the involved tributary bronchi. Coexistent pneumonia and/or atelectasis is frequently found and often the clinical picture includes hemoptysis, pleuritic pain, purulent sputum and the coughing up of mucus plugs. Localization to the upper lobes is found in approximately 80 per cent of the cases. In the chest roentgenogram, the characteristic branched or V-shaped shadow of the bronchial cast is visible at the segmental or subsegmental rather than the lobar level. As in the cases of bronchocele, persistence of aeration distal to complete obstruction is thought to be the result of collateral air drift. In the majority of cases the lung parenchyma near the mucoid impaction has shown either no change in volume or some degree of atelectasis because of hindrance to collateral air drift by the associated infection. Rarely, localized emphysema has been observed near the zone of involvement.[23] Bronchography invariably demonstrates nonfilling of the involved segments until the mucus plug is either removed by bronchial lavage or spontaneously raised by coughing.

Aside from its clinical importance as a predisposing factor to distal suppuration, bronchiectasis and abscess formation, mucoid impaction frequently simulates other diseases such as tuberculosis, lung abscess, and lung cancer. Careful history and complete work-up, including laminagraphy and bronchography are mandatory if unnecessary thoracotomy is to be avoided. The roentgenograms of a patient with mucoid impaction of the bronchus are shown in Figure 5, *A-D*.

SUMMARY

The clinical, roentgenographic and pathologic findings in 2 children with bronchial atresia and bronchial mucocele are presented and the literature on the subject is briefly reviewed.

Emphasis is placed on the derangement of regional ventilation which results in focal hyperinflation of the lung.

The similarities and differences between mucoid impaction of the bronchus and bronchial mucocele are also discussed.

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BRONCHOCELE AND BLOCKED BRONCHIECTASIS*

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IN THE last 4 years, 6 cases have come to our attention because of a tumor-like lesion which was demonstrated in the lungs of otherwise asymptomatic patients. These lesions proved to be bronchoceles and a precise roentgenologic pattern was seen.

Our purpose is to demonstrate the roentgenologic aspect of a bronchocele, or blocked bronchiectasis as it is often called. The pathologic picture will also be presented to give a better understanding of this lesion which previously has been reported in association with tuberculosis,[3,5] the chronic pneumopathies [2,9] or as bronchial atresia.[4]

REPORT OF CASES

CASE 1. B.D. was an asymptomatic 41 year old woman. A routine roentgenogram of the chest showed a mass in her left lung.

Laboratory tests and investigation for tuberculosis and mycosis were normal.

Roentgenograms of the chest revealed 3 finger-like structures in the middle third of the left lung (Fig. 1). Tomograms in both anteroposterior and lateral projections (Fig. 2 and 3) showed very clearly that these structures were contiguous and had the form of branching bronchi. The lung distal to these lesions appeared normal. Bronchography disclosed absence of filling of the superior segmental bronchus of the lingula from its point of origin. The inferior segmental bronchus of the lingula filled incompletely and was displaced downward by the dilated bronchi of the upper segment.

A lingulectomy was performed.

Macroscopic examination revealed a cylindrical mass in the lingula. Cross section showed a membrane (Fig. 4) occluding the superior segmental bronchus of the lingula a few millimeters from its point of origin. It should be noted that the blind end proximal to the membrane was not filled during bronchography, probably due to rigidity of this bronchus near the septum. Two centimeters distal to the membrane, the dilated segmental bronchus divided into 3 branches of 0.6 to 1.2 cm. in diameter (Fig. 5). These ended in blind pouches 1 cm. from the pleural surface.

Histologic examination showed normal bronchial epithelium proximal to the membrane with no inflammatory infiltration of the bronchial wall. The membrane consisted of fibromuscular tissue without inflammation. The cystic bronchi contained a viscous greenish substance negative for tuberculosis and mycosis. No epithelium was demonstrated. The walls of the dilated bronchi were in places entirely fibrotic. Two fragmented cartilaginous rings and a lone granuloma with caseous material were seen. Minimal infiltration of lym-

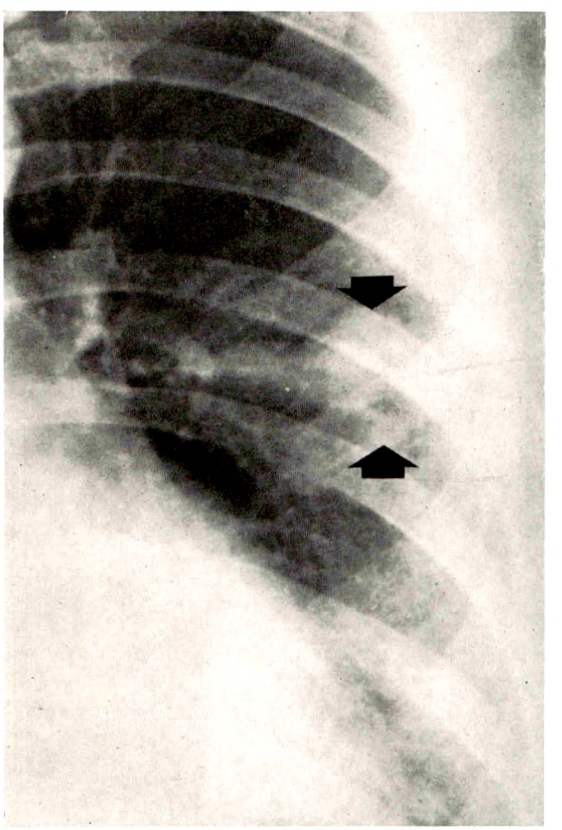


FIG. 1. Case 1. The lesion is roughly rounded in contour on the plain roentgenogram of the chest in the posteroanterior projection (arrows).

* From the Department of Radiology, Notre-Dame Hospital, Montreal, Quebec, Canada.

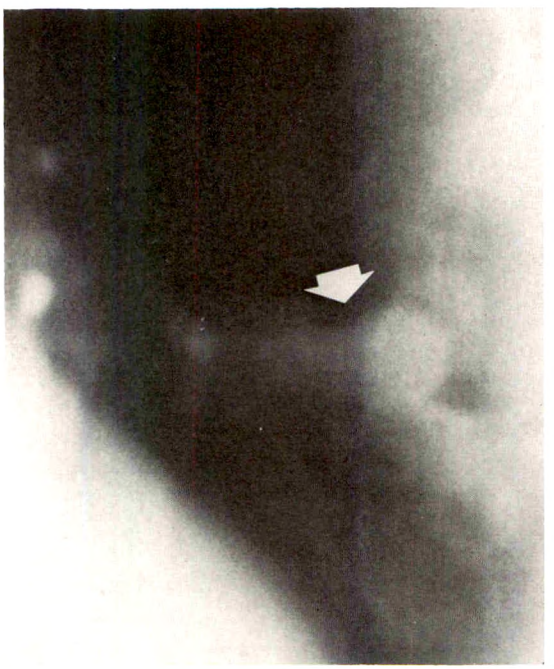


FIG. 2. Tomogram in the anteroposterior projection shows the branching pattern of the bronchocele (arrow).

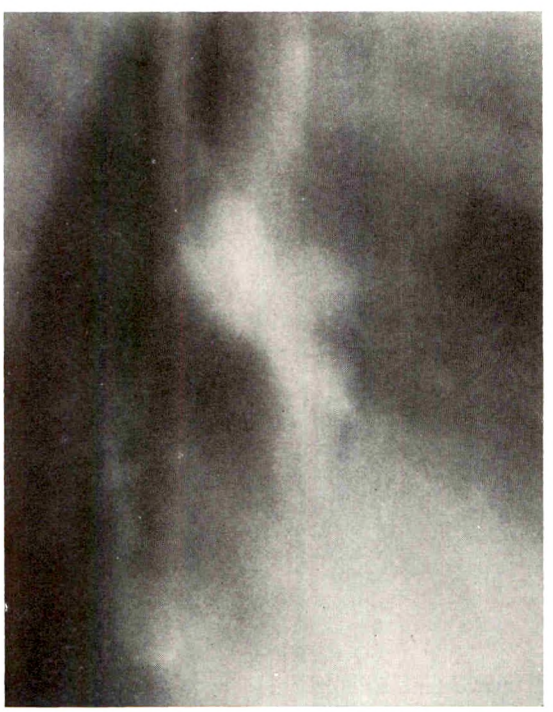


FIG. 3. Lateral tomogram reveals an entirely different pattern because the bronchi are demonstrated end-on.

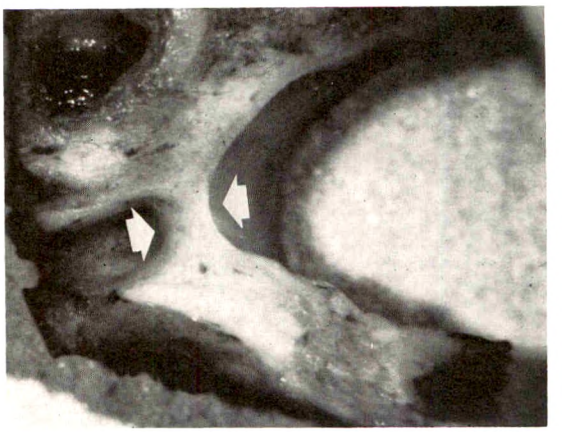


FIG. 4. The specimen is cut longitudinally and a membrane is clearly demonstrated (arrows). The cavity on the right is the proximal part of the bronchocele.

phocytes and plasmocytes was demonstrated. The surrounding lung showed atelectasis and no anthracotic pigment.

CASE II. R.P. was a 36 year old male, whose lesion was also found on a routine chest roentgenogram. Physical examination was negative.

All laboratory tests were normal including the skin tests and bacteriologic investigation for tuberculosis and mycosis.

Roentgenograms of the chest showed a faint rounded shadow of increased density (Fig. 6) superimposed on the inferior aspect of the right hilum and barely seen in the lateral projection. Tomograms in anteroposterior (Fig. 7) and lateral projections clearly demonstrated 3 ovoid structures of increased density with a branching pattern. These structures were the segmental bronchus to the posterior basal segment of the right lower lobe and 2 of its



FIG. 5. Transverse section shows the dilated bronchi.

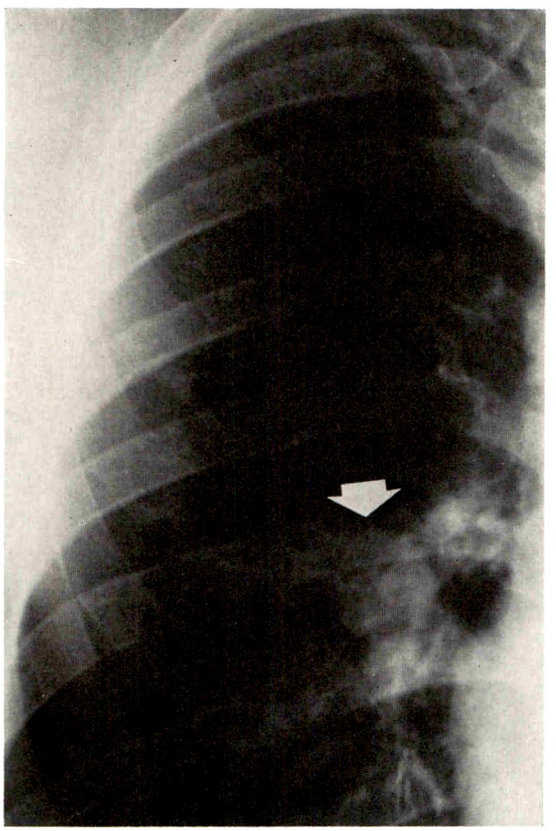


Fig. 6. Case II. The lesion appears oval on the plain roentgenogram and it is barely visualized (arrow).

branches. Bronchography, as in Case I, showed a sharp cut-off (Fig. 8), with complete absence of filling of this segmental bronchus.

The patient was operated.

Gross examination showed a polylobulated cystic mass 3 cm. in diameter filled with a viscous yellow-green substance. The walls were smooth, grayish and glistening.

Histologic examination showed a right lower lobe filled with anthracotic pigment. The cavities were lined with normal bronchial epithelium with many areas of ulceration. The right lower lobe contained many areas of fibrosis. Since the specimen was cut transversely, no membrane was demonstrated and the pathologic diagnosis was that of cystic bronchiectasis or bronchogenic cyst.

We felt that this diagnosis was not consistent with the roentgenologic findings.

Case III. N.F. was a 42 year old male with minimal symptoms of chronic bronchitis. A poorly defined lesion was demonstrated in the left upper lobe on plain roentgenograms of the chest (Fig. 9). This lesion did not change in roentgen appearance over a 2 year period. Tomograms in the anteroposterior projection very clearly demonstrated the finger-like lesions with the branching pattern almost always seen in bronchocele (Fig. 10).

Laboratory tests were normal. Bacteriology and skin tests were negative for tuberculosis and mycosis.

A left upper lobectomy was performed.

Gross examination showed a cystic trilobed structure. Transverse section revealed 3 cystic bronchi, from which thick, greenish, purulent material flowed.

Microscopic examination showed this material to be acidophilic. The dilated bronchi were lined by a cylindrical epithelium which was absent in many areas. The walls contained fibrous tissue with smooth muscle and cartilage. In some areas a lymphocytic and plasmocytic infiltrate was present, although not prominent. The specimen was not cut along its greater

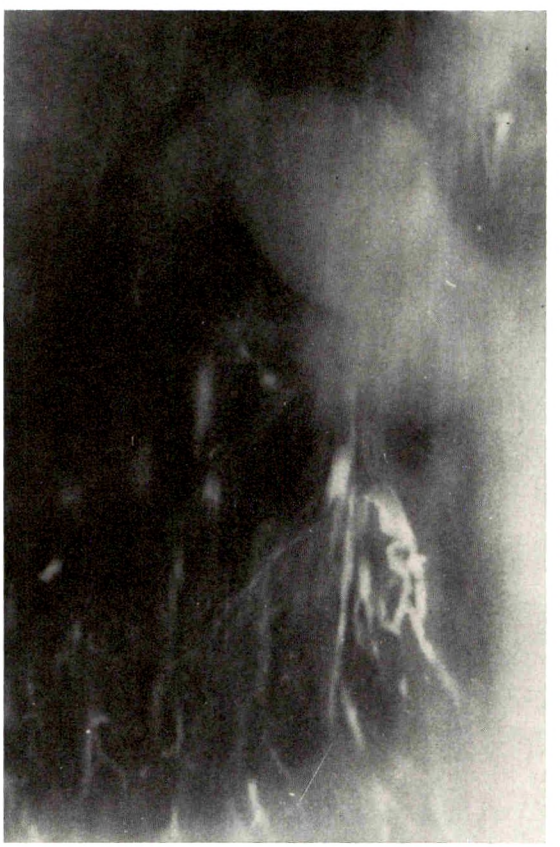


Fig. 7. Tomogram shows the typical finger-like branching pattern.

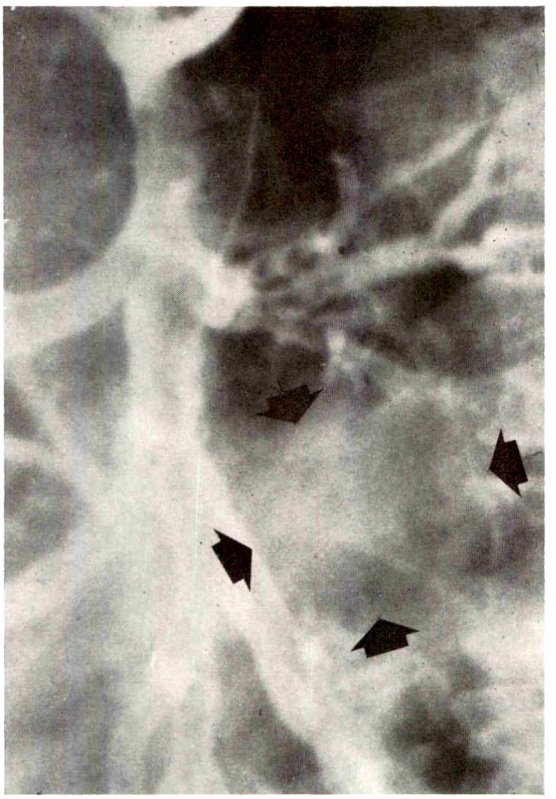


FIG. 8. Bronchogram shows the sharp amputation of the bronchus in the posterior basal segment of the right lower lobe (arrows).

axis, thus no membrane was seen. Bronchogenic cyst was the pathologic diagnosis.

We felt that this diagnosis did not correlate with the roentgenologic appearance of the lesion.

CASES IV, V and VI were 2 males and 1 female, respectively, 25, 48 and 42 years of age. All 3 were asymptomatic. Figures 11 and 12 show the tomograms in both anteroposterior and lateral projections of Case IV. In this patient the branching pattern was best seen in the lateral projection. An oblique view of Case V (Fig. 13) showed the finger-like appearance of the lesion. The only film of good quality we have of Case VI (Fig. 14) shows the oval appearance of the lesion. In the oblique projection, this same lesion had a finger-like branching appearance. Unfortunately no pathologic material was obtained from these last 3 patients.

They are presented to show that the diagnosis of bronchocele can be strongly suspected on roentgenologic examination.

PATHOLOGY

Bronchocele simply means a dilated bronchus or a group of bronchi filled with fluid. Thus, if they contain either pus or mucus, they will be called bronchopyocele or bronchomucocele.

Different theories have been put forward to explain their formation. Mayer and Rappaport[6] stated that emphysematous changes, bronchogenic cyst and bronchiectasis are all the result of developmental defects of the bronchial tree during infancy. Infection could be the main etiologic factor by creating a localized hypoplasia of the bronchial tree leading, as previously mentioned, to emphysema, bronchiectasis and cysts. Ramsay and Byron[7] also stressed the common origin of bronchocele, bronchogenic cyst and congenital bronchiectasis. They showed a septum in a bronchocele

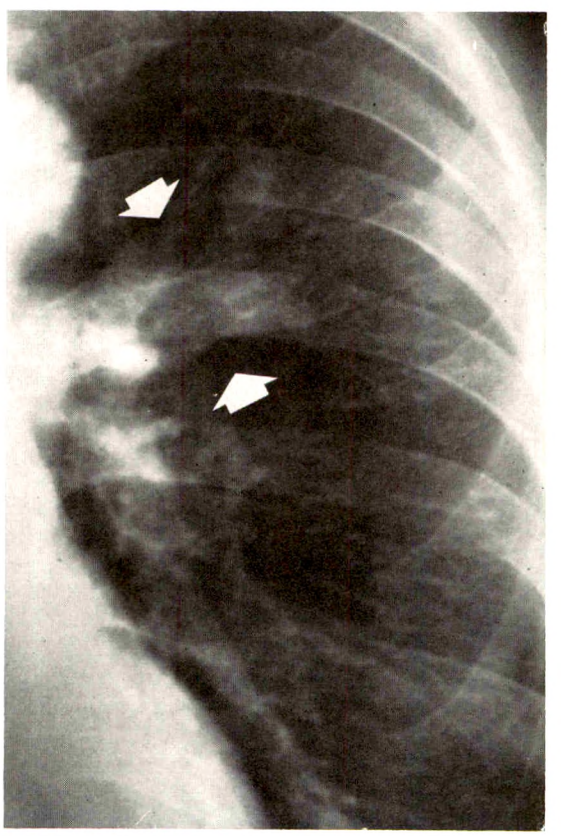


FIG. 9. Case III. The diagnosis can be suspected on the plain roentgenogram because the lesion divides similar to the bronchial tree (arrows).

and a bronchogenic cyst. In their case, the membrane was demonstrated with a probe during surgery but the pathologist failed to demonstrate it because only cross sections were made. Case 1 thus appears to be the only case in the literature we reviewed where photographic material of the septum was obtained. The most likely theory to explain the presence of this septum would be the following: development of the lung is known to continue until 7 years and probably 14 years of age by an increase in the number of lobules and not by an increase in their volume. For some unknown reason, possibly infection, a bronchial segment fails to develop properly, remains atretic and a membrane is present at this level. Such a membrane is demonstrated in Case 1 (Fig. 4). The bronchus then continues to develop normally. Mucus being continually produced dilates the bronchi distal to this membrane. This is the stage of bronchocele that is an oval or, more frequently, finger-like lesions with a branching pattern because they are really branching bronchi. The membrane can rupture, air will then be present in the bronchi and they become bronchiectatic. If the membrane remains intact, the increase in mucus will change the finger-like bronchocele into a round bronchogenic cyst. As stated previously by Israel and Chebat,[5] tuberculous lesions are often demonstrated in the walls of some bronchoceles. We think that this confirms the role that infection may play in the development of this anomaly. Unfortunately septa were not searched for in their series. Curry and Curry[4] reported 2 cases with roentgenographic lesions identical to our cases, calling them atresia.

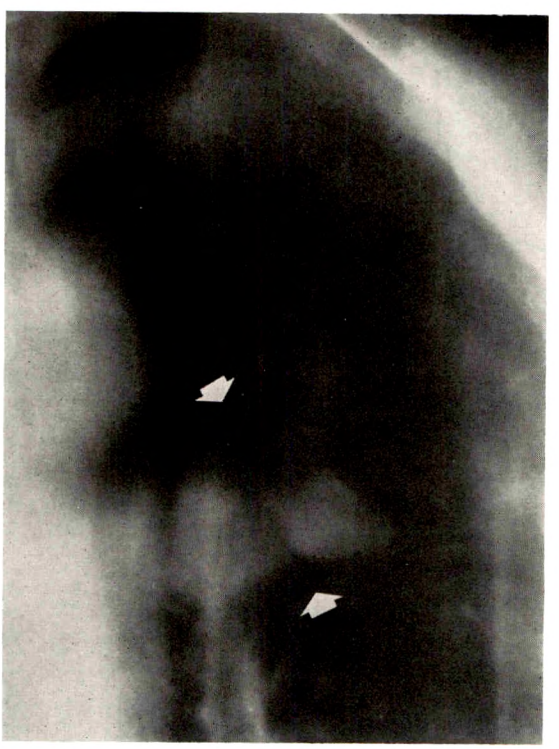


Fig. 10. Tomogram in the anteroposterior projection shows the lesion to better advantage because the incident roentgen rays are perpendicular to the longer axis of the lesion (arrows).

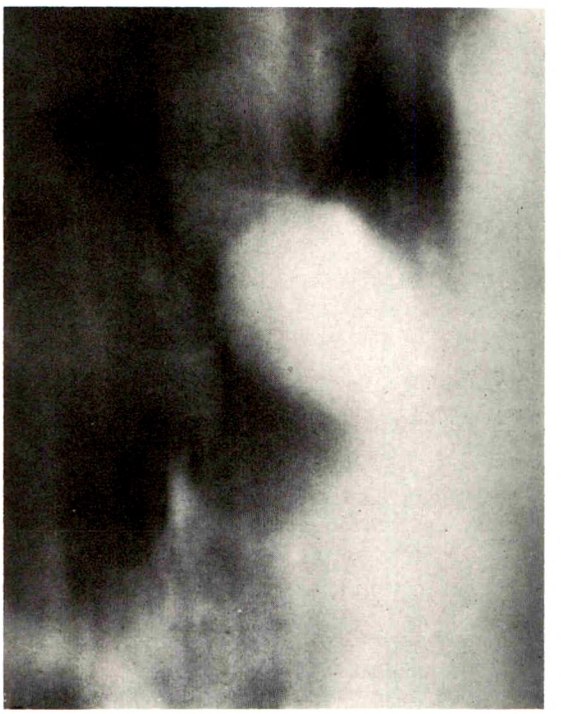


Fig. 11. Case IV. Tomogram in the anteroposterior projection. The lesion appears oval because the roentgen beam is directed parallel to the longer axis of the lesion.

It is evident that an infectious process, tuberculous or otherwise, could be only one of many possible causes of bronchocele.

The question then is whether a septum is always present in bronchoceles. It seems

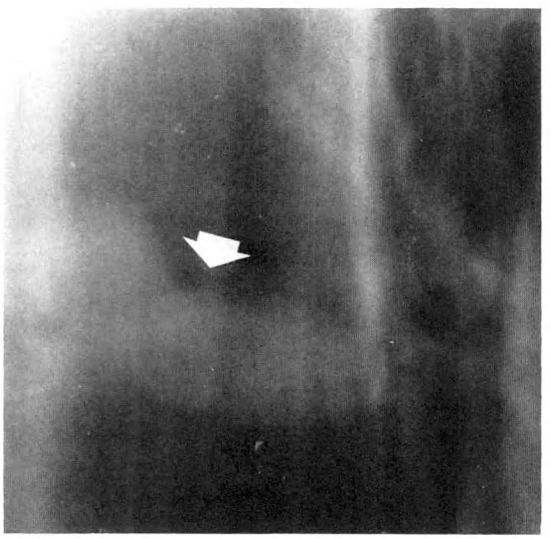


FIG. 12. Lateral tomogram shows the true finger-like appearance of the lesion (arrow).

obvious from our Case I and from the literature that the septum is unlikely to be demonstrated if the bronchocele is cut along its small axis. This question will be answered only when longitudinal sections are made in all suspected cases of bronchocele. Alveoli distal to a bronchocele are ventilated through collateral channels. Air flow is impaired and emphysema often develops distal to the lesion. On the other hand, atelectasis is sometimes present.



FIG. 13. Case V. The left antero-oblique projection was necessary to demonstrate this lesion along its longer axis (arrows).

DISCUSSION

Bronchoceles now appear to be a well differentiated roentgenologic entity. Being airless they show an homogeneous density. Their contour is fairly typical; they are ovoid or finger-like with a branching pattern. They are often very difficult to see on plain roentgenograms of the chest but are readily demonstrated by tomograms with the incident rays exactly parallel or perpendicular to the longer axis of the lesion. If the roentgen rays are parallel, an oval lesion will be shown. The branching pattern will be evident only if the roentgen rays are perpendicular. This branching pattern must be looked for since it is characteristic of the lesion. Bronchography should be performed as it will show absence of filling with a sharp cut-off of the diseased bronchus.

Two of our cases showed no change over a period of many years, nonetheless it is believed that resection should be performed because the lung distal to a bronchocele is

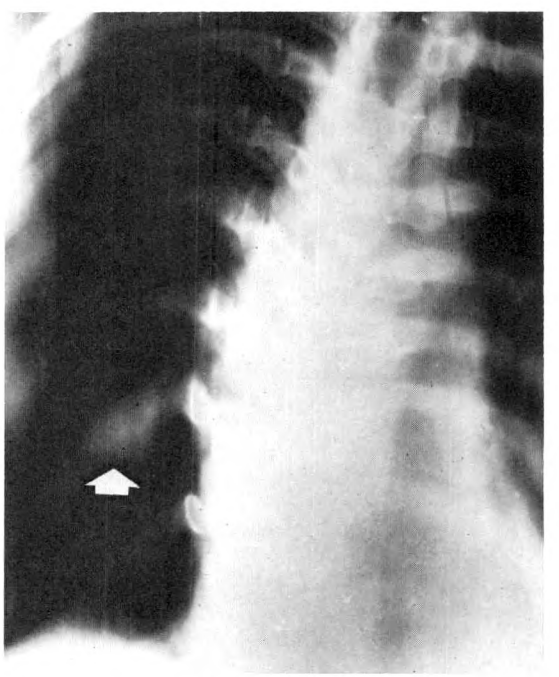


FIG. 14. Case VI. This lesion also shows an oval appearance in the anteroposterior projection (arrow).

patterns blended with anomalous bronchial epithelium." Other opinions included "multiple benign neurilemmomas," "multiple benign leiomyomas," and "metastatic leiomyosarcoma." A. A. Liebow[15] expressed the opinion that the microscopic sections of the lesion represented a slowly growing leiomyomatous type of metastasis from a possible fibroleiomyoma removed with the uterus 25 years previously. He noted that such cases have been previously described.[26]

The microscopic sections demonstrated lesions which were circumscribed by compressed parenchyma and fibrous tissue. At the periphery and sometimes extending for a short distance into the nodule were tubular structures lined by bronchiolar epithelium (Fig. 4, *A* and *B*). The nodules were composed mostly of interdigitating bundles and whorls of spindle-shaped cells which often appeared to be closely related to, or even to arise from numerous vascular channels which were present (Fig. 4, *B* and *C*). The spindle-shaped cells were acidophilic and sometimes exhibited fibrillary structures, particularly in close proximity to the vessels (Fig. 4*C*). However, the majority of cells appeared to be fibroblastic and collagen was present throughout the tumor. Connective tissue stains confirmed this preponderance of fibrous tissue elements. There was no significant anaplasia. It now is felt that these findings represent lesions which are an example of multiple nodular fibroleiomyomatous hamartomas.

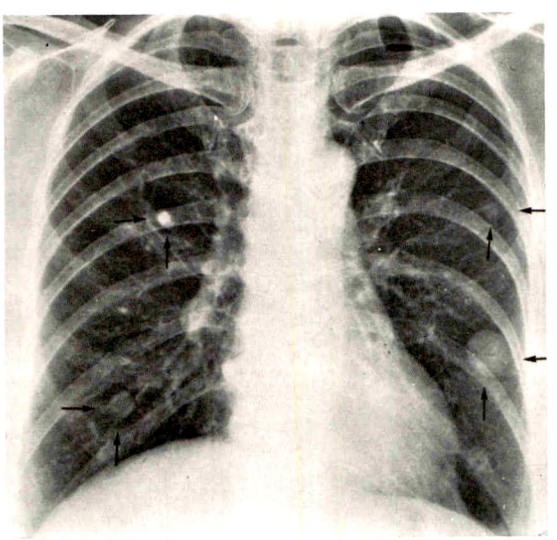


Fig. 1. *July 5, 1962.* Initial roentgenogram showing 2 well circumscribed nodules in the left lung and 2 nodules in the right lung. (The right upper lung nodule is a benign calcified granuloma.)

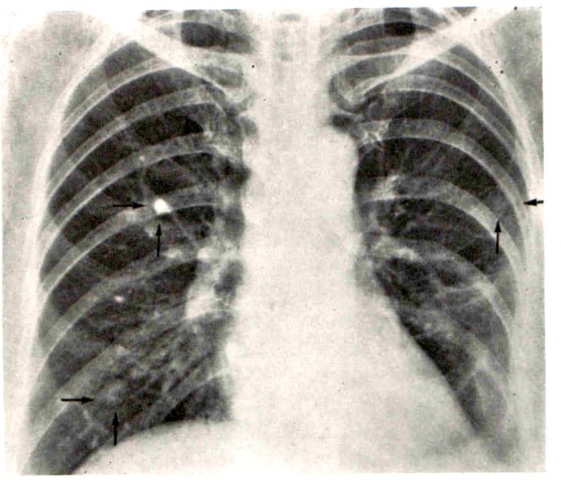


Fig. 2. *July 24, 1962.* Postoperative roentgenogram after removal of nodule at the left base with postoperative pleural reaction. Three remaining nodules in both lungs.

Roentgenograms taken immediately following the second thoracotomy in 1962 showed no change in the remaining single nodule on the left. However, several roentgenograms taken in 1966, 1967, and finally in 1968 showed a slight but definite increase in the size of the remaining nodule in the left upper lung (Fig. 5). No additional new nodules have been identified. Postoperative fibrotic scars remain in both lung fields at the sites of the previous wedge resections.

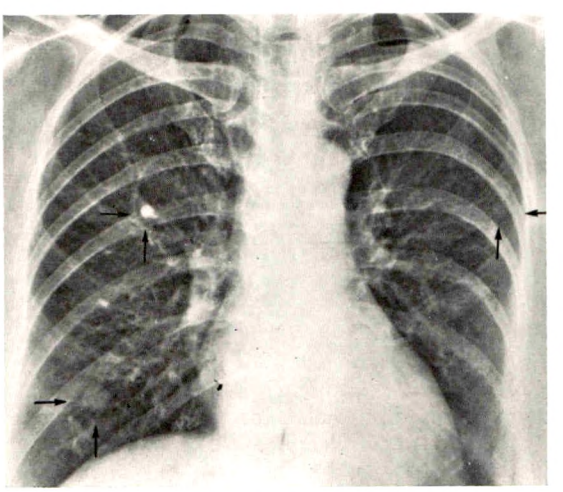


Fig. 3. *November 29, 1962.* Enlarging pulmonary nodule at right base. Other nodules remain unchanged in size.

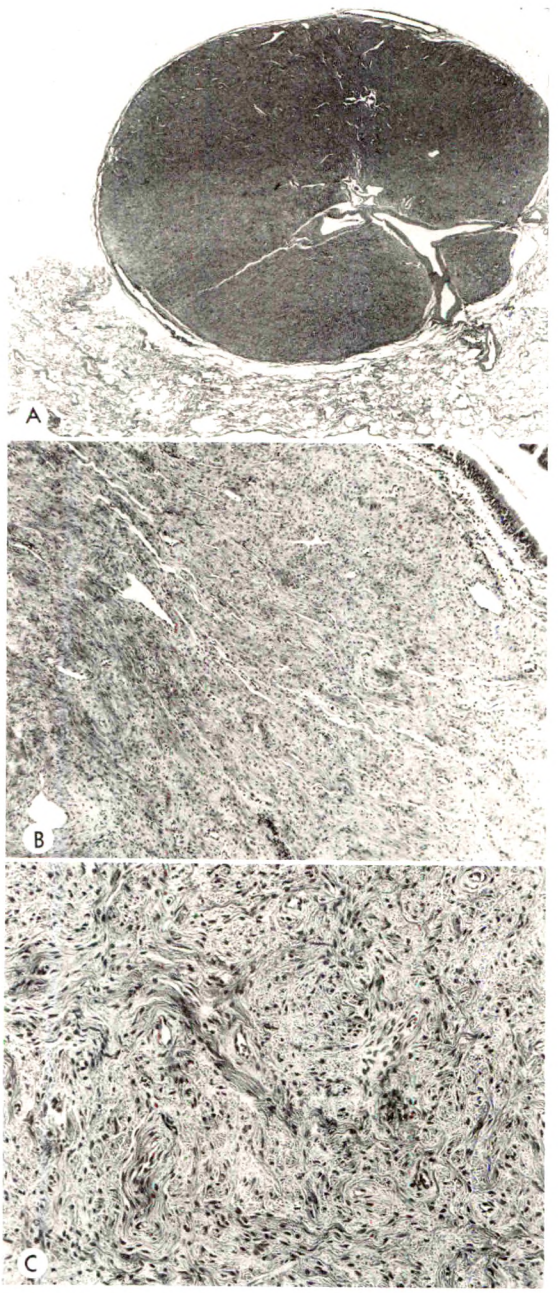


FIG. 4. (*A*) Well demarcated pulmonary tumor nodule. (Hematoxylin and eosin ×7.) (*B*) Bronchiolar structures are seen at the periphery of the nodule but not within the nodule. Whorled, interdigitating bands of smooth muscle separated by fibrous and hyalinized fibrous tissue elements. (Hematoxylin and eosin ×75.) (*C*) The individual tumor cells are spindle-shaped and possess elongated nuclei with blunt ends. The cytoplasm is eosinophilic and fibrillary. Prominent vascular channels are present. There is complete absence of anaplasia. (Hematoxylin and eosin ×150.)

DISCUSSION

The term hamartoma was introduced in 1904 by Albrecht[2] to define tumors composed of an abnormal mixture of the tissue elements which are normally present at the body site or within the organ from which the tumor originated. As noted by Keers and Smith,[13] "the abnormality could be either in the quantity, in the arrangement, in the degree of maturity, or in a combination of these faults," and "the term was so defined as to cover the majority of benign growths wherever they occurred." Although cases of multiple nodular pulmonary fibroleiomyomatous hamartomas are extremely rare, they may be recognized on the basis of their unusual clinical, roentgenographic and histopathologic characteristics.

All previously reported cases, including the present case, have occurred in adult females ranging in age from 38 to 58 years.[5,13,16,17,21,25] Only one of these patients was more than 50 years of age[5] and her pulmonary lesions were known to have been present for 22 years. Chest roentgenograms resulting in detection of the nodules were performed for "mass" or "routine" screening of 3 asymptomatic patients,[5,13,25] upper respiratory infection,[21] tracheobronchitis,[16] annual follow-up for tuberculosis,[17] and chronic bronchial asthma in the present case. Specific symptoms and physical signs related to the lesions were lacking in all of the cases described.

The present case as well as 2 other patients[16,25] had histories indicating that a hysterectomy had been performed for leiomyomata many years previously. Another patient[21] had a benign tumor of the cervix removed several years previously. With these histories, the possibility of so-called "metastasizing benign leiomyoma" has been raised by other authors.[11,18,23,24,26] In our opinion, the leiomyomas at other sites are unrelated to the lung lesions in patients with multiple nodular pulmonary fibroleiomyomatous hamartomas. Other lesions and diseases which have occurred in patients with multiple nodular fibroleiomyomatous hamartomas include a peri-

cardial cyst, basal cell carcinoma of the face and ears, benign tumor of the breast, and small hemangiomas of the face, neck and trunk[25]; diaphragmatic hernia[16]; pulmonary tuberculosis[17]; multiple colloid adenomatous goiter[5]; healed tuberculosis, diabetes mellitus, and bronchial asthma in the present case reported. Multiple leiomyomas of the skin are reported in 1 case.[5] Although leiomyomas or cutaneous hamartomas of the skin composed of smooth muscle are well recognized, there have been no previously reported multiple cutaneous lesions associated with pulmonary leiomyomas. An autosomal, probably recessive gene with about 33 per cent penetrance has been implicated in the hereditary transmission of the multiple skin lesions[5] but no studies have been made as to the possibility of hereditary transmission of the pulmonary lesions.

The multiple nodules which are visualized on the roentgenogram are usually bilateral, of uniform density, well rounded, sharply circumscribed, noncalcified, noncavitating, and have no surrounding pulmonary parenchymal reaction. As measured on the film, they vary in size from 0.5 to 4.5 cm. in diameter, although smaller nodules have been described at thoracotomy measuring only 2 mm.[5,13] Unsuspected nodules which are not seen on the usual plain roentgenogram have been well demonstrated by means of planigrams which may yield additional small multiple lesions.[16] As in the present case, nodules have been found at thoracotomy which were not visualized on the preoperative roentgenogram.[5,13]

Nodules have been noted to increase in size[5,13,17,21,25] and in number[5,17,21] over varying periods of observation. In 1 case[5] there was actual shrinkage and complete disappearance of the nodules 11 years after their onset with subsequent reappearance of nodules at a later date. Growth of solitary benign angiomatous and chondromatous types of pulmonary hamartomas as demonstrated by an increase in size on serial chest roentgenograms has been observed frequently.[12,14] The growth of these types

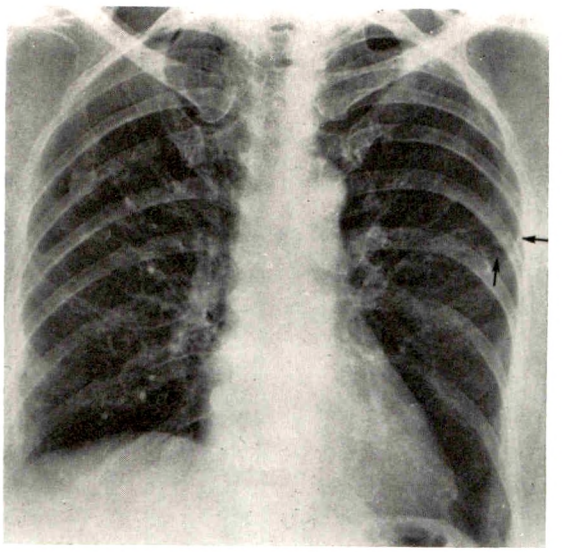


FIG. 5. *August 22, 1968.* Chest roentgenogram 6 years after initial examination. All of the nodules which have been removed surgically have not recurred. Note slight increase in size of the single remaining nodule in the left upper lung.

of hamartomas is slow and is usually demonstrable only after an observation period of more than 3 years. The rate of growth for the larger nodule is a little more pronounced than for the smaller lesions.[12] These observations might apply to multiple fibroleiomyomatous hamartomas as well. In all previously reported cases, the benign course and changing roentgenologic pattern apparently occurred without significant impairment of pulmonary function studies.

The discovery of bilateral, multiple, rounded, sharply circumscribed nodules on roentgenogram might be regarded as narrowing the differential diagnosis with a general bias in favor of malignancy. Benign tumors and developmental abnormalities with the exception of arteriovenous aneurysms are generally single. However, pulmonary arteriovenous malformations may be multiple in up to one-third of all cases. Infectious diseases such as coccidioidomycosis, histoplasmosis, tuberculosis, and hematogeneous lung abscesses should be excluded. Parasitic diseases such as Paragonimus westermani and hydatid cysts may result in bilateral pulmonary nodular lesions. Immunologic causes such as rheumatoid nodules and nodular lesions

associated with Wegener's granulomatosis, nodular sarcoidosis, beryllium poisoning, eosinophilic granuloma, amyloidosis, and even mucoid impaction associated with asthma, are also included for consideration in a differential diagnosis. Distal spread of laryngotracheal papillomatosis, lobulated nodules of plasmacytomas, and even bilateral nodules associated with lymphosarcoma have been described. Yet when one is confronted with the occurrence of bilateral, circular, well circumscribed opacities, metastatic malignancy must be primarily considered in the differential diagnosis and multiple pulmonary fibroleiomyomatous hamartomas included only as an exception to the rule that benign tumors of the lung are almost invariably single. Even after the clinical and laboratory studies have been performed, the considerable problem of differential diagnosis can often only be resolved by open thoracotomy and biopsy. The usefulness of percutaneous pulmonary needle aspiration biopsy[28] is suggested if other cases are encountered in the future, especially for verification of similar tissue changes in the remaining lesions of the ipsolateral or contralateral lung after exploratory thoracotomy.

Pathologically, the gross anatomic descriptions of the pulmonary nodules by various authors have been strikingly similar.[5,13,16,17,21,25] The nodules were well circumscribed, encapsulated, rounded or slightly lobulated, and clearly demarcated from lung parenchyma. The external surfaces were described as smooth or slightly irregular or nodular. The cut surfaces were firm, fibrous or rubbery, smooth, slightly granular or sometimes lobulated. In some cases small cysts were noted, in others the absence of cysts was mentioned. No visible cysts were noted on the sectioned surface of the present case. The recorded size of the nodules using direct measurements of the specimens ranged from 0.2 to 3 cm. in greatest dimension. The roentgenographic measurements are slightly larger than the direct specimen measurement due to the slight magnification of the projected nodules on the roentgenogram.

The previous microscopic descriptions [5,13,16,17,21,25] of fibroleiomyomatous hamartomas of the lung are similar to those demonstrated in the present case. The lesions are clearly demarcated from adjacent lung parenchyma by compressed parenchymal and fibrous tissue as demonstrated in Figure 4*A*. There are whorled, interlacing, interdigitating, interwoven or communicating bands of smooth muscle which are separated by collagen, hyalinized material, fibroblastic elements of fibrous tissue (Fig. 4, *B* and *C*). The cytoplasm is usually stated to be eosinophilic and fibrillary (Fig. 4*C*). Vascular channels are described as prominent in some of the lesions (Fig. 4*C*). Structures variously described as distorted, cleft-like spaces; duct-like structures; tubular structures; and cystic spaces lined by flattened, cuboidal, columnar, or ciliated columnar epithelium often identified as bronchiolar in type, are noted in some cases. At least 2 authors[13,16] have stated that these tubular structures appear to arise from the outer surface of the nodule and extend into the lesion. Bronchiolar structures are seen at the periphery of the nodules in our case as demonstrated in Figure 4, *A* and *B*, but are not present within the nodules. The majority of authors mention specifically that no cartilage was present. Adipose tissue was present in 1 case.[16] One author[13] reported that "the lesions appear to be the result of a developmental abnormality, they are not ordinary leiomyoma but some kind of hamartoma," and he quotes A. J. McCall as stating "many of the solid areas are similar in appearance to uterine leiomyomas." In the present case, Masson trichome stain indicated that the collagen producing element was at least as prevalent as the leiomyomatous component. In 1 case it has been postulated that lesions which became smaller in size do so through "collagenation and scarring."[5]

The complete absence of anaplasia is emphasized in our case and by most other authors.[13,16,17,21,25] Castleman,[5] however, in describing the largest nodule which was contiguous to a bronchus as "also leio-

myomatous but very cellular, with a fair number of mitoses," thought he would be remiss if he didn't call this lesion a slowly growing leiomyosarcoma rather than a benign leiomyoma. His patient[4] was still alive 7 years after his report in 1963 with no recurrence of tumors. He states that this is not surprising, because as he indicated originally, the lesion was of very low grade malignancy.

The microscopic differential diagnosis of multiple pulmonary fibroleiomyomatous hamartomas must include the following lesions which contain "spindle-cells" and might present similar histopathologic characteristics: (1) solitary leiomyoma of the lung[1,3,27,29]; (2) diffuse fibroleiomyomatous hamartomatosis (diffuse myomatosis and cyst formation) of the lung[6,7,22]; (3) peripheral and multiple bronchial adenomas,[9] especially if containing a large number of spindle-shaped epithelial cells as illustrated in Castleman's Figure 17; (4) so-called metastasizing leiomyoma (or fibroleiomyoma) of the uterus[11,24,26]; (5) possibly low-grade primary pulmonary leiomyosarcoma [20]; and lastly, and much less likely because of the clinical features present, (6) hamartomatous pulmonary lesions associated with the tuberous sclerosis complex.[19] The histopathologic characteristics of these lesions are sufficiently detailed in the references cited so that further comment is unnecessary with one exception, *i.e.*, the so-called metastasizing leiomyoma. In our opinion this lesion may not exist. Four cases of "metastasizing leiomyoma" arising in the uterus have been reported[11,24,26] and only 2 have metastasized to the lungs.[24,26] (The remaining 2 had pelvic extension of disease.) In 1 case with lung metastasis,[24] the lung lesion was solitary and was acknowledged by the authors as possibly representing a "fortuitous combination" of leiomyoma involving more than one organ system, *i.e.*, benign leiomyoma primary in the uterus and benign leiomyoma, primary in the lung. This patient remained well for 10 years following resection of the lung tumor and was subsequently lost to follow-up. In our opinion, it seems more likely that these were coincidental primary lesions in the uterus and lung and did not represent metastasis to the lung from the leiomyoma in the uterus. The other case[26] is also extremely unusual. The primary lesion in the uterus could have been a leiomyoma or might have represented intravenous leiomyomatosis as subsequently reported by others.[10] The lesions in the lung, on the other hand, are identical clinically, roentgenographically, and pathologically with those described in patients with diffuse fibroleiomyomatous hamartomatosis.[6,7,22] If the former is correct, this is the only reported case of intravenous leiomyomatosis with lung metastasis. We believe a more likely explanation is coincidental benign leiomyoma (or intravenous leiomyomatosis) of the uterus and diffuse fibroleiomyomatous hamartomatosis of the lungs. We, therefore, doubt that benign leiomyoma of the uterus ever metastasizes to the lung and would exclude this possibility from our differential diagnosis.

The prognosis to date has been uniformly good. All the cases reported have remained well. Two cases were followed for long periods prior to the removal of the nodules, one case as long as 12 years[17] and an additional case for as long as 29 years.[4,5] The present case represents a patient who is alive and well 8 years following the discovery of the initial lesion.

SUMMARY

The clinical, roentgenographic and histopathologic findings in a case of multiple pulmonary fibroleiomyomatous hamartomas are presented together with a review of 7 previously reported cases.

All patients were relatively asymptomatic females, between 38 and 58 years of age.

The histopathologic characteristics of the pulmonary nodules are presented and the differential diagnosis is discussed.

The roentgenographic findings consist of multiple, well circumscribed, noncalcified, rounded nodules in both lungs. The multiple nodules are an exception to the general

rule that benign tumors of the lungs almost invariably present as solitary lesions.

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NEPHROBRONCHOLITHIASIS*

REPORT OF A CASE SECONDARY TO RENAL LITHIASIS WITH A NEPHROBRONCHIAL FISTULA

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THE presence of broncholiths (bronchus+G. *lithos*, stone) within the lumen of the tracheobronchial tree has intrigued the medical profession since the days of Aristotle (384–322 B.C.). In 1600 A.D., Schenck a Grafenberg first reported 17 cases of broncholithiasis.[4] Broncholiths may originate within the lumen of the bronchus, but may also form in the bronchial wall, in lymph nodes surrounding the bronchus, or in the adjacent pulmonary parenchyma with subsequent erosion into the bronchial lumen.

The purpose of this paper is to describe a case of broncholithiasis of nonpulmonary origin, resulting from a complication of renal lithiasis associated with a nephrobronchial fistula, which to the best of our knowledge has not been previously reported.

REPORT OF A CASE

The patient, a 68 year old Caucasian male insurance adjuster, was admitted to the Los Angeles County-USC Medical Center with a history of a chronic cough productive of copious sputum, occasionally blood-streaked, of 25 years' duration. Four months previously he also had experienced an episode of massive hemoptysis. A chest roentgenogram at that time (Fig. 1) showed a 1 cm. rounded area of calcification overlying the inferior edge of the right heart border. In addition, there were several areas of pulmonary infiltration at the left base, 2 of which were somewhat irregularly rounded. There was no evidence of any calcifications on the left. The left heart border was sharply defined, but there was some obliteration of the left side of the diaphragm. The areas of the infiltrate were felt to lie in the left lower lobe. The remainder of the lung fields were clear.

There was also a history of drainage of a left psoas abscess approximately 27 years previously, with a persistent draining sinus in the left flank, which never completely closed. Four months previously, cultures of the material from the sinus tract revealed *Streptococcus viridans*, *Staphylococcus albus*, *Proteus vulgaris*, and *Pseudomonas aeriginosa*. Clinical and laboratory work-up for tuberculosis, coccidioidomycosis, histoplasmosis, and blastomycosis were noncontributory. Subsequent smears of the sinus tract showed numerous gram positive cocci, but no acid-fast bacilli or fungi were ever found on direct smear.

A few weeks prior to the present admission, the patient stated that he had coughed up "stones." The patient's course was rapidly downhill and he died 1 week after his admission. The chest roentgenogram 1 week prior to his death (Fig. 2) again showed the previously described single calcification in the right lower lung field, which had not changed appreciably. However, in the left lung there were 2 irregular dense calcifications just above the left leaf of the diaphragm measuring approximately 2 and 1.5 cm. in diameter. They appeared partly laminated and had somewhat of the shape of staghorn calculi. A third less well-defined oval calcification was noted, partly obscured by the overlying left half of the cardiac shadow.

The postmortem examination revealed the left lung to be completely adherent to the parietal pleura and it could only be separated from the diaphragm by sharp dissection. A sinus tract extended from the left retroperitoneal area through the diaphragm into the pulmonary parenchyma. There were several large irregular black calcified stones distributed along a nephrobronchial fistula. Three of these stones had actually penetrated the left lower lobe bronchi and lodged in the lower bronchial tree. The left lower lobe bronchus was markedly dilated and contained clotted blood. The remainder of the tracheobronchial tree, the hilar

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FIG. 1. *August 18, 1955.* Irregular noncalcified areas of infiltration at the left base. Single 1 cm. rounded calcification on the right is obscured by the overlying heart border and adjacent diaphragm.

vessels, and the tracheobronchial lymph nodes showed no significant abnormalities.

The left kidney was replaced by a large fatty mass measuring 12×20 cm. Upon sectioning this fatty mass, several pieces were identified as renal pelvic tissue containing fragmented calculi varying in size from 1 to 4 cm. In addition, several small abscesses were identified in the renal pelvic area. A sinus tract extending directly from one of the abscesses through the retroperitoneal musculature emerged through the left flank, and contained several smaller calculi.

Analysis of the calculi in the left lower lobe bronchus and calculi in the kidney pelvic area revealed similar chemical compositions. The stones were primarily inorganic and contained principally calcium phosphate.

DISCUSSION

Bronchial stones originating within the lumen of the tracheobronchial tree usually develop following the deposition of calcium about a foreign body nucleus. Broncholiths which develop in the bronchial wall occur following calcification or ossification of the elastic cartilage of a bronchus. Calcified hilar or paratracheal lymph nodes may erode through the wall of a bronchus lodging within the lumen. The most common causes of these eroding calcified lymph nodes leading to broncholithiasis are histoplasmosis and tuberculosis.[3] Pneumoliths arising in lung parenchyma may develop in abscesses, granulomas, hamartomas, ossifying adenomas, cystic calcifications, tumors, hematomas, infarcts, or silicotic masses, subsequently migrating and eroding through the tracheobronchial wall. Broncholiths may vary in weight from a few grains to as large as 139 grams,[4] and *Histoplasma capsulatum* spherules have been cultured from these calcifications.[5] The usual broncholith is hard, irregular in shape, and gray-white in color.[4] It may fragment in the process of expectoration and occasionally will show anthracotic areas. The usual composition is that of calcium phosphate or calcium carbonate in proportions similar to that of bone. In the case described, the chemical composition was primarily calcium phosphate.

The roentgenologic findings of the usual case of broncholithiasis are variable. The mediastinal or pulmonary areas may be normal. The finding of calcification in paratracheal lymph nodes, tracheobronchial elastic cartilage, or in adjacent lung parenchyma, is of aid in making the diagnosis of broncholithiasis, especially when this is suspected clinically. Occasionally a broncholith will obstruct a bronchus causing either collapse or overaeration (ball valve mechanism) of either an entire lobe or a segment of a lobe. When a bronchus is obstructed, a patient may present with a distal inflammatory infiltrate or a mass density mistaken for a neoplastic process. Tomography and bronchography will assist in demonstrating the intraluminal presence of a broncholith. If previous roentgenograms are available and an area of calcification is noted disappearing or changing in position from the original study, this is of value. If a broncholith is suspected resulting from a nephrobronchial fistula, contrast studies to show the communication between the kidney and the bronchus would be of value. Calcifications in broncholiths must be differentiated from other causes of localized intrathoracic calcifica-

tions.[2] These include calcifications in paratracheal lymph nodes, granulomas, abscesses, fibrotic scars, silicotic masses, infarcts, fungus balls, hematomas, amyloidomas, hamartomas, calcified neoplasms either primary or metastatic, and metastatic calcifications of hypervitaminosis D and hyperparathyroidism. A foreign body of metallic or calcific density must always be excluded. Calcifications in an ectopic intrathoracic kidney associated with a herniation through the foramen of Bochdalek is a theoretic consideration in a differential diagnosis, although no description of this entity could be found in the literature.

Clinically the most common presenting symptoms of broncholithiasis are cough, hemoptysis, and expectoration of a lung stone.[3] Although the case described presented with all of these findings, the association of the broncholith formation with a nephrobronchial fistula and renal lithiasis is quite unusual. Renal fistulas usually result from urinary tract infections of long-standing duration with abscess formation. Tuberculosis must always be considered in a differential diagnosis, but surgical procedures on the kidney and, rarely, trauma or tumors may also cause renal fistula formation. Fistulas between the kidney and pleural cavity, lung, or bronchus are almost always the result of infections.[1] Nephrobronchial fistulas, although rare, have been described, but as far as can be determined, this is the first reported case of broncholithiasis secondary to a nephrobronchial fistula.

SUMMARY

A case of nephrobroncholithiasis secondary to a nephrobronchial fistula and renal lithiasis is described. The usual symptoms of broncholithiasis, namely, cough, hemoptysis, and expectoration of calculi, were present. The roentgenologic findings of calcifications in the lower lung field resembling renal calculi associated with a draining sinus in the flank should lead one to suspect the diagnosis of nephrobroncholithiasis.

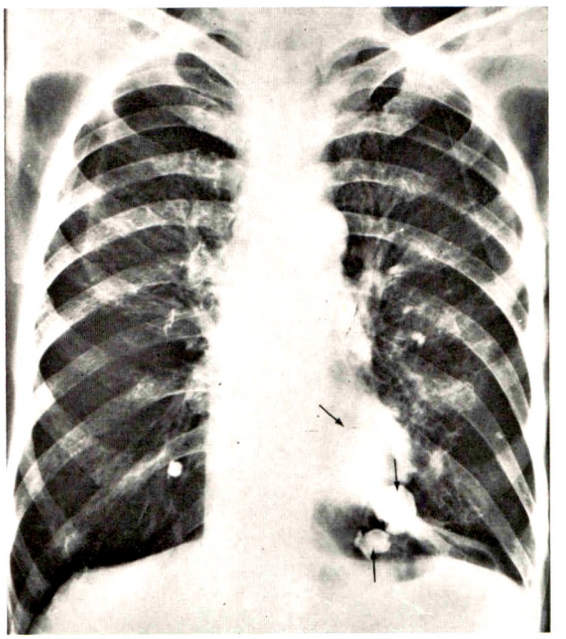


FIG. 2. *December 7, 1955.* Over a period of 4 months, 2 well defined, irregularly rounded, partly laminated calcifications have appeared above the left leaf of the diaphragm, bearing a strong resemblance to renal calculi (lower 2 arrows). Poorly visualized oval calcification is obscured by the overlying left heart border (upper arrow). Small calcification on the right is better visualized, and is unchanged since the previous examination.

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A CHARACTERISTIC PULMONARY FINDING IN UNILATERAL COMPLETE BRONCHIAL TRANSECTION*

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THE present case is described to corroborate the pulmonary finding reported previously as a specific roentgenologic sign for traumatic transection of a mainstem bronchus.[1]

REPORT OF A CASE

A 26 year old man sustained a closed chest trauma in an automobile accident and was brought to the Emergency Ward of the Massachusetts General Hospital shortly thereafter. Upon arrival, he was comfortable at rest. His vital signs were stable. There was no period of severe dyspnea.

An upright posteroanterior chest roentgenogram (Fig. 1) was obtained. This demonstrated fractures of the right first, second, third, and fourth ribs, a right hydropneumothorax, fracture of the left clavicle, subcutaneous emphysema, and total collapse of the right lung which fell away from the mediastinum and right main-stem bronchus.

At thoracotomy, the right upper lobe bronchus and intermediate bronchus were completely transected. The right upper lobe lay separated from the rest of the lung. The major vessels were intact. Both bronchi were successfully reconstructed.

A follow-up upright posteroanterior chest roentgenogram (Fig. 2) showed a well-expanded right lung with minimal postsurgical change.

DISCUSSION

Because of complete transection of both the right upper lobe bronchus and the intermediate bronchus, the collapsed right lung fell away from the mediastinum and right main-stem bronchus, and toward the most dependent site, in this case the right hemidiaphragm.

The prior case report[1] pointed out a previously unreported sign of complete transection of a main-stem bronchus: the affected lung, having lost the anchoring support normally provided by the main bronchus, falls away from the mediastinum rather than collapsing toward it as with a pneumothorax. The affected portion of the lung will fall to the most dependent site, depending on the patient's position. In the previously reported case the patient's clinical state permitted only supine roent-

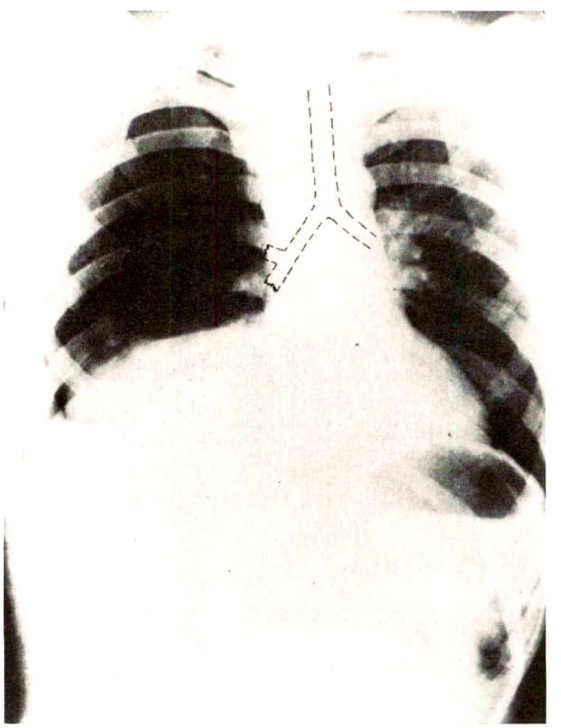


FIG. 1. Traumatic transections of the right upper and intermediate bronchi shown on roentgenogram taken in the upright position. The superior border of the collapsed lung has fallen well below the level of the right main-stem bronchus.

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genograms; the affected lung fell posteriorly and laterally.

In the present case, upright roentgenograms were possible. Since both the upper lobe and intermediate bronchi were transected, the collapsed right lung fell toward the right hemidiaphragm. The diagnosis of transection of the main-stem bronchi can be made with confidence because the superior margin of the collapsed right lung has fallen below the level of the take-off of the right upper lobe bronchus from the right main-stem bronchus (Fig. 1).

In cases of uncomplicated pneumothorax with complete lung collapse, the superior margin of the collapsed lung necessarily lies at or above the level of the intact upper lobe bronchus take-off to which it is attached (Fig. 3).

Apical and lateral pleural adhesions would prevent the lung from falling freely into the pleural cavity even if the main-stem bronchus were transected. Hence, the absence of the "falling lung" sign in the upright position does not rule out transection.

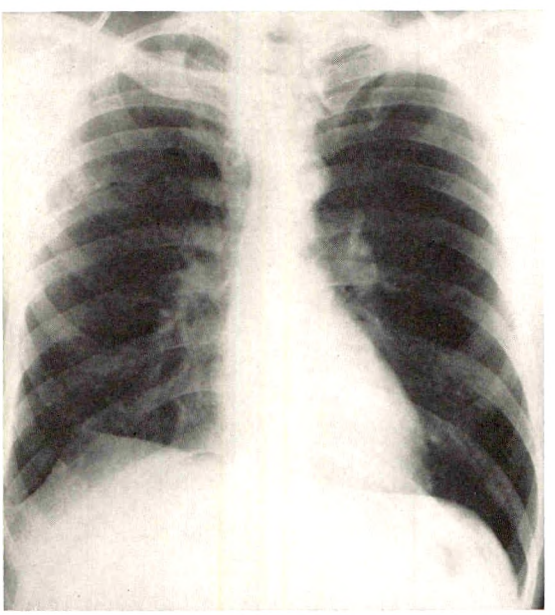


Fig. 2. Roentgenogram taken 6 years after repair of the transected bronchi. The chest shows only post-traumatic and postoperative changes. The right lung is well expanded.

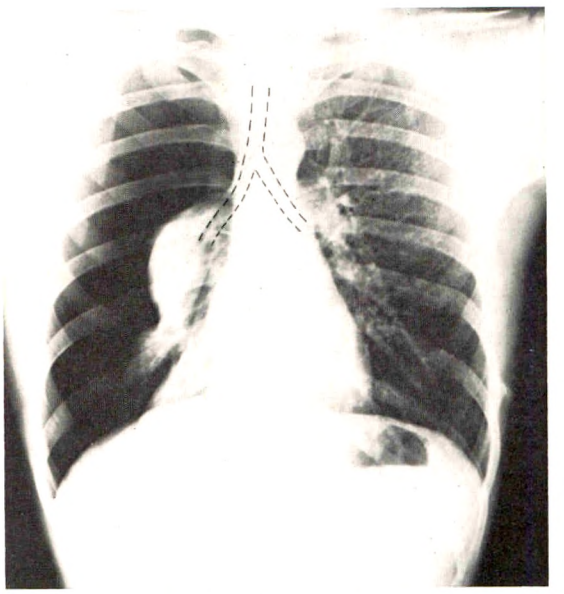


Fig. 3. An example of uncomplicated pneumothorax. The superior margin of the collapsed right lung lies at the level of the right main-stem bronchus.

The previously reported signs of traumatic transection of the bronchus include: (1) pneumothorax; (2) fracture of the upper ribs on the side of the pneumothorax; (3) subcutaneous or mediastinal emphysema; (4) mild dyspnea without cyanosis; (5) sharp angulation or obstruction in the course of an air-filled bronchus; (6) air surrounding the bronchus; and (7) pulmonary infiltrate (caused by aspirated blood or by hemorrhage into the lacerated lung).[1–3,5,6] These are indirect signs. The presently described sign, when present either in upright or supine roentgenograms, is a direct sign of complete transection.

SUMMARY

A corroborative case of traumatic complete transection of the right upper lobe and intermediate bronchi is reported.

As in the previously reported case, the lung fell away from the mediastinum toward the most dependent portion of the thorax. In the upright roentgenogram, the superior margin of the collapsed right lung lay below the level of the transected bronchi. In an uncomplicated pneumothorax, the superior border of the collapsed

lung must remain above the level of the main-stem bronchus.

The previously reported findings in traumatic transection of a main bronchus are all indirect. The "falling lung" sign gives direct evidence for traumatic transection of the bronchus.

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THE ROENTGEN MANIFESTATIONS OF THORACIC ACTINOMYCOSIS*

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THE demonstrated effectiveness of penicillin and other antibiotics in actinomycosis in recent years emphasizes the importance of the early accurate diagnosis of this disease. The past mortality rate of nearly 90 per cent[23] has been reversed with modern treatment and a 90 per cent cure rate is now possible.[15]

However, the diagnosis still remains a challenge for the radiologist as well as for the clinician. No recent updating of the roentgen signs of this disease has appeared to keep pace with the therapeutic advances. This is the purpose of the present report, which is based on 15 cases encountered at the University of Cincinnati Medical Center in which chest roentgenograms were still available.

MICROBIOLOGY

Important studies by Erikson,[10] Thompson,[32] and Pine *et al.*[25] have cleared up much of the confusion surrounding the identity of the organism that causes actinomycosis in humans. It is now universally accepted that most of these infections are caused by *Actinomyces israeli*, a frequent oral inhabitant of healthy people. It is a different species from *Actinomyces bovis*, the cause of lumpy jaw in cattle. Waksman and Henrici[34] have classified the *Actinomyces* with respect to other closely related organisms. In addition to *A. bovis* and *A. israeli*, 3 other species, *A. naeslundi*, *A. ericksoni*, and *A. propionicus*, have been isolated from human material (sinuses, pleural fluid, lung abscess).[6] Thompson[32] has pointed out that the species are not host specific, since some bovine infections are definitely caused by *A. israeli*, and a few human cases are caused by *A. bovis*. All are nonacid-fast anaerobes.

Closely related to the *Actinomyces* is the genus *Nocardia*, the cause of nocardiosis. These organisms are acid-fast and most species are aerobic, so that they are easily distinguished from the *Actinomyces*.

In spite of the clinical resemblance of actinomycosis to fungal infection, and notwithstanding the mycelial form of the organism which originally led to its name (ray fungus), the actinomycetes have been firmly established as bacteria.[8] The more important biologic properties on which this is predicated are: lack of a nuclear membrane, the constituents of the cell wall, and the almost universal sensitivity to ordinary antibiotics and insensitivity to the polyenes (amphotericin B, nystatin). Nevertheless, like almost all physicians, we consider actinomycosis a fungus disease, stubbornly maintaining that it behaves like one, if not therapeutically, certainly clinically and roentgenologically.

EPIDEMIOLOGY

Actinomycosis is a worldwide disease and no age, sex, or race is immune. Bates and Cruickshank,[2] in a review of 85 cases of thoracic actinomycosis, found 2 peak age periods—11 to 20 and 30 to 50. Men are affected about 3 times as often as women. Earlier reports of a higher incidence in rural areas have not been substantiated by recent experiences.[2,15]

PATHOGENESIS AND PATHOLOGY

Primary pulmonary actinomycosis is believed to arise from inhaling infected material from the teeth, tongue, or tonsils. Local and systemic conditions necessary for the establishment of clinical infection remain conjectural.

The primary lesion may appear in the

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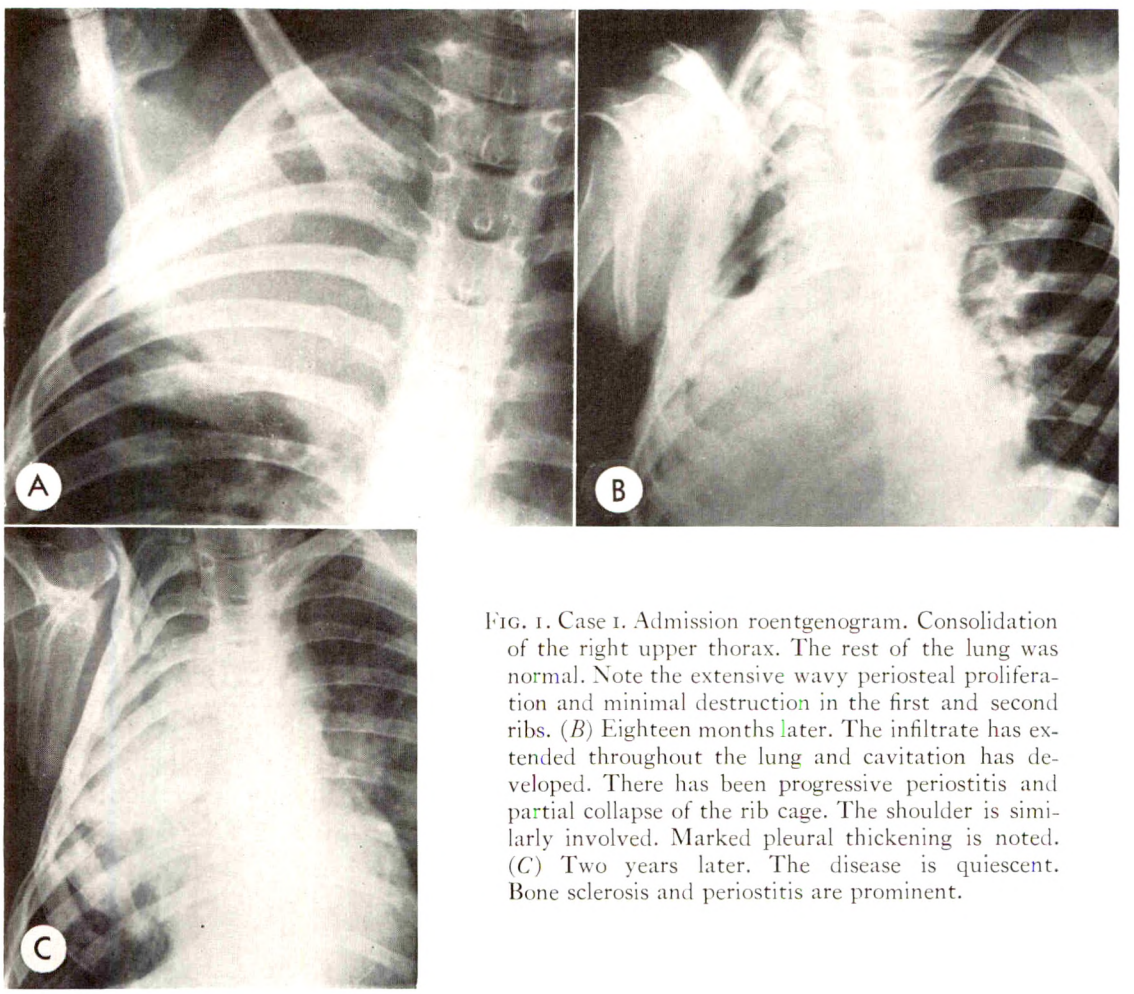


FIG. 1. Case 1. Admission roentgenogram. Consolidation of the right upper thorax. The rest of the lung was normal. Note the extensive wavy periosteal proliferation and minimal destruction in the first and second ribs. (*B*) Eighteen months later. The infiltrate has extended throughout the lung and cavitation has developed. There has been progressive periostitis and partial collapse of the rib cage. The shoulder is similarly involved. Marked pleural thickening is noted. (*C*) Two years later. The disease is quiescent. Bone sclerosis and periostitis are prominent.

peribronchial tissue, in the bronchioles, or in the alveoli. It consists of a central region of suppuration containing typical colonies of *Actinomyces* ("sulfur granules"), surrounded by granulomatous reaction.

Spread is generally by direct invasion, without regard for tissue planes and boundaries. The pleura, including the interlobar septa, is often involved and shows marked fibrosis and thickening, especially adjacent to the pulmonary focus.[30] Extension through the parietal pleura is frequent and results in chest wall lesions. Mediastinal infection may result from penetration of the tracheobronchial wall by a mucosal lesion or via direct extension from the lung. Tracheoesophageal fistula sometimes occurs. The pericardium and heart may also become infected by direct extension. Dutton and Inclan[9] collected 86 cases of cardiac involvement, an incidence of about 2 per cent. Involvement of the spine by direct extension is not uncommon.[5,7]

Erosion into a blood vessel sometimes produces hematogeneous spread to distant organs, such as the brain.[4] A disseminated miliary form also occurs.[12,28]

Secondary thoracic involvement may occasionally result from cervicofacial or abdominal actinomycosis. Cecal actinomycosis may spread to the liver via the portal venous system and then extend directly through the diaphragm into the right lung base.[28] Of the 85 patients with thoracic actinomycosis reported by Bates and Cruickshank,[2] 70 were primary in the chest and 15 were secondary to cervicofacial or abdominal involvement.

CLINICAL FINDINGS

Actinomycosis is a chronic granulomatous disease characterized by suppuration, abscess formation, and draining sinuses. It has been classified into 3 clinical forms: cervicofacial, thoracic, and abdominal. Each, of course, has its own findings, but we will consider here only the thoracic manifestations. Symptoms usually arise late in the course of the disease because of the relatively low virulence of *A. israeli*. The major clinical findings in our 15 patients were cough,[12] low grade fever,[12] weight loss,[9] and pain.[9] Hemoptysis was infrequent. Chest wall swelling was present in 8 patients, whereas draining sinuses were noted in only 5.

Signs of congestive failure, endocarditis, constrictive pericarditis, or pericardial effusion are occasionally encountered, indicating cardiopericardial extension of the disease.[9] Similarly, neurologic symptoms and signs may signify spinal involvement.

REPORT OF CASES

Case I. An 11 year old white girl was first admitted to the Cincinnati Children's Hospital in April 1942, with complaints of back pain, fever, and cough for 4 weeks. There was tenderness and soft-tissue swelling over the right suprascapular region, axilla, and anterosuperior chest wall. Rales and percussion dullness were noted over the right upper lung. She had evening temperature elevation up to 102° F. Except for mild anemia and moderate leukocytosis, laboratory studies were normal.

Roentgen examination of the chest (Fig. 1*A*) revealed consolidation of the entire right upper lobe. The right upper ribs showed extensive periosteal reaction with areas of destruction.

The suprascapular swelling was incised and drained, revealing typical sulfur granules of *Actinomyces*. Lipiodol injection of a persistent draining sinus failed to show connection with the pleura or mediastinum.

Blood transfusions, radiation therapy, sulfanilamide, and Lugol's solution brought about no improvement. Nine months later, more draining sinuses of the chest wall appeared and communications with the esophagus and a bronchus were demonstrated. Cavitation developed in the right upper lobe and pronounced destruction of the shoulder girdle was now evident roentgenographically (Fig. 1*B*). Signs of pulmonary osteoarthropathy appeared.

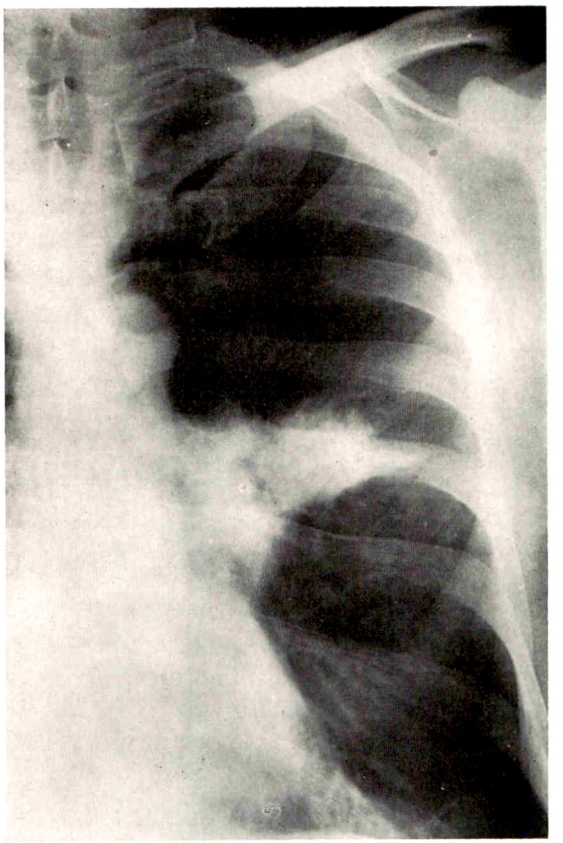


Fig. 2. Case II. Left parahilar density is seen tapering toward the lung periphery. Laminagraphy showed a tiny cavity in it. Bronchogram was normal. (Courtesy of Dr. Jan Schwarz and Dr. Gerald Baum,[29] reproduced with permission of *Seminars in Roentgenology*.)

Sulfamerazine and small doses of penicillin, which had just become available, resulted in marked improvement (Fig. 1*C*) and eventual complete control of the disease.

Comment. This case demonstrates many of the clinical and roentgenographic manifestations of this disfiguring infection. The contiguous involvement of lung, pleura, mediastinum, and thoracic wall is almost diagnostic. The wavy periostitis at the site of bone lesions is also typical. Pulmonary osteoarthropathy, an uncommon finding, reflects the chronicity of the intrathoracic process.

Case II. This 50 year old white man was admitted to the Cincinnati Jewish Hospital with

chills and cough of 5 weeks' duration. Temperature was 101° F. The physical examination was unremarkable.

The chest roentgenogram demonstrated a density in the left parahilar area tapering toward the lung periphery (Fig. 2). Laminagraphy confirmed the presence of a tumor-like mass and demonstrated a tiny cavity, but added no further information. Bronchography demonstrated patency of all the major bronchi. Bronchoscopy was normal. Sputums were negative for tubercle bacilli and tumor cells. A scalene lymph node biopsy revealed no tumor. Left upper lobectomy demonstrated a chronic pneumonia with numerous abscesses containing typical actinomycotic granules. The patient made an uneventful recovery.

Comment. In 5 of our patients and in all 7 patients of Bates and Cruickshank[2] with lung resection, the diagnosis was first established by operation. Others have reported a similar experience.[21,22,26,33] Although resection is not the treatment of choice for actinomycosis, operation for possible malignancy should not be delayed because of the outside chance of encountering this disease.[21] The bronchographic demonstration of normal bronchi entering the lesion (the open bronchus sign[11]) should have excluded neoplasm in this patient.

CASE III. This 46 year old Caucasian waiter was admitted with a 6 weeks' history of fever, weight loss, cough, and expectoration of copious sputum, occasionally blood-tinged. His temperature was normal. Laboratory examination revealed a white blood cell count of 26,900 with 63 per cent polymorphonuclear leukocytes. Sputum and gastric washings were negative for tubercle bacilli and fungi.

Roentgenograms of the chest (Fig. 3*A*) revealed extensive infiltrate in the upper two-thirds of the right thorax containing many small cavities. Elevation and tenting of the right hemidiaphragm and slight shift of the trachea to the right were present. Pleural thickening was noted over the right upper lobe. Slight periosteal elevation was seen along several ribs. Bronchography demonstrated irregular narrowing and distortion of the right main bronchus with almost complete obstruc-

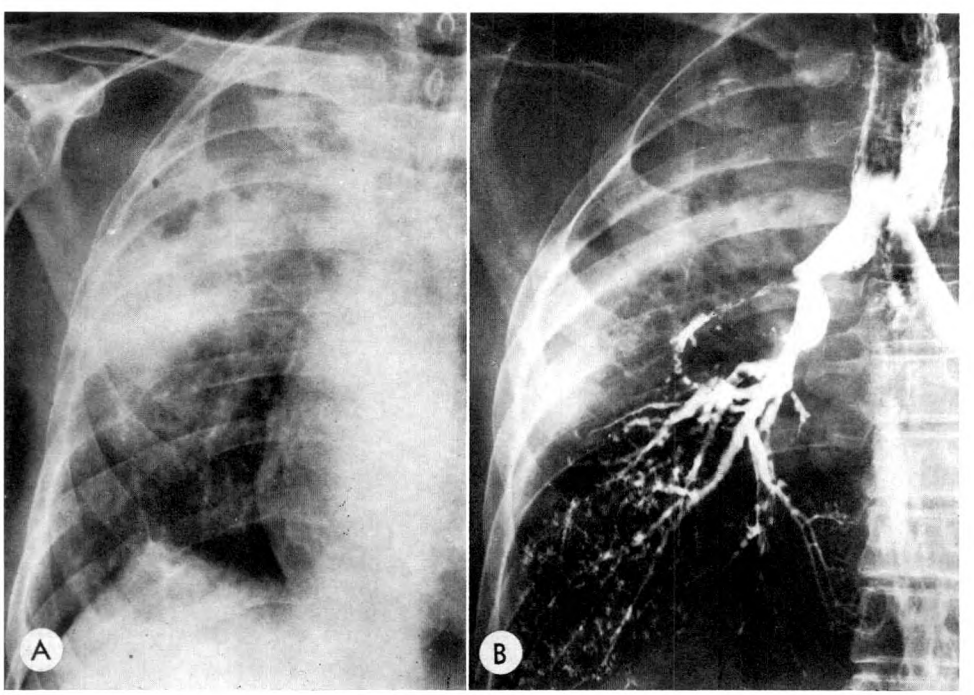


FIG. 3. Case III. (*A*) Extensive infiltrate is present with cavitation in the right upper lung. Also present are slight periostitis of the upper ribs, pleural thickening, and elevation of the right hemidiaphragm. (*B*) The bronchogram demonstrates irregular narrowing of the right main bronchus with almost complete obstruction of the upper lobe bronchus.

tion of the upper lobe bronchus (Fig. 3*B*). Lateral laminagrams indicated that the lesion had crossed the major fissure into the lower lobe. A diagnosis of actinomycosis was suggested.

Bronchoscopy revealed irregular granular thickening of the mucosa of the right main bronchus with partial obstruction. Bronchial biopsy showed chronic inflammatory fibrosis. On exploratory thoracotomy, obliteration of the pleural space over a consolidated right upper lobe was found. The right bronchus was tightly bound down by inflammatory tissue. A pneumonectomy was performed.

Individual lobes could not be identified grossly because of the extensive fibrosis and contraction. Histologic sections showed severe pneumonitis with bronchiectasis, marked pulmonary fibrosis, and multiple abscesses containing ray fungus.

Comment. Although some of the roentgen features (specifically, the violation of the fissure and the periostitis) favored fungus disease, the bronchoscopic and bronchographic findings were strongly suggestive of a tumor, so operation was performed.

Case IV. This 56 year old alcoholic man was admitted with a history of epigastric pain, chronic productive cough, weakness, and 25 pound weight loss over an 8 month period. His temperature was 99° F. Right anterior cervical and bilateral axillary lymph nodes were palpable. There was clubbing of the fingers. Laboratory studies were normal.

Chest roentgenograms revealed large bullae bilaterally. A left parahilar mass was noted (Fig. 4*A*). Bronchography showed slight narrowing of the left lower lobe bronchus with incomplete filling of the superior segment bronchus. The basilar segments were slightly bronchiectatic (Fig. 4*B*).

Bronchoscopy was normal, as was scalene lymph node biopsy. Sputum smears and cultures were negative for tubercle bacilli. One sputum smear was reported as showing granules composed of branching hyphae.

Left thoracotomy revealed a firm, irregular mass, approximately 6 cm. in diameter, in the superior segment of the left lower lobe with many enlarged lymph nodes around the hilum. Left lower lobectomy was performed. Histologic sections showed caseating granulomas consistent with tuberculosis in the lymph nodes; the superior segment lesion showed extensive, acute, and organizing pneumonia with abscess formation and granulomatous reaction. Colonies of *Actinomyces* were grown from the pus in the abscesses.

Postoperatively, the patient received penicillin, sulfadiazine, and isoniazid. He was doing well 4 years later.

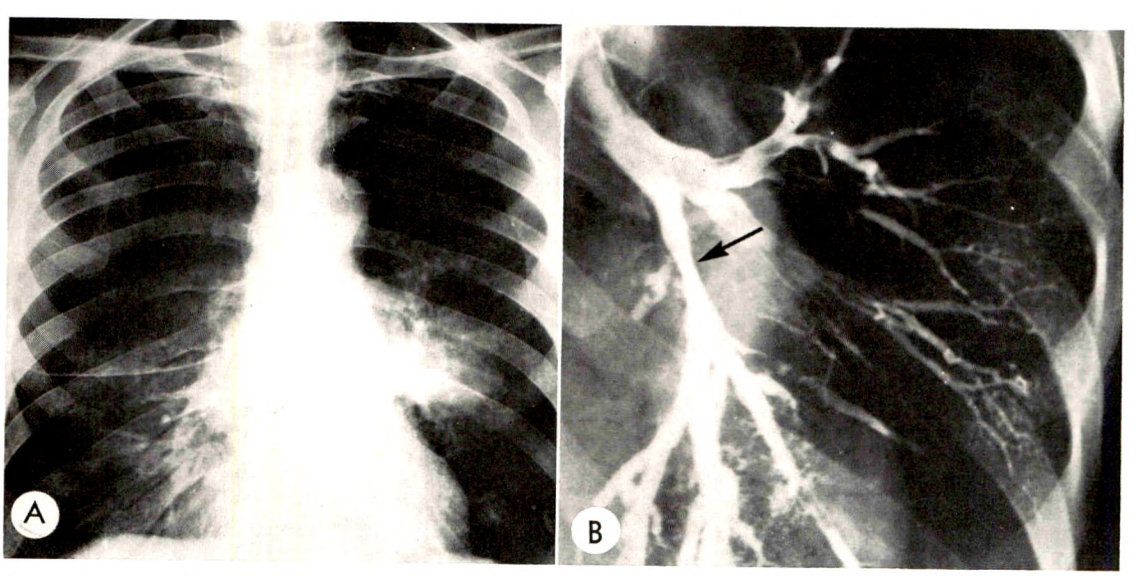


Fig. 4. Case IV. (*A*) Extensive bullous disease is noted bilaterally. There is a left parahilar lesion. (*B*) The bronchogram demonstrates slight narrowing of the lower lobe bronchus (arrow) with incomplete filling of its superior segment and slight bronchiectasis of the basal segments.

Comment. This patient certainly had actinomycosis and probably coexisting tuberculosis. At least 4 cases with this combination have been reported.[2,19] In another patient in our series, tuberculosis developed in the same region that had been involved by actinomycosis 9 years earlier.

ROENTGEN MANIFESTATIONS

Table 1 summarizes the roentgen findings in our 15 patients. Pulmonary involvement took the form of a mass (Fig. 2; and 4, *A* and *B*), chronic fibrocavitary involvement (Fig. 5, *A* and *B*), or chronic alveolar infiltrate (Fig. 1, *A–C*). The size ranged from a small patch to complete involvement of a whole lung. Extension across a fissure into adjacent lobes was not uncommon (Fig. 5, *A* and *B*; and 6, *A* and *B*). One or more small cavities were present in almost half the patients (Fig. 7). Large cavities were rare. Following treatment and recovery, cavities may persist as bulla-like shadows.

Miliary dissemination, a rare manifestation of hematogeneous actinomycosis,[12,28] was not observed in our series. Two patients had localized honeycombing in and adjacent to an area of actinomycotic involvement.

TABLE I

SUMMARY OF CHEST ROENTGEN MANIFESTATIONS IN 15 PATIENTS

		No. of Patients
Mass lesion		6
Chronic alveolar infiltrate		6
Pulmonary fibrosis		3
Cavitation		6
Transgression of an interlobar fissure		4
Pleural disease		12
Effusion or empyema	6	
Pleural thickening alone	6	
Esophagopleural and bronchopleural fistula		1
Chest wall involvement		9
Soft-tissue swelling	4	
Periostitis of ribs	4	
Frank rib destruction	2	
Vertebral destruction	1	
Pulmonary osteoarthropathy		1

Bronchograms of 6 patients were available. Three appeared normal; 1 showed slight bronchial narrowing and mild cylindrical bronchiectasis (Fig. 4*B*); another had mild bronchiectasis alone; and 1 showed irregular bronchial obstruction (Fig. 3*B*).

Pleural involvement occurred in most of our patients. This took the form of a small effusion or empyema, or merely pleural thickening. A characteristic stranding of the pleura, like the strings of a parachute, may occasionally occur (Fig. 3*A*; and 7). Mediastinal widening, indicative of direct invasion of the mediastinum, may occur.[2]

Involvement of the thoracic wall is an important roentgen manifestation of actinomycosis. It was seen in over half of our patients and may take the form of soft-tissue swelling (Fig. 6, *A* and *B*; and 8), sinus tracts, bone involvement, and even disfiguration (Case 1). Injection of cutaneous fistulas with contrast medium will usually reveal extension into the lung or pleural sac (Fig. 9, *A* and *B*) and occasionally into the trachea, esophagus, or even the abdomen.[2,24]

Bone changes consist of periosteal proliferation or frank destruction involving one or more ribs.[29] The shoulder girdle or sternum may be similarly affected.[2] The periostitis may have a peculiar wavy configuration (Fig. 1, *A–C*; and 9, *A* and *B*) which is highly suggestive of actinomycosis. One of our patients with rib destruction had both actinomycosis and squamous cell carcinoma in the same region. Which of these caused the bone destruction could not be ascertained. One patient (Case 1) with severe local periostitis of the ribs adjacent to a pulmonary lesion also showed the typical generalized periostitis of pulmonary osteoarthropathy.

Actinomycotic involvement of the spine is usually the result of extension from the mediastinum or retroperitoneum. Erosion of both the body and processes of the affected vertebrae as well as involvement of the adjacent ribs is often seen (Fig. 10).[5,7,20,35] The typical wavy periostitis may be

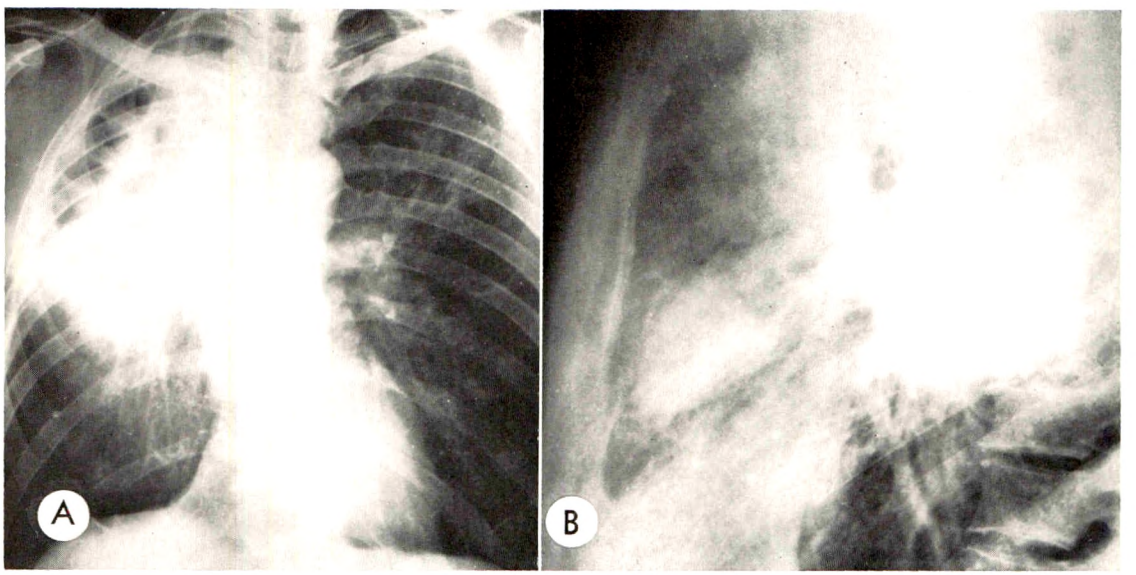


Fig. 5. A 53 year old man with dyspnea, pleuritic pain, and abscess over right upper chest wall. (*A*) Poster o anterior view. There is fibrocavitary infiltrate in the right upper lung field and bulla-like lucencies along the periphery of the upper thorax. An ill-defined infiltrate is present in the left upper lung field. (*B*) The right lateral view demonstrates transgression of the right major fissure and thickening of the retrosternal tissues.

present on the transverse processes as well as the adjacent ribs. Destruction combined with new bone formation produces a mottled effect in the vertebral bodies. Unlike tuberculosis, collapse of the vertebral bodies and intervertebral disk narrowing are not features of the disease.

DISCUSSION

The diagnosis of thoracic actinomycosis

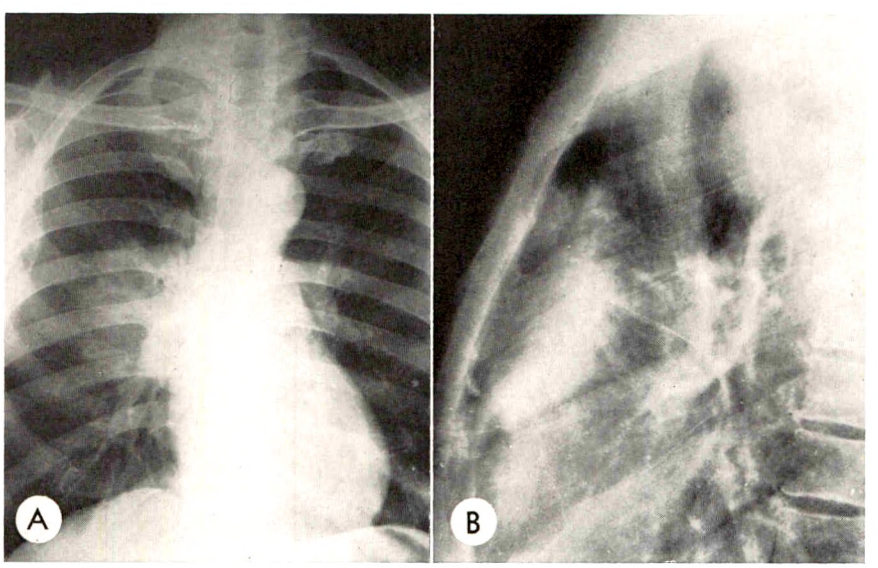


Fig. 6. (*A* and *B*) A 58 year old man with tender, firm swelling over the right sternoclavicular area, chest pain, cough, and weight loss. There is an infiltrate in the right anterior chest, crossing the minor fissure. The density in the right apex in both views represents the soft-tissue mass.

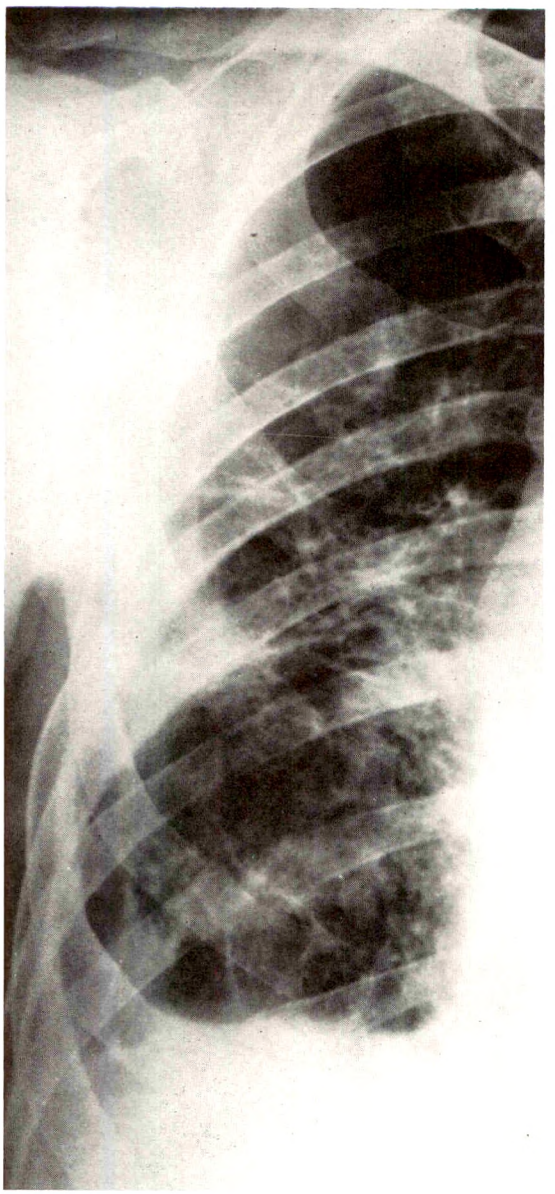


FIG. 7. A 61 year old man with a chronic, draining, granulating lesion on the right chest wall. There is extensive chronic infiltrate and strand-like pleural thickening in the right lower thorax. The periosteal reaction along the lower ribs is poorly shown on this study.

is established in the laboratory. Recovery of *A. israeli* from infected tissue is the only proof of the disease. However, clinical suspicion is important also, and this may come from the roentgen findings. Chest wall involvement—swelling, draining sinus, wavy periostitis, and rib destruction—is highly suggestive. Another important clue is the penetration of an interlobar fissure, seldom encountered roentgenologically except in fungus diseases. The roentgen manifestations are otherwise nonspecific. Nonetheless, any chronic indolent pulmonary infiltrate should alert the radiologist, clinician, and pathologist to the possibility of thoracic actinomycosis.

Chronic infiltrate with cavitation is more common in pulmonary tuberculosis and is often seen with other fungus infections, such as blastomycosis, torulosis, and even histoplasmosis. Carcinoma is also simulated, although, as noted earlier, the bronchogram usually helps to make the distinction.

In 1931, Kirklin and Hefke,[17] upon analyzing 14 cases of thoracic actinomycosis and reviewing the literature up to that time, asserted that the combination of bone changes, thickened pleura, empyema, and

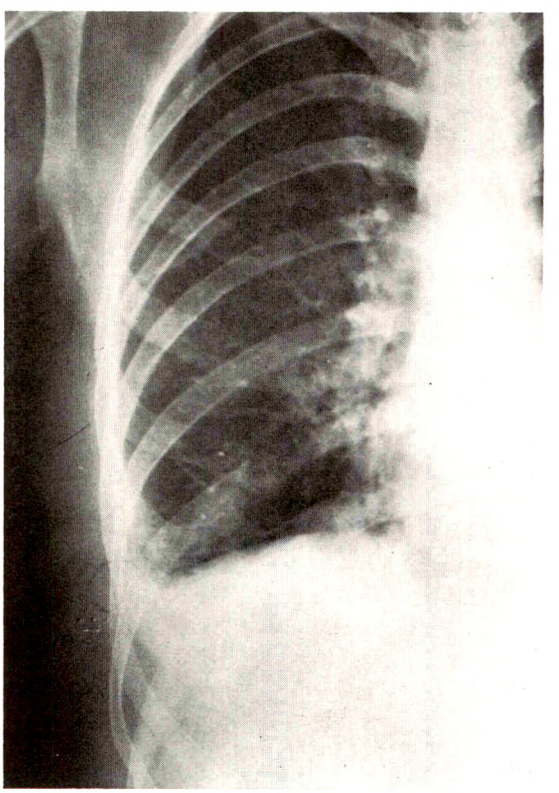


FIG. 8. A 26 year old woman with cough, fever, weight loss, and marked swelling of the soft tissues along the right chest wall. Minimal pleural disease and pulmonary infiltrate are noted.

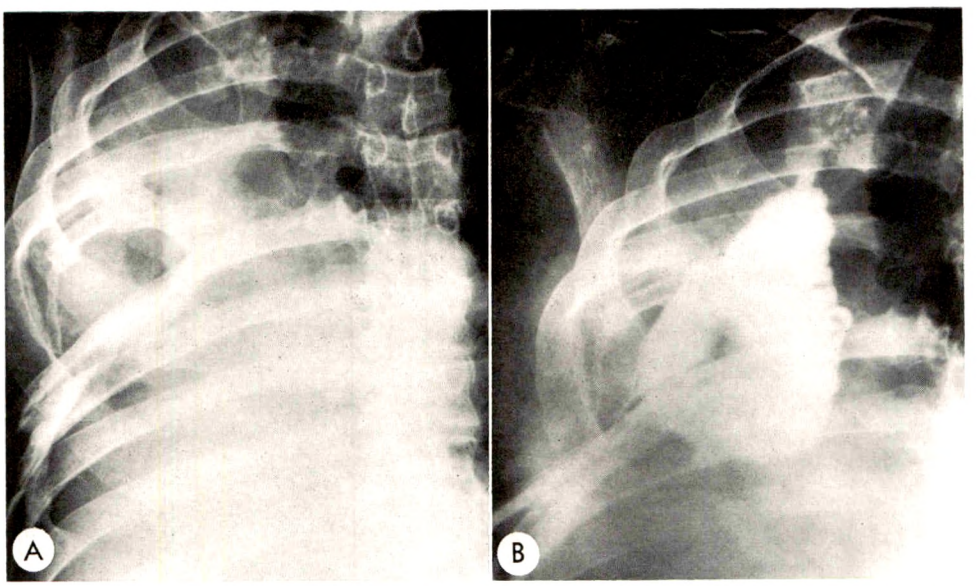


Fig. 9. Large persistent empyema cavity in a middle-aged man who had had a right pneumonectomy 2 years earlier for actinomycosis. (*A*) A Bucky roentgenogram. The 6th rib has been resected. Note the wavy periostitis involving the 5th rib. (*B*) The empyema cavity has been injected with lipiodol.

pulmonary involvement is more indicative of actinomycosis than of any other disease. Bates and Cruickshank[2] considered periosteal reaction of several adjacent ribs in the absence of empyema the only pathognomonic roentgenologic sign of thoracic actinomycosis. In general, we concur with these views. In addition, we consider a pulmonary lesion that extends across an interlobar septum or through the pleura to involve the chest wall to be strongly suggestive of actinomycosis, even in the absence of bone involvement.

SUMMARY

Fifteen cases of thoracic actinomycosis have been reviewed at the University of Cincinnati Medical Center and their roentgenograms analyzed. Although the roentgen manifestations of the disease are for the most part nonspecific, the following features should suggest the diagnosis:

1. Extension of a pulmonary lesion through the thoracic wall.
2. Wavy periostitis or frank destruction of ribs associated with contiguous chronic pulmonary involvement.
3. Penetration of a pulmonary lesion through an interlobar fissure.
4. A characteristic pattern of vertebral destruction.



Fig. 10. A 15 year old boy with actinomycotic involvement of the thoracolumbar spine. Note the destructive lesion in L 2 (arrow) and mottling in the bodies of T 11 and 12. The vertebral bodies and disks have maintained their heights.

Any chronic pulmonary infiltrate, especially if accompanied by cavitation or pleural involvement, is suspect.

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ROENTGENOGRAPHIC MEASUREMENTS OF DIAPHRAGMATIC MOTION-RESPIRATORY COMPLIANCE*

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LUNG and thoracic cage expansion reflect total respiratory effort and compliance, and may be indirectly demonstrated and measured by the extent of diaphragmatic motility. Lung compliance is directly related to lung volume,[4] and any appreciable increase or decrease in lung volume will be reflected by the extent of diaphragmatic motion.

The pulmonary and thoracic compliance can be accurately measured by pulmonary function studies. The respiratory compliance values vary in the normal and abnormal patient with age, sex, height and weight, rate of respiration, posture and position and by the degree of and type of disease or abnormality. Because of such a high degree of individual variability in health and disease, average pathophysiologic measurements and statistics are utilized clinically, and used only as a guide in the clinical management of the patient with pulmonary or thoracic cage dysfunction or disease. Pulmonary function studies to determine pulmonary, thoracic and total respiratory compliance are routinely performed in well equipped pulmonary function laboratories. Measurements of diaphragmatic excursion, in this area, are little used and are of unknown value.

A search of the medical literature produces a great deal of material on the anatomy and physiology of the diaphragm, and on its relation with pathophysiologic conditions, as related to the chest and abdomen. In only a few papers is there mention of the use of measurements of diaphragmatic motion in estimating pulmonary or thoracic cage compliance or extent of pulmonary or thoracic cage disease or dysfunction.

In the present series of 200 cases, diaphragmatic excursion was measured on roentgenograms taken during unforced and forced expiration in an attempt at estimating pulmonary and thoracic cage compliance in patients with pulmonary or thoracic cage pathology.

MATERIAL AND METHOD

This group of patients comprises 200 veterans, most of whom had routine chest roentgenograms taken as part of a follow-up examination in evaluating the progress of known lung or thoracic cage pathology. A small number of the patients had chest roentgenograms taken as part of an initial physical work-up. All of the patients were ambulatory, cooperative and followed precise breathing instructions. The roentgenographic examinations were all performed in the Department of Radiology of the Veterans Administration Clinic.

The chest roentgenograms were easy to take and were all made on 14×17 inch x-ray film in the upright posteroanterior position, at a 6 foot distance on deep inspiration, and on forced and unforced expiration. For this study, each patient had a minimum of 3 chest roentgenograms taken which were used for measurement of diaphragmatic motion. Technically, the patient was told to take a deep breath and to hold his breath, at this time the first chest roentgenogram was taken. Then he was told to take another deep breath and "let it all out;" at this point, the second chest roentgenogram was made. Lastly, he was told to take another deep breath, "let it all out, and now force out some more;" at this point the third chest roentgenogram was taken.

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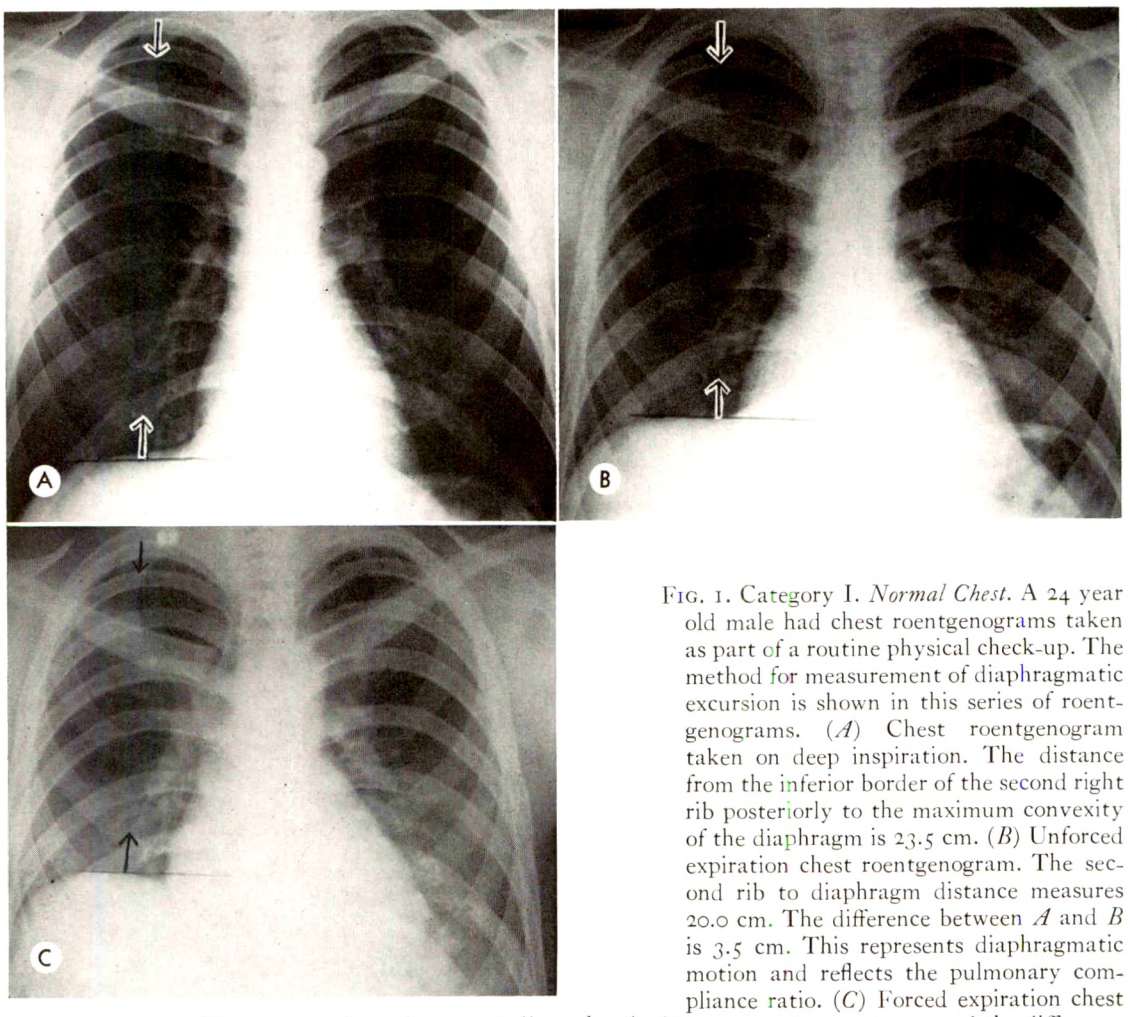


FIG. 1. Category I. *Normal Chest.* A 24 year old male had chest roentgenograms taken as part of a routine physical check-up. The method for measurement of diaphragmatic excursion is shown in this series of roentgenograms. (*A*) Chest roentgenogram taken on deep inspiration. The distance from the inferior border of the second right rib posteriorly to the maximum convexity of the diaphragm is 23.5 cm. (*B*) Unforced expiration chest roentgenogram. The second rib to diaphragm distance measures 20.0 cm. The difference between *A* and *B* is 3.5 cm. This represents diaphragmatic motion and reflects the pulmonary compliance ratio. (*C*) Forced expiration chest roentgenogram. The distance from the second rib to the diaphragm measures 17.5 cm., and the difference between *A* and *C* is 6.0 cm., representing diaphragmatic maximum excursion and reflecting the total respiratory compliance.

The rates of 3.5 cm. and 6.0 cm. in *B* and *C* correspond to normal diaphragmatic motion on normal expiration and with forced expiration.

Diaphragmatic excursion is determined by comparison of 2 chest roentgenograms. The distance from the lower border of the posterior end of the second rib to the highest point of convexity of the diaphragm is measured in centimeters. The differences, in centimeters, between the deep inspiration chest roentgenogram and the unforced expiration chest roentgenogram measures diaphragmatic motion and reflects pulmonary compliance. The difference, in centimeters, between the deep inspiration and forced expiration reflects the total respiratory compliance. Measurements of diaphragmatic excursion on chest roentgenograms taken on deep inspiration, unforced and forced expiration are illustrated in Figure 1, *A-C*.

In this group of 200 patients, the diagnosis of pulmonary or thoracic cage disease had been previously made on a clinical basis from previous hospitalizations, and, in most cases, following pulmonary function studies. Except for a few female patients, this group comprised male veterans of World Wars I and II, the Korean and the

Table I

NUMBER OF CASES BY CATEGORY AND AGE

Category	Age (years)			Total
	18–30	31–50	51–77	
I. Normal chest	3	7	3	13
II. Obstructive lung disease				
a. Asthma	8	13	15	36
b. Bronchitis	5	12	14	31
c. Emphysema	1	6	20	27
d. Fibrosis		1	1	2
III. Restrictive lung disease	7	15	7	29
IV. Muscular and thoracic cage disease	15	13	8	36
V. Miscellaneous	6	13	7	26
Total	45	80	75	200

Vietnam conflicts. The ages ranged from 18–77 years; there were 45 in the 18–30 year age group, 80 in the 31–50 year age group and 75 from 51–77 years of age (Table 1).

Because of the variety of pathology in this series of cases, it was useful to categorize these patients into 5 groups on the basis of their clinical diagnosis.

CATEGORIES

I. *Normal Chest.* Chest roentgenograms were taken as part of a complete clinical work-up. Included in this group are patients with obesity, and patients with gastrointestinal symptoms.

Table II

AVERAGE DIAPHRAGMATIC EXCURSION BY AGE

Group	Age 18–30 Years			Age 31–50 Years			Age 51–77 Years			Total for all Ages		
	No. of Cases	Average Unforced Expiration (cm.)	Average Forced Expiration (cm.)	No. of Cases	Average Unforced Expiration (cm.)	Average Forced Expiration (cm.)	No. of Cases	Average Unforced Expiration (cm.)	Average Forced Expiration (cm.)	Average Unforced Expiration (cm.)	Average Forced Expiration (cm.)	No. of Cases
I. Normal chest	3	4.0	6.1	7	1.6	4.2	3	2.7	3.7	2.3	4.5	13
II. Obstructive lung disease												
a. Asthma	8	3.3	6.0	13	2.9	4.3	15	2.4	4.8	2.8	4.7	36
b. Bronchitis	5	2.7	5.5	12	2.6	5.1	14	2.5	5.3	2.5	5.0	31
c. Emphysema	1	2.5	3.0	6	1.0	3.1	20	1.8	3.3	1.6	3.3	27
d. Fibrosis				1	1.5	2.5	1	.2	4.5	1.7	3.5	2
III. Restrictive lung disease	7	2.7	3.6	15	1.8	4.0	7	1.7	3.1	1.9	3.7	29
IV. Muscular and thoracic cage disease	15	2.5	5.1	13	2.8	5.0	8	2.5	5.2	2.6	5.0	36
V. Miscellaneous	6	2.2	4.1	13	3.0	5.0	7	2.3	4.8	2.5	4.7	26
Total	45			80			75					200

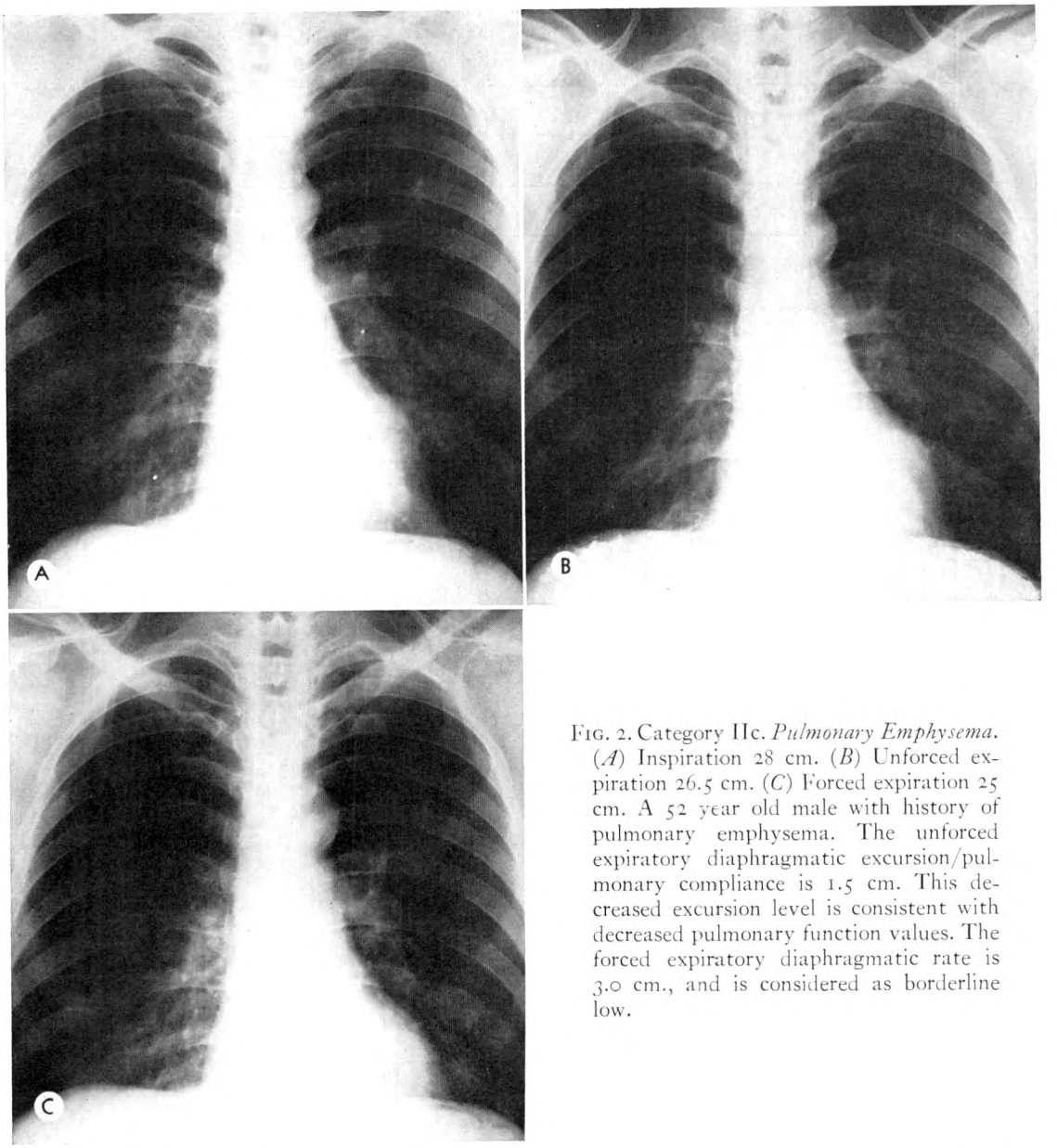


Fig. 2. Category IIc. *Pulmonary Emphysema.* (*A*) Inspiration 28 cm. (*B*) Unforced expiration 26.5 cm. (*C*) Forced expiration 25 cm. A 52 year old male with history of pulmonary emphysema. The unforced expiratory diaphragmatic excursion/pulmonary compliance is 1.5 cm. This decreased excursion level is consistent with decreased pulmonary function values. The forced expiratory diaphragmatic rate is 3.0 cm., and is considered as borderline low.

II. *Obstructive Lung Disease.* Patients with a history of pulmonary emphysema, chronic bronchitis, bronchial asthma and interstitial pulmonary fibrosis.

III. *Restrictive Lung Disease.* Disease conditions secondary to inflammatory lung disease, pulmonary tuberculosis, mycotic disease, lung and mediastinal tumors and pleural fibrosis.

IV. *Muscular and Thoracic Cage Pathology.* This group includes patients with chest or respiratory problems secondary to trauma (auto accidents, gunshot and stab wounds) and postsurgical chest.

V. *Miscellaneous.* Patients with a history of chest pain, cough, dyspnea, sinusitis, heart disease, allergy, and hypertension.

RESULTS

The average diaphragmatic excursion rates, in centimeters, are tabulated for all

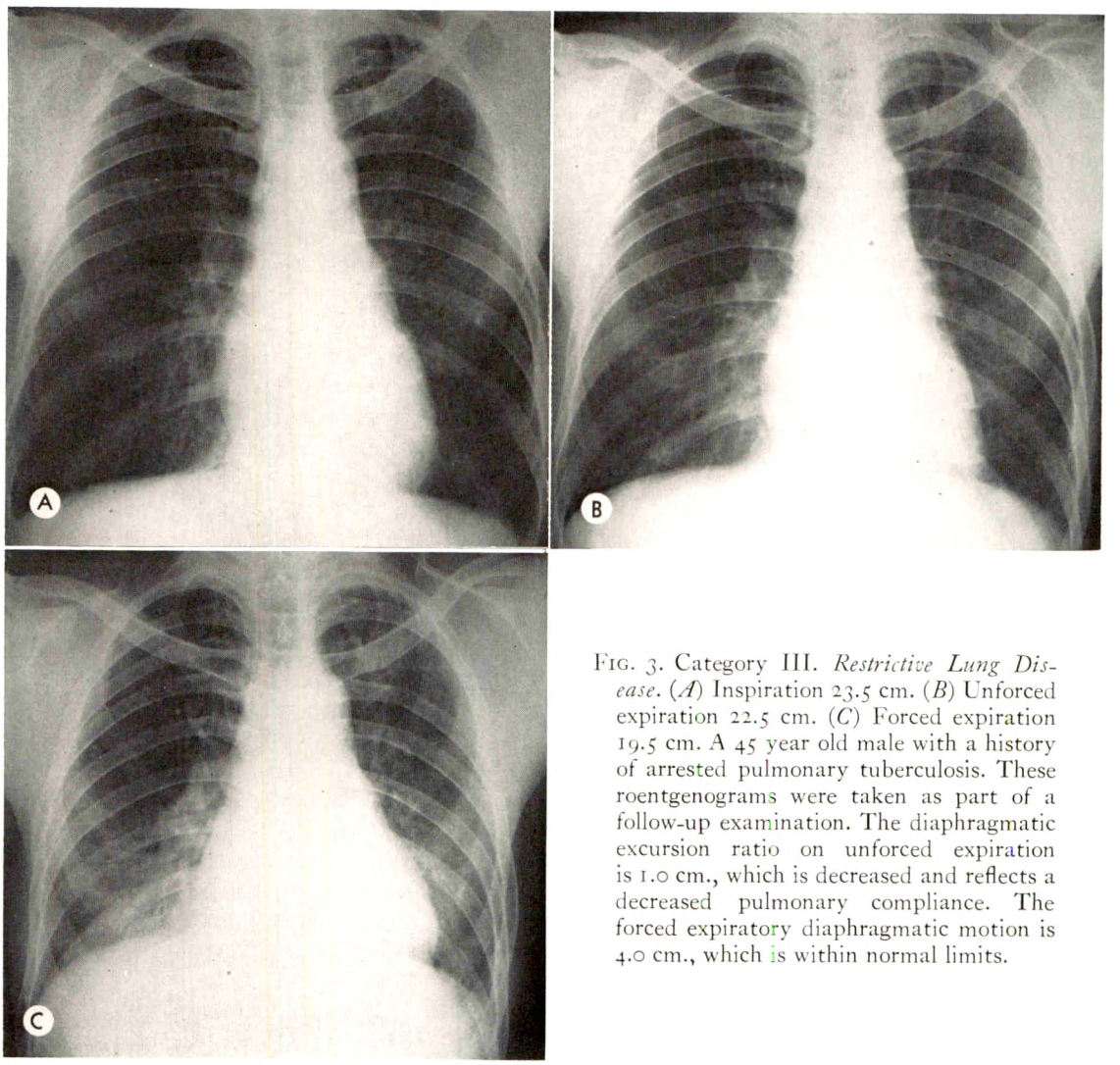


Fig. 3. Category III. *Restrictive Lung Disease.* (*A*) Inspiration 23.5 cm. (*B*) Unforced expiration 22.5 cm. (*C*) Forced expiration 19.5 cm. A 45 year old male with a history of arrested pulmonary tuberculosis. These roentgenograms were taken as part of a follow-up examination. The diaphragmatic excursion ratio on unforced expiration is 1.0 cm., which is decreased and reflects a decreased pulmonary compliance. The forced expiratory diaphragmatic motion is 4.0 cm., which is within normal limits.

age groups and for the separate categories (Table II).

In the normal patient, the average unforced diaphragmatic excursion was 2.3 cm. and the forced, average diaphragmatic excursion was 4.5 cm. In those patients with obstructive lung disease, the average unforced diaphragmatic excursion was lowest; for those patients with pulmonary emphysema and interstitial pulmonary fibrosis, the average diaphragmatic excursion rates were 1.6 cm. and 1.7 cm., respectively. In those patients with restrictive lung disease, the unforced diaphragmatic excursion was low and averaged 1.9 cm. The diaphragmatic excursion ratios for all other groups, in forced and unforced expiration, appear to be within normal physiologic ranges.

Representative chest roentgenograms for each of the 5 categories taken on deep inspiration, unforced and forced expiration are shown in Figures 1 to 5.

DISCUSSION

Normal respiration is mainly effected by the muscular action of the diaphragm which accounts for 60–75 per cent of the total air breathed. Inspiration is the result of contraction of the diaphragm, external

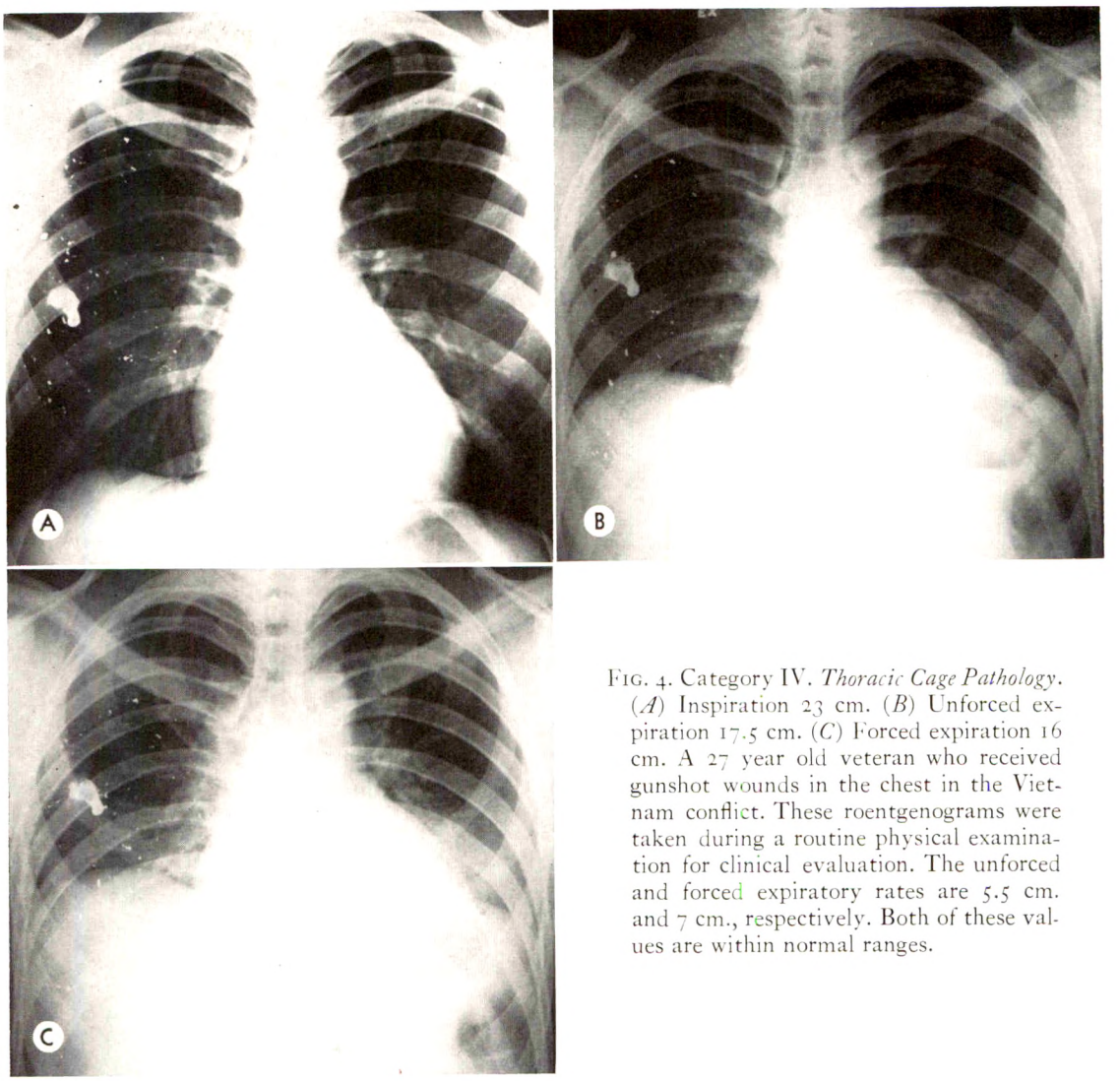


Fig. 4. Category IV. *Thoracic Cage Pathology.* (*A*) Inspiration 23 cm. (*B*) Unforced expiration 17.5 cm. (*C*) Forced expiration 16 cm. A 27 year old veteran who received gunshot wounds in the chest in the Vietnam conflict. These roentgenograms were taken during a routine physical examination for clinical evaluation. The unforced and forced expiratory rates are 5.5 cm. and 7 cm., respectively. Both of these values are within normal ranges.

intercostal muscles, serratus superior and levator costarum muscles.[2] Expiration is effected by the intercostal muscles and contraction of the abdominal wall muscles,[2] causing the diaphragm to move a distance equal to 2 interspaces and the diameter of 1 rib—about 5 cm.[5] Lung elasticity alone will produce about 1.5 cm. diaphragmatic movement. Forced expiration results from the contraction of the internal intercostal muscles, pectoralis major and minor, abdominal and latissimus dorsi muscles[2] and produces an average excursion of the right hemidiaphragm of 6.6 cm. and of the left hemidiaphragm up to 7.7 cm.[5]

The participation of the diaphragm in respiration cannot be overemphasized. Clinically, diaphragmatic motion is best observed under the fluoroscope and on cineroentgenography. Diaphragmatic excursion can be measured on 1 double exposed chest roentgenogram taken during inspiration and expiration, or when chest roentgenograms are taken separately during inspiratory and expiratory phases of respiration, or by means of roentgenokymography.

Clinically, the forced expiratory chest roentgenogram is taken to demonstrate diaphragmatic motility and to record decreased diaphragmatic excursion in patients with: phrenic nerve paralysis, pleural

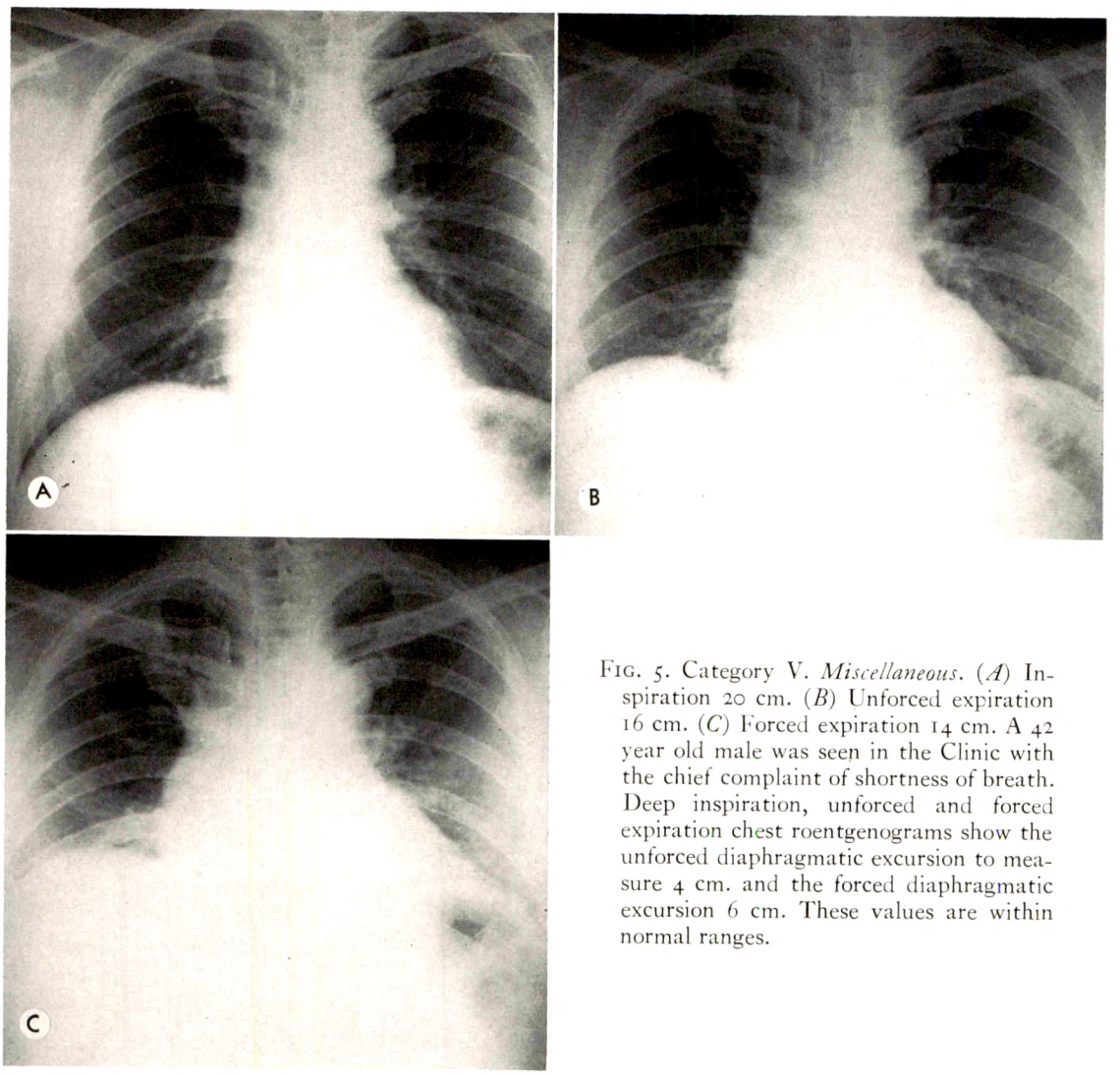


Fig. 5. Category V. *Miscellaneous*. (*A*) Inspiration 20 cm. (*B*) Unforced expiration 16 cm. (*C*) Forced expiration 14 cm. A 42 year old male was seen in the Clinic with the chief complaint of shortness of breath. Deep inspiration, unforced and forced expiration chest roentgenograms show the unforced diaphragmatic excursion to measure 4 cm. and the forced diaphragmatic excursion 6 cm. These values are within normal ranges.

fibrosis, atelectasis, pneumonia, bronchial obstruction, pulmonary emphysema, subdiaphragmatic abscess, pulmonary tumor, myasthenia gravis and in patients with ascites, obesity, kyphoscoliosis and pregnancy.

Measurement of the extent of diaphragmatic motion on the unforced expiration chest roentgenogram records lung compliance. Lung compliance is directly related to lung volume;[4] hence, diaphragmatic motion. Pulmonary function tests show a reduced pulmonary compliance in severe chronic obstructive pulmonary emphysema, with diffuse pulmonary and interstitial pulmonary fibrosis, chronic bronchitis, bronchial asthma, pulmonary atelectasis, pneumonia, pulmonary congestion and edema and with pleural fibrosis.[1] In this group, therefore, the diaphragmatic excursion on unforced expiration should be decreased.

Recently, Milne and Bass[6] studied the range of diaphragmatic excursion in 900 patients, recorded the dynamic lung compliance on unforced expiration roentgenograms and found good correlation, in these results, with pulmonary function studies. In 130 of these patients the functional pulmonary compliance tests compared with the diaphragmatic excursion ratios. In the group with pulmonary emphysema the

diaphragmatic excursions ranged from 0.3 to 2 cm., and the entire group with chronic obstructive pulmonary disease had an average excursion of 1.25 cm. In this series, the controls had a diaphragmatic excursion between 2.5 cm. and 4.5 cm.[6]

The unforced (pulmonary compliance) diaphragmatic excursion ranges or rates, in our normal patients, in those with chest trauma, and in our miscellaneous group, correspond well with Milne and Bass' control group. The average diaphragmatic excursion rates in our patients with pulmonary emphysema, pulmonary fibrosis and for the group with chronic restrictive lung disease was from 1.6 to 1.9 cm. These results also correlate well with Milne and Bass' statistics for a similar group. From the accumulated data, it would appear that measurements of the extent of diaphragmatic motion, from unforced expiratory chest roentgenograms, might give us some insight on the status of pulmonary compliance.

For each category in the group of cases reported here, the total series of cases is small and would not be considered of definite statistical value. However, a distinct pattern or degree of diaphragmatic motion appears to be present for each medical group. A more detailed study of many more patients with a variety of single or multiple diseases, in different ages and with various degrees of illness, should be evaluated roentgenographically and correlated with pulmonary function studies. This work, I believe, will produce more meaningful and clinically useful statistics, which might be applicable to and aid in the diagnosis and treatment of pulmonary diseases and thoracic cage pathology.

CONCLUSIONS AND SUMMARY

The extent and degree of mobility of the diaphragm was measured on deep inspiration and on forced and unforced expiration chest roentgenograms in 200 patients. Clinically, the forced expiratory postero-anterior chest roentgenogram is taken to evaluate the extent of diaphragmatic motility, as a means of establishing pulmonary, pleural, intra-abdominal or thoracic cage disease or dysfunction. The degree of diaphragmatic motion during forced maximal expiration reflects total vital capacity, and the extent of diaphragmatic excursion during normal expiration measures pulmonary compliance. The average extent of diaphragmatic motion corresponds to indices of pulmonary compliance and total vital capacity, and was calculated from measurements made on chest roentgenograms. Previous studies correlating pulmonary function results with indices of diaphragmatic excursions, in the same cases, suggested that this knowledge could be of supplemental value and might be useful in the diagnosis and therapy of thoracic cage and pulmonary diseases.

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PHLEBOGRAPHY IN THE PREVENTION OF RECURRENT PULMONARY EMBOLISM—TECHNIQUES AND VALUE*

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PULMONARY embolism kills at least 2,500 people in Great Britain every year (Registrar General's Report 1967). In the United States of America the death rate is thought to be 47,000 each year and to contribute to 150,000 deaths in all.[29]

A proportion of these deaths follow a second or third embolus. Barker and Priestley[2] in a review of the natural history of 1,665 cases of postoperative thromboembolism found that 30 per cent of patients who survived 1 pulmonary embolus had a second, and that this was fatal in 19 per cent.

Thus it is clear that once a patient has had 1 embolus, urgent steps must be taken to prevent a recurrence.

Intravenous heparin will reduce the mortality rate and also the incidence of second emboli[1,4,8,10,11,13,23,24,27,30,32,35,37] but its effectiveness is uncertain. For example, although Barritt and Jordan[4] had no fatal recurrences, Ochsner and co-workers[35] reported a second fatal embolus rate of 11.7 per cent.

The alternative to anticoagulants in preventing recurrence is vein ligation or plication,[1,5,8,9,13–15,18,19,23,24,35,36,38] but they do have significant recurrence rates. Vena cava ligation is the most reliable surgical measure although it is associated with definite postphlebitic complications.[16] We have found that a combination of anticoagulant therapy and surgery based on the results of phlebography is the most effective way of preventing recurrent pulmonary embolism with minimum disability to the patient.

In this report we describe the techniques used for phlebography of the leg and pelvic veins and indicate how the results have been used in the prevention of recurrent pulmonary embolism.

The results of our treatment with anticoagulants supplemented by surgery are compared with a group of patients treated with anticoagulants alone who were not investigated by phlebography.

MATERIAL

Fifty patients (trial group) who on clinical, electrocardiographic and chest roentgenographic grounds were considered to have had a pulmonary embolism were subjected to phlebography. The patients were all treated with anticoagulants with additional surgery as indicated by phlebography.

This group was compared, by random selection, with 50 patients (control group) whose case notes gave a definite clinical diagnosis of pulmonary embolism, none of whom had had phlebography and who had been treated with anticoagulants alone.

Both groups were treated with intravenous heparin followed by warfarin in similar dosage and controlled by the same coagulation laboratory. The warfarin was continued, on an average, for 3 months.

METHODS OF INVESTIGATION

PHLEBOGRAPHY OF LEGS

A tilting table equipped with an image intensifier, a television, and an automatic exposure device is used. The table is tilted 40°, foot downwards. A vein on the dorsum of the foot, preferably towards the lateral side, is selected and a 20 gauge

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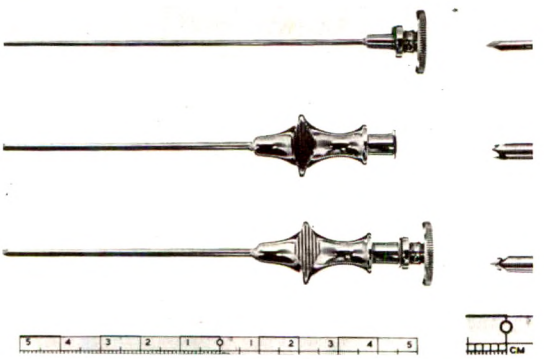


Fig. 1. The authors' intraosseous cannula. On the right the 3 faceted drill tip is shown in close-up. (Reproduced from *Brit. J. Radiol.*)

needle bent to about 30° is introduced into the vein in a distal (upstream) direction.

If percutaneous puncture is impossible a small cutdown is made. This was only necessary in 2 (4 per cent) of the 50 patients.

A single tourniquet is applied just above the ankle; a self-fastening type is useful as it permits small changes in tension.

The needle is connected by a plastic tube to a syringe containing the contrast medium meglumine iothalamate 60 per cent.

The foot is internally rotated to separate the images of the tibia and fibula and a hand injection is begun under television control. If necessary, the tension of the tourniquet is adjusted so that the 3 main groups of deep veins of the calf are seen to fill. The tourniquet is not applied tightly enough to completely occlude the superficial veins.

The flow of the contrast medium is followed on the television screen and 14×14 inch (35.5×35.5 cm.) films split into 3 are exposed at intervals so that the whole venous system from the ankle to the inferior vena cava is included. To show the iliac veins and inferior vena cava the tourniquet is removed so that the contrast material trapped below it flows into the veins, improving opacification.

The upper part of the profunda femoris vein, as far as a competent valve, is demonstrated by having the patient perform the Valsalva maneuver when the common femoral vein is filled with contrast medium.

After each injection the venous system is flushed immediately with normal saline to clear the veins of contrast medium; this is checked with the television.

Both legs are examined in all patients to exclude subclinical thrombosis.

The amount of contrast material usually required is 50 ml. for each side. After the first series, the roentgenograms are examined. Critical and doubtful areas are reexamined so that in some instances as much as 300 ml. may be needed.

If the iliac veins and inferior vena cava have not been sufficiently well demonstrated to exclude a thrombus, or if the full extent of a thrombus has not been shown, pelvic phlebography is required.

Percutaneous Femoral Pelvic Phlebography

With the patient in the supine position using local anesthesia, 2 cannulae are introduced percutaneously into the femoral veins and threaded a short distance to prevent extravasation of contrast medium.

Simultaneous hand injections of 50 ml. of sodium iothalamate 70 per cent are performed. Five 14×14 inch (35.5×35.5 cm.) films are exposed at intervals of 2 seconds—the roentgenographic field including the lower part of the inferior vena cava. A Valsalva maneuver is performed half-way through the injection to show the internal iliac veins as far as a competent valve.

Intraosseous Pelvic Phlebography

Intraosseous phlebography is indicated in those patients in whom a femoral vein was shown to be occluded, on the peripheral phlebogram, or who had swelling of the whole limb indicating high obstruction, or in whom percutaneous femoral venepuncture had failed. This examination, being painful, requires a general anesthetic.

With the patient in the prone position 2 intraosseous cannulae (Fig. 1)[29] are introduced into the greater femoral trochanters under television control.[28] The cannulae are

connected to 2 separate pressure pumps and bilateral simultaneous injections of 50 ml. of sodium iothalamate 70 per cent are made. Ten 14×14 inch (35.5×35.5 cm.) films are exposed, at 1 per second, of the pelvis to include the lower inferior vena cava. Filming is started about the middle of the injection. After the injection the pelvic veins are cleared of contrast medium with normal saline.

In some instances a combined technique of femoral pelvic phlebography on one side with pertrochanteric phlebography on the other is used.[21,28] For convenience this examination is carried out in the supine position.

INTERPRETATION OF PHLEBOGRAMS

The diagnosis of recent thrombus depends on demonstrating filling defects within an opacified vein. The phlebograms should show the full extent of thrombus (Fig. 2). Nonfilling of a vein can only be indicative of thrombosis if the vein above and below and the collaterals are filled with contrast medium (Fig. 3). Fresh thrombus appears as a radiolucent defect within the vein with a thin white line representing contrast medium between the thrombus and vein wall on each side (Fig. 4). As the thrombus ages the roentgenographic appearances change. The white line is lost in

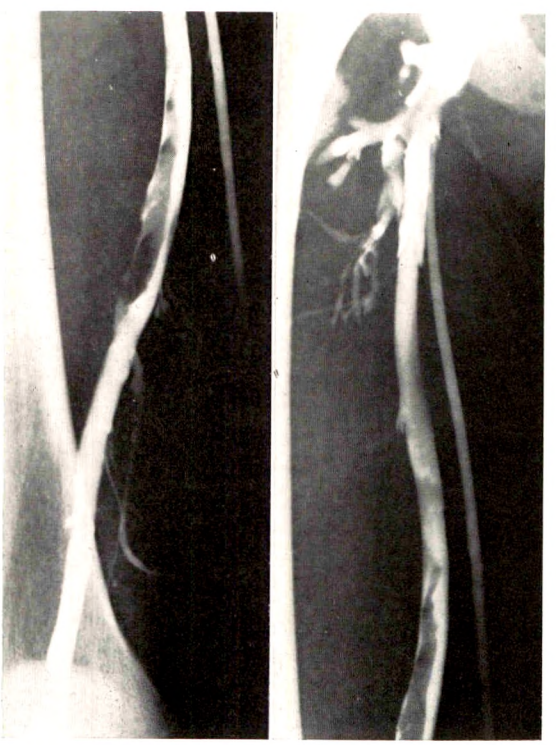


Fig. 2. Recent thrombus in femoral vein. Note that the complete extent of the thrombus has been shown.

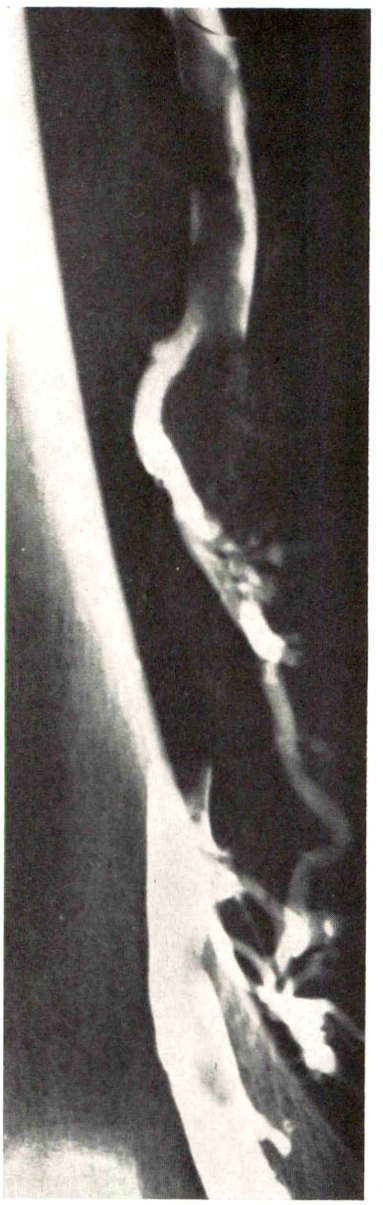


Fig. 3. Occlusion of a segment of the femoral vein. Note that the vein above and below the occlusion and a collateral are filled with contrast medium.



FIG. 4. Recent thrombus in femoral vein. Note the thin white line representing contrast medium around the thrombus.

parts or over a complete segment of the vein, indicating adherence (Fig. 5) or occlusion (Fig. 3). Later this white line becomes thicker and more sharply defined (Fig. 6) as the clot retracts. Finally the vein may recanalize, leaving an irregular lumen often with several channels (Fig. 7, *A* and *B*).

TREATMENT

Peripheral phlebograms gave an adequate demonstration of the major veins from ankle to inferior vena cava in 37 (7 per cent) of the patients in the trial group; percutaneous femoral pelvic phlebography was needed in 9 (18 per cent) and intraosseous pertrochanteric phlebography in 4 (8 per cent).

Thrombus was found in the calf veins in 17 patients; in the common and superficial femoral veins in 15; in the common femoral and iliac veins in 6; and in the inferior vena cava in 1. The thrombus was recent and nonadherent and judged to be a potentially fatal embolus in 19 patients.

The treatment used in our 50 patients has been described in detail elsewhere[5,7] and is summarized in Figure 8.

RESULTS

The results of this treatment have been published in detail elsewhere.[7] In the 50 patients there were no deaths from recurrent emboli. Three patients in whom anticoagulants had to be stopped because of complications had further minor emboli, giving a recurrence rate of 6 per cent.

In the 50 control patients (treated with anticoagulants and who had no phlebography) 13 (26 per cent) had a second embolus while on anticoagulants and 7 (14 per cent) died, giving a death rate of 14 per cent. Another 7 had emboli after stopping anticoagulants making a total recurrence rate of 40 per cent.

Late complications such as aching of legs, swelling and varicose veins were almost identical—57 per cent in the trial group and 50 per cent in the control group.

DISCUSSION

Phlebography is the most reliable method available at the present time for the demonstration of venous thrombosis and its extent.

I^{125} fibrinogen[33] will indicate the presence

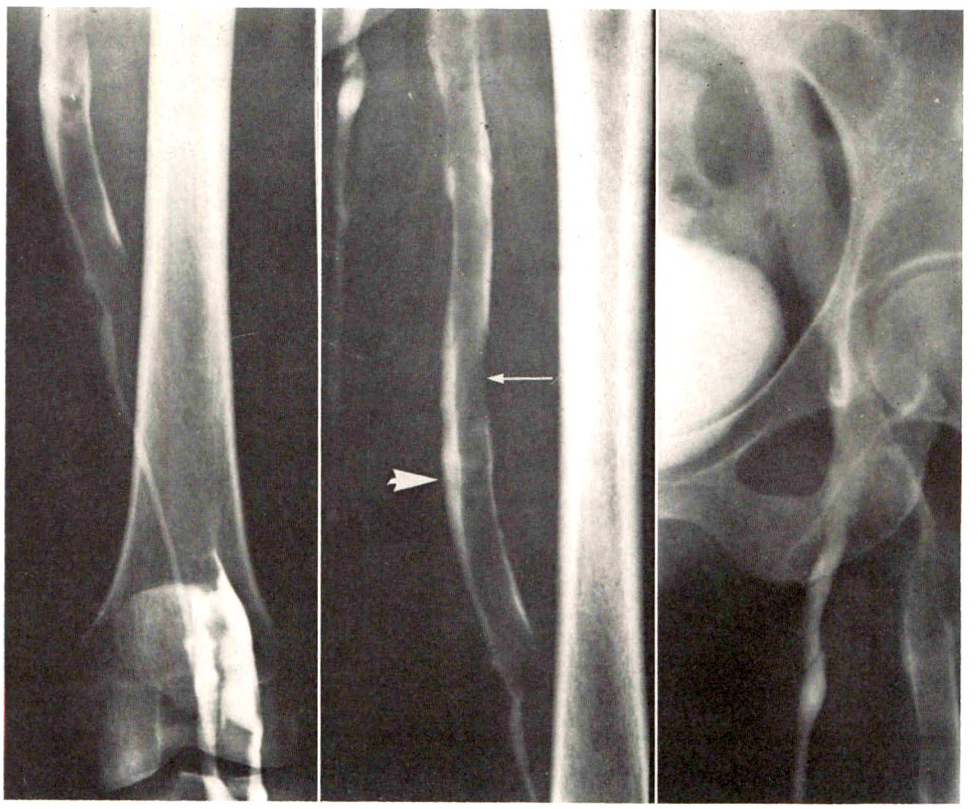


Fig. 5. Recent thrombus in femoral vein showing adherence (←) and aging (→). Note that the femoral and external iliac veins are shown clear of thrombus by filling through the long saphenous vein. (Reproduced from the *Brit. M. J.*[5])

of a thrombus but not its full extent and has the further major disadvantage of being valueless in the iliac veins from which most fatal emboli arise.[31] The ultrasonic Doppler technique[20] indicates an occlusion of the deep venous system but not its cause or extent. It can be useful as an indication of the site of obstruction so that the appropriate phlebography (peripheral or pelvic) can be carried out.

Both these methods are, therefore, screening tests and need to be supplemented by phlebography.

As we use television screening and employ only posteroanterior projections, bilateral peripheral phlebography with our technique can be performed in about half an hour. The phlebograms obtained in a single projection have proved adequate for the treatment of our patients. Percutaneous femoral pelvic phlebography can also be performed quickly and under local anesthesia so that only 4 (8 per cent) of our patients required intraosseous pelvic phlebography with the additional hazard of general anesthesia.

Using the newer contrast media no pain or discomfort was experienced by the patients, and the only complications were minor wound infections in the 2 patients requiring cutdowns.

No thrombosis caused by phlebography was encountered, probably due to the flushing out of the veins of the contrast medium following the examination. No patient had a further pulmonary embolus immediately following phlebography and we feel that this potential danger has been exaggerated.

We agreed that a filling of the deep venous system by gravitation of contrast medium and fewer artifacts due to failure

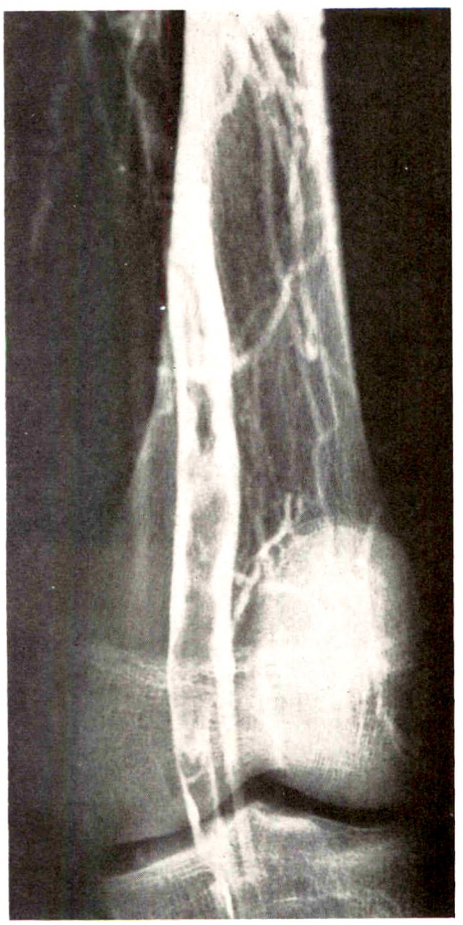


Fig. 6. Thrombus in popliteal vein and lower femoral vein. The thrombus is retracted with a more clearly defined margin.

of mixing occur if the examination is carried out in the upright position.[33] But many of our patients with pulmonary emboli would not tolerate this position and our compromise of tilting the table downwards 40° is satisfactory. If the contrast medium is injected towards the toes, the deep venous system will always fill to some extent if it is patent.[5–7] This filling is enhanced by the single ankle tourniquet. We select a vein towards the lateral side of the foot in order to avoid direct communication with the long saphenous vein which would divert too much contrast medium into the superficial system. It is not part of our technique to totally occlude the superficial system; pulmonary emboli can arise from superficial veins,[24] but, more importantly in an obstructed femoral vein, filling of the vein above from the long saphenous vein (Fig. 5) may make additional pelvic phlebography unnecessary.

Both legs must be examined in all patients. Nineteen patients (38 per cent), who had no clinical signs, in this series had thrombus demonstrated by phlebography.

For intraosseous pertrochanteric pelvic phlebography we have devised[29] an improved intraosseous cannula (Fig. 1) which has a wider bore, is stronger and has 3 cutting surfaces for easier introduction. Roentgenograms are taken in the prone position,[21] as the contrast medium, being heavier than blood, produces better filling of the external and common iliac veins in this position. Two separate pumps must be used to inject the contrast medium in intraosseous phlebography, as a 'Y' connector and single pump is not satisfactory since the contrast medium tends to go into the side presenting the least resistance.

False positive diagnosis of thrombus has not proved a problem. Valves can easily be identified as sharp bicuspid defects; streaming and failure of mixing of contrast medium with the blood may mimic thrombus on a single phlebogram but can be excluded if the doubtful area is re-examined.[5,17]

The frequency of false negatives cannot be established with certainty. Cotton and Clark[12] have shown in their corrosion cast studies the vast network of soleal sinusoids which is impossible to completely fill by phlebography. Studied carefully, a tell-tale small thrombus can usually be seen at one or the other end of an unfilled segment of vein in the calf, indicating that the vein is occluded rather than not filled.

In the prevention of recurrent pulmonary emboli, if no thrombus is demonstrated in the calf veins, it seems safe to assume that either the veins are clear or that the thrombus is adherent and therefore unlikely to cause a serious embolus. This impression has been confirmed by radioactive fibrinogen studies in some patients and by subsequent clinical events in others.

Above the popliteal vein there is no problem, for if the deep veins of the calf fill,

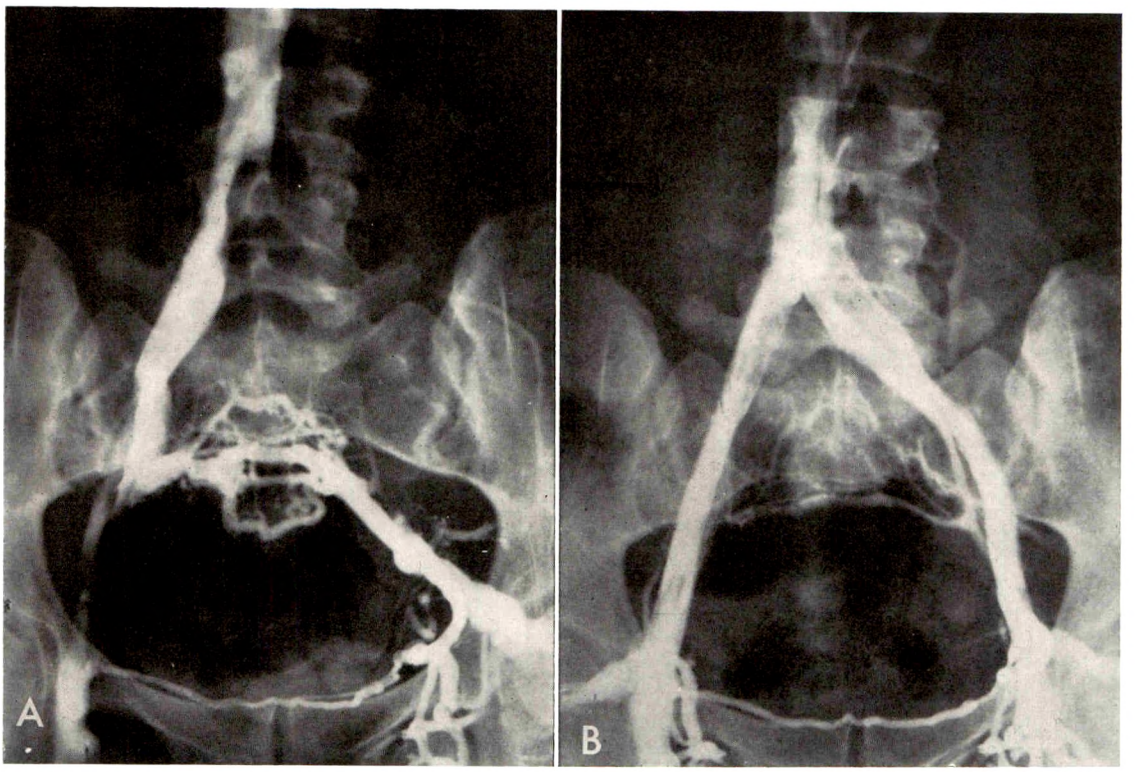


Fig. 7. (*A*) Total occlusion of left external and common iliac veins. (*B*) Repeat phlebogram 1 year later showing recanalization of the veins. Note irregularity of lumen. (Reproduced from *Brit. M. J.*[5])

nonfilling of the more proximal veins indicates occlusion. Potentially fatal emboli usually arise in the iliofemoral segments[5,31]—segments which can easily be demonstrated by phlebography.

Our treatment based on phlebography, has reduced the number of recurrent emboli. We believe that this is a valid conclusion, since the anticoagulant therapy was the same in both the trial and control groups and the oral anticoagulants were controlled by the same laboratory. The initial dose of heparin could have been inadequate but this applies to both groups as clotting times were not measured.

A further advantage of our method of treatment is that within the trial group there were significantly more late leg symptoms in patients treated with anticoagulants and surgery (73 per cent) than in those treated with anticoagulants alone (48 per cent). This indicates that if anticoagulants without surgery can be safely employed, it is clearly desirable.

SUMMARY

Techniques for phlebography of the leg and pelvic veins in patients with suspected

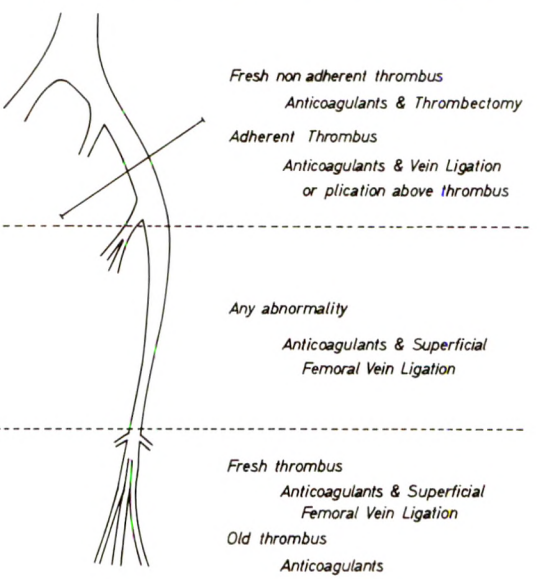


Fig. 8. Simplified plan of treatment used to prevent recurrent embolism. (Reproduced from *Brit. M. J.*[7])

venous thrombosis are described. The methods used are simple, rapidly performed, painless and free of complications.

It is suggested that phlebography is a reliable method for demonstrating the presence and extent of venous thrombosis and that treatment based on its use can reduce the recurrence rate of pulmonary embolism while minimizing the longterm complications of surgical treatment.

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ROENTGENOGRAPHIC APPEARANCE OF AORTO-CORONARY ARTERY BY-PASS USING A REVERSED SAPHENOUS VEIN*

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CORONARY artery surgery has been performed for over one decade using multiple techniques including endarterectomy, endarterectomy with patch graft reconstruction, and gas endarterectomy.[2,3] In the last few years, a more widely applicable method of treatment has been developed by Johnson and co-workers[6] and Favaloro.[4] In this method, a reversed saphenous vein graft is anastomosed between the ascending aorta and the coronary artery distal to the atherosclerotic obstruction.

The purpose of this paper is to outline briefly the surgical technique and to describe the angiographic appearance of the variously positioned venous by-passes.

SURGICAL TECHNIQUE

A saphenous vein is excised and reversed in order to avoid restriction of flow by the valves. The vein is anastomosed to the ascending aorta anteriorly and a few centimeters above the origin of the right coronary artery. When the left anterior descending or left main coronary artery is affected, a vein is connected either to the anterior descending or the diagonal branch of the left anterior descending coronary artery (Fig. 1). When the right coronary artery is by-passed, the reversed saphenous vein is anastomosed to the distal right or to the posterior descending coronary artery and proximally to the ascending aorta (Fig. 1). Note that on the left, the saphenous vein from the aorta arches cephalad over the main pulmonary artery before joining the anterior descending coronary artery (Fig. 1). When both the right and left anterior descending coronary arteries are diseased and need to be by-passed, the surgeon can perform either two separate by-passes as described above or a Y anastomosis. In the latter case, a right by-pass is performed in the usual fashion, but, in addition, a side-to-end anastomosis of a second vein graft is performed, which joins a branch of the left coronary artery and the right-sided vein graft (Insert of Fig. 1).

The surgeon marks the origin of the aortico-vein anastomosis by 2 metallic washers sutured on the adjacent aortic tissue above and below the anastomosis (Fig. 8).

ANGIOGRAPHIC TECHNIQUE

Vein by-pass angiograms are routinely obtained 2 weeks after aortico-coronary artery vein grafts.

The right coronary-vein by-pass angiogram is technically easy to perform. The saphenous vein has a large lumen and its

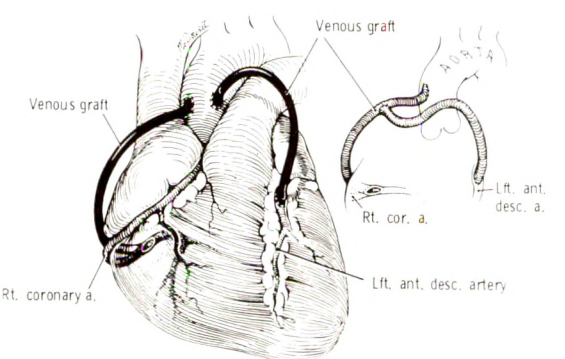


FIG. 1. Schematic representation of the aorto-coronary by-passes. The insert shows the Y by-pass procedure.

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entrance is marked by 2 washers. When one utilizes the femoral approach, a large right Amplatz coronary catheter[1] is used. The shape of this catheter is specific for the right vein graft. In fact, the tendency is for the catheter to penetrate too far into the vessel and one thus runs the risk of significantly restricting the flow. The by-passed vessel can be seen in anteroposterior and lateral projections in the following locations: in the anteroposterior projection, the graft goes to the right, following more or less the atrioventricular groove (Fig. 2). In the lateral projection, in distinction to the right coronary artery, the vein graft may follow the posterior surface of the heart until it reaches the posterior descending coronary artery (Fig. 3). When contrast material is injected, one can opacify the entire by-pass as well as the posterior descending and the distal right coronary arteries (Fig. 3). These angiographic findings suggest that the by-pass seems to increase blood supply to the myocardium. We have noticed that when the catheter is wedged into the venous graft and flow is restricted, angina may occur rapidly, which further indicates that the by-pass contributes to the perfusion of the myocardium.

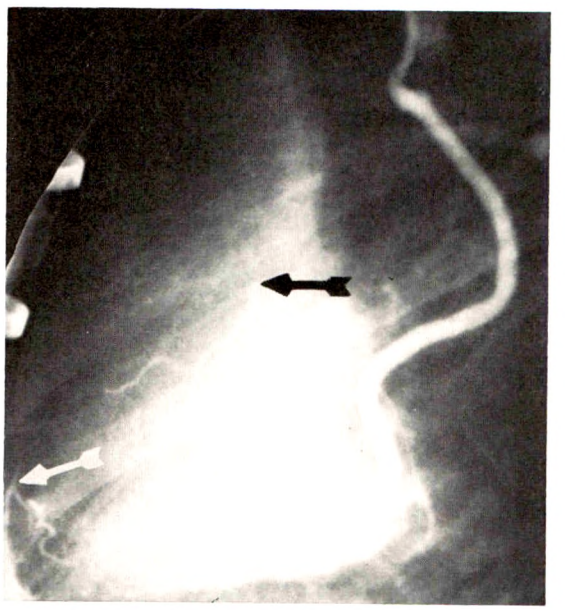


Fig. 3. Lateral view of a "right by-pass." Note opacification of the right coronary artery to the point of severe occlusion (black arrow). Via collaterals, the anterior descending coronary artery is also opacified (white arrow).

The left graft also commonly arises anteriorly, although more cephalic than

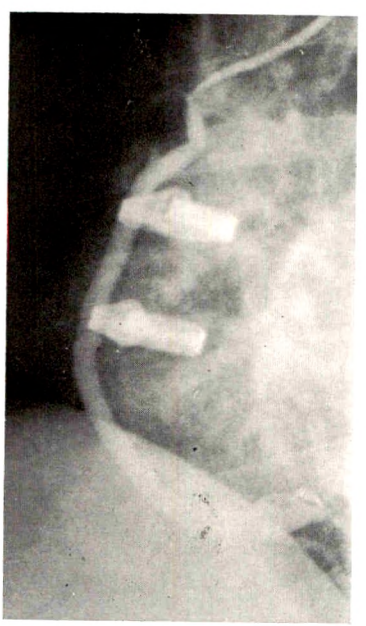


Fig. 2. Anteroposterior view of a "right by-pass." The vein is connected distally to the posterior descending coronary artery.

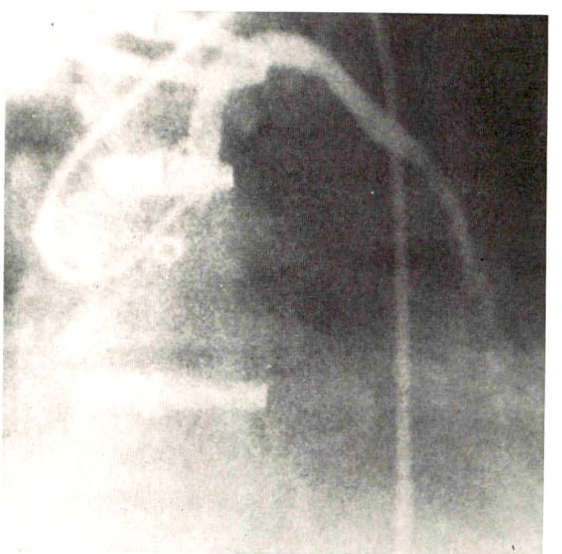


Fig. 4. Anteroposterior view of a single "left by-pass." Note the position of the catheter in between the 2 metallic washers. The vein is connected to the anterior descending coronary artery.

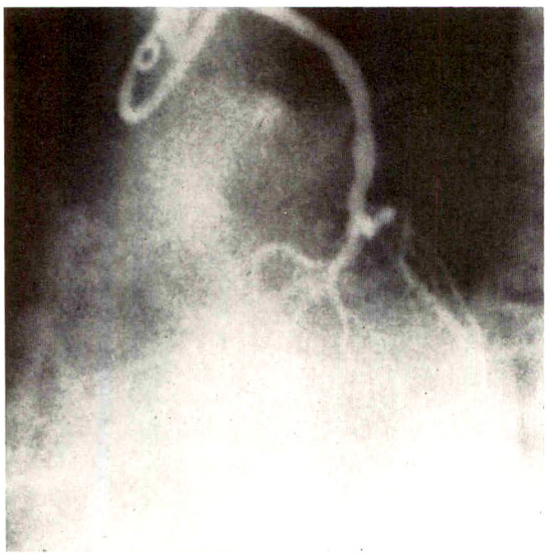


FIG. 5. Left anterior oblique projection of a "left by-pass." Note the retrograde filling of the main left coronary artery and of the circumflex coronary artery.

the right and more to the left of the aorta (Fig. 4). The left coronary catheter as described by Judkins[8] is not suitable for this catheterization. The left vein by-pass is directed in a cephalad direction from the aorta. A loop has to be formed in the catheter which will point upward in order to enter the vessel (Fig. 5). The shape of the right coronary catheter as developed by Amplatz and co-workers[1] is less satisfactory than one can fashion with polyethylene material, yet it can be used. We presently prefer the Type III "head hunter"[5] for catheterization of the left by-pass. This catheter is also suitable for the right graft and allows the radiologist to perform the entire procedure with one device.

Opacification of the left by-pass reveals the vein graft, as already mentioned above, arising from the aorta and turning cephalad over the main pulmonary artery until it communicates with the anterior descending coronary artery (Fig. 4). Such an injection can also opacify the diagonal branches of the anterior descending coronary artery as well as the circumflex artery when the main left coronary artery is stenosed or occluded; or when the injection pressure is high enough to cause retrograde flow from the point of anastomosis to the main left artery (Fig. 5).

Angiographic demonstration of the Y anastomosis is technically quite easy, but presents a larger risk. The origin of the Y by-pass in the aorta is identical to the origin of the right by-pass (Fig. 6 and 7). Injec-

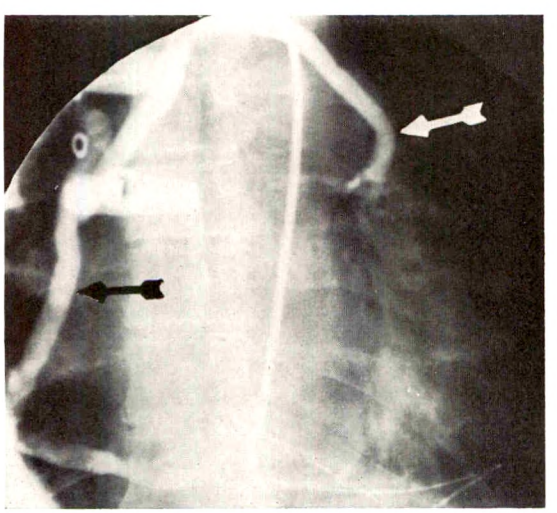


FIG. 6. Anteroposterior view of a Y by-pass. The black arrow shows the right limb, whereas the white arrow points to the left limb.

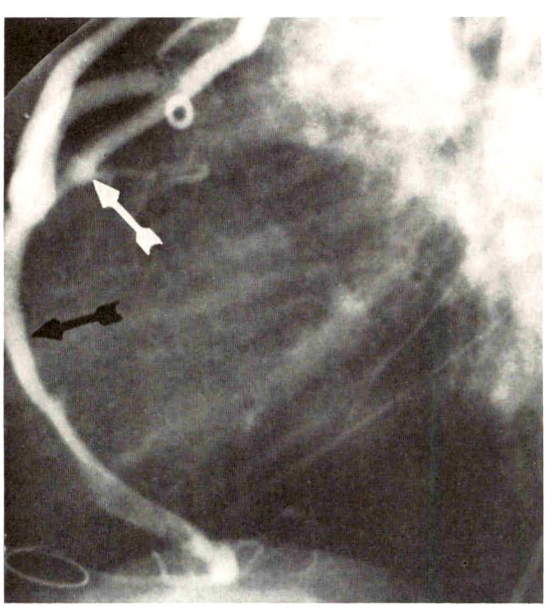


FIG. 7. Lateral projection of a Y by-pass. The black arrow points to the right limb. The white arrow shows the connection of the vein to the left anterior descending coronary artery.

tion of a Y by-pass can result in ventricular fibrillation, since both the right and left coronary arteries receive contrast material simultaneously. In our laboratory we encountered 1 case of ventricular fibrillation during such an angiography. After defibrillation, the procedure was continued without any ill effects to the patient. It is recommended that when a Y anastomosis is injected, smaller volumes of contrast medium be used and the speed of injection be decreased.

ANGIOGRAPHIC RESULTS

Follow-up angiography of patients having undergone such a surgical procedure is very important. Most grafts remain patent but we have seen several cases of a clotted by-pass. The occlusion usually occurs proximally close to the site of anastomosis to the ascending aorta (Fig. 8). When a Y by-pass clots, usually only 1 limb is affected (Fig. 9). Vein graft patency depends on the amount of distal blood flow, which itself is a function of the amount of myocardium supplied by the distal coronary artery, the amount of scarring present in the perfused myocardium, and the diameter of the coronary artery at the site of anastomosis.[7]

CONCLUSION

The anastomosis of a reversed saphenous vein by-pass between the aorta and the distal portion of a stenotic or occluded coronary artery is performed with the the hope of immediate improvement of myocardial ischemia. Pre and postoperative angiographic studies are necessary to carry out a successful procedure and to evaluate one's results.

Examples of these angiograms are presented and roentgenographic technical details are discussed.

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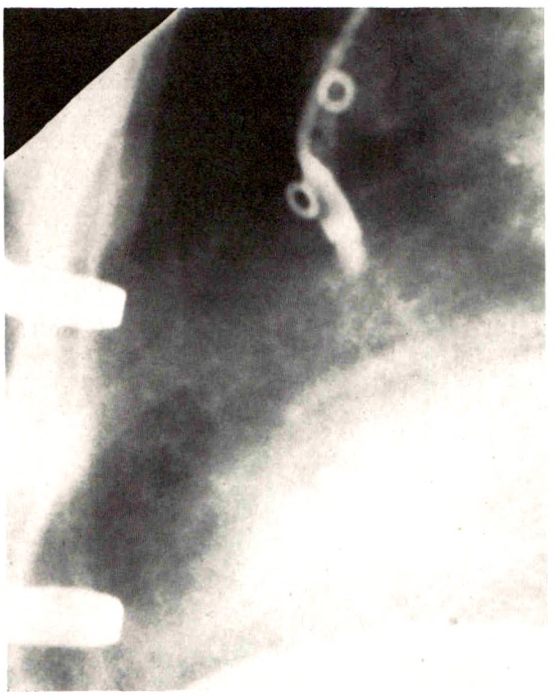


Fig. 8. Lateral view of an occluded "right by-pass." Note the position of the catheter in between the metallic washers.

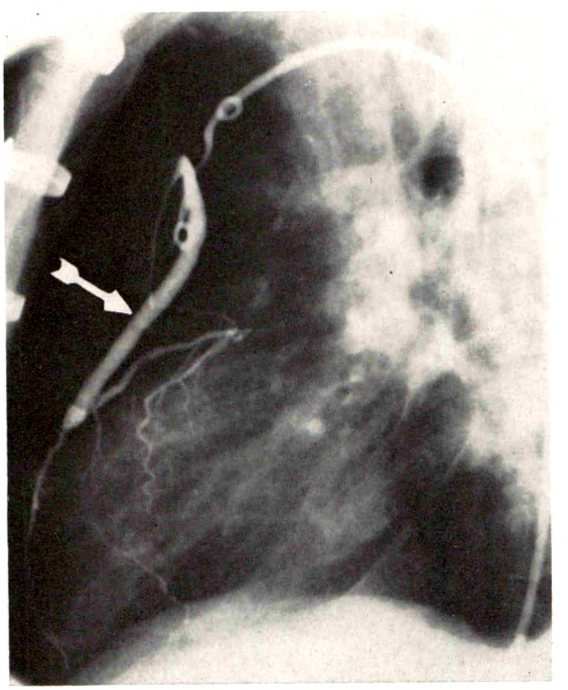


Fig. 9. Lateral view of a partially occluded Y by-pass. The white arrow points to the occlusion of the right limb. The left by-pass is connected to the anterior descending coronary artery. Note the retrograde filling to the origin of the first diagonal branch.

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Vol. 110, No. 4

REVERSIBILITY OF LEFT VENTRICULAR DYSFUNCTION FOLLOWING AORTO-CORONARY BY-PASS GRAFTS*

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CORONARY arteriography can adequately delineate the morphologic lesions of the coronary arteries. Also, the localization of severe coronary obstruction, as well as the presence or absence of collateral circulation in areas of the myocardium, can usually be correlated with abnormalities of left ventricular contraction on left ventricular angiography.[1,3,4] In a good proportion of patients, however, significant coronary artery obstructions are not accompanied by any detectable alteration of left ventricular contraction. Rarely, presumably in patients with cardiomyopathies, localized abnormalities of left ventricular contraction are observed without evidence of coronary artery disease.[3,4]

On the other hand, direct qualitative study of the left ventricular angiogram, when routinely associated with coronary arteriography, gives adequate information concerning left ventricular synergy and asynergy. Moreover, it has been shown that most patients with asynergy on the left ventricular angiogram exhibit some degree of left ventricular dysfunction, when assessed by other parameters.[3,4]

The purpose of this report is to demonstrate that left ventricular asynergy may be partially or totally reversible in a good proportion of patients with coronary artery disease following aorto-coronary by-pass grafts. Thus, coronary artery surgery can be successful not only to relieve angina pectoris but also to correct abnormal left ventricular dynamics in these patients.

SURGICAL MATERIAL AND TECHNIQUE

Between September 1969 and April 1970, 68 aorto-coronary by-pass grafts were performed on 53 patients (Table 1). Twenty-seven grafts were connected to the right coronary artery, 37 to the anterior descending coronary artery and 4 to the circumflex coronary artery. Thirteen double grafts and 1 triple graft were performed. Also, 13 internal mammary artery implantations were associated to by-pass grafts in a different location. The immediate postoperative evolution of these patients was surprisingly uneventful. The operative mortality was 3.7 per cent.

The surgical technique included a split sternal approach. A segment of saphenous vein was excised, inverted and carefully anastomosed in a termino-lateral fashion with multiple separate sutures of 6 O silk, under heart lung by-pass, first to a preselected healthy distal segment of a coronary artery, and then to the ascending aorta under partial occlusion of the aorta. A fairly long venous segment was used and care was taken that no tension was applied to the wall of the coronary artery after the heart was relaxed into its position in the chest.

MATERIAL OF THE STUDY

Forty-one patients with aorto-coronary saphenous vein grafts were re-evaluated angiographically between 12 and 20 days after surgery. All patients had had a selective coronary arteriogram and left ventricular angiogram at the time of their preoperative evaluation. The re-evaluation included selective opacification of the saphenous vein graft in different projections and a second left ventricular angio-

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Table I

AORTO-CORONARY BY-PASS GRAFTS FROM OCTOBER 1969 TO APRIL 1970

(53 Patients)

Site and Number of Anastomoses		Early Postoperative Result	
		Patency	Thrombosis
To right coronary artery	27	22	5
To anterior descending coronary artery	37	33	4
To circumflex coronary artery	4	3	1
Total*	68	58 (85%)	10 (15%)

* Mortality rate 3.7 per cent.

gram in all subjects. Selective coronary arteriography was repeated in only a few patients in whom it was felt indicated.

METHODS

Selective coronary arteriography and by-pass graft opacification were obtained by a percutaneous femoral approach using preformed polyethylene catheters. These catheter techniques and roentgenologic techniques have been previously described.[2,6]

Ciné-ventriculography was also performed through a retrograde percutaneous femoral approach. A power injection of 45 to 55 ml. of diatrizoate methylglucamine 76 per cent* was delivered to the left ventricle through multiple-hole catheters at a rate of 25 ml. per second. The ciné filming was made in a single plane, a 30° right anterior oblique position, with a 35 mm. Arriflex or Mitchell camera at film speed of 60 or 90 frames per second. The injection was carried out under fluoroscopic television control and magnetoscopic recording. The magnetic tape was reviewed immediately and the injection repeated when the procedure did not provide an excellent roentgenologic quality.

The ciné-ventriculogram was directly analyzed, on a Tage-Arno projector, for size, shape and wall motion both in continous movement and frame by frame. Following this, a sheet of paper was taped to the screen and the inner contours of the left ventricle were drawn successively at the end of systole and diastole during normally conducted beats. This superimposed image of left ventricular wall motion is useful to appreciate the extent and degree of left ventricular asynergy.

The left ventricle was considered normal when synchronous, concentric and uniform contraction of all its segments could be demonstrated. Alterations of left ventricular contraction, defined as asynergy, have been previously described[3–5] as follows:

* Renografin. E. R. Squibb & Sons, New York, New York.

Table II

COMPARISON OF PRE AND POSTOPERATIVE CINÉ-VENTRICULOGRAMS

(41 patients)

Group	Ventriculogram		No. of Patients	Comments
	Before Surgery	After Surgery		
A	Normal	Normal	11	
B	Normal	Worse	2	Appearance of inferior akinesis due to thrombosis of venous graft and distal right coronary artery
C	Abnormal	Unchanged	13	Thrombosis of graft in 5; persistent dysfunction in 8
D	Abnormal	Improved	15	Improved contraction but residual hypokinesis in 8; complete return to normal in 7

Hypokinesis or localized or diffuse reduction of ventricular contraction

Akinesis or absence of contraction in a portion of left ventricular wall

Dyskinesis or paradoxical systolic movement of an area of the ventricular wall

Asynchronism or lack of coordination in the contraction of different areas.

These alterations of contraction can be localized to the anterior, apical or inferior wall of the left ventricle. Wall thickness in the area of asynergy can also often be assessed with films of excellent quality.

RESULTS

The results, comparing the ciné-ventriculograms obtained before and after surgery in 41 patients, are shown in Table II. The patients are classified into 4 different groups.

In Group A, 11 patients had normal ciné-ventriculograms before and after surgery. Both left ventricular angiograms showed excellent contraction of all segments of the left ventricle.

In Group B, 2 patients had a normal ciné-ventriculogram before surgery with evidence of deterioration after surgery. In both instances, thrombosis of the saphenous vein graft and distal right coronary artery resulted in the appearance, on the postoperative angiogram, of an akinetic portion of the inferior wall of the left ventricle.

In Group C, 13 patients exhibited, on the preoperative angiogram, alterations of left ventricular contraction which remained essentially unchanged after aorto-coronary by-pass. Five patients of this group had thrombosis of the saphenous vein graft and this could explain the absence of improvement noted after surgery. In the other 8 patients, there was persistent abnormal contraction of the left ventricular wall in the area where a patent graft had been implanted.

Group D consisted of 15 patients with abnormal ciné-ventriculograms before surgery which improved following surgery. The improvement was partial in 8 patients and complete recuperation of a normal contraction occurred in 7 patients.

Table III

VENTRICULAR CONTRACTION AFTER SUCCESSFUL AORTO-CORONARY GRAFTS

(23 patients)

Postoperative Results		
Complete Return to Normal	Improved Contraction but Residual Hypokinesis	Unchanged
7 (30%)	8 (35%)	8 (35%)

Thus, 23 patients had patent aorto-coronary grafts and abnormal preoperative ventriculograms (Table III). Fifteen, or 65 per cent, showed either improvement or complete recuperation of ventricular asynergy, whereas 8, or 35 per cent, exhibited no change after surgery.

The results were also studied in relation to the pattern and degree of preoperative left ventricular asynergy (Table IV). Mild or moderately severe hypokinesis was present in 8 patients. In 7 of these subjects, complete return to a normal pattern of left ventricular contraction occurred after aorto-coronary by-pass surgery. Akinesis, on the other hand, was present on the preoperative angiogram in 15 patients and improved in 8, sometimes markedly, with persistence, however, of some degree of hypokinesis. In the other 7 patients, the akinesis remained unchanged.

In Table V, the postoperative results were compared to the site of ventricular asynergy before surgery. The alterations of contraction were localized to the inferior wall of the left ventricle in 6 patients. Following by-pass surgery on the right coronary artery, these abnormalities remained unchanged in 5 patients and a single patient with hypokinesis exhibited a return to a completely normal pattern of left ventricular contraction. On the other hand, one can see that, following by-pass surgery

TABLE IV

POSTOPERATIVE RESULTS OF VENTRICULAR ASYNERGY

(23 patients)

Pattern of Ventricular Asynergy Before Surgery		Postoperative Results	
Hypokinesis	8	complete return to normal in	7
		unchanged in	1
Akinesis	15	complete return to normal	0
		partially improved in	8
		unchanged in	7

to the anterior descending coronary artery in 17 patients, improvement of the akinetic segment of the anterior wall of the left ventricle was demonstrated in 8 patients. Also, all 6 patients with hypokinesis of the anterior wall exhibited a complete return to a normal pattern of left ventricular contraction.

Figures 1 and 2, *A–D* illustrate the case of a 40 year old white male with a history of myocardial infarction 4 years previously and typical severe angina pectoris. The electrocardiogram showed an old inferior wall myocardial infarction with inferior and anterolateral ischemia. The vectocardiogram also suggested a strong probability of infarction in the anteroseptal area. The selective coronary arteriogram showed complete proximal obstruction of the anterior descending coronary artery with distal reopacification from collaterals and a 90 per cent obstruction of the right coronary artery past its anterior marginal branch. At surgery, the anterior descending and right coronary arteries were bypassed below their obstruction with saphenous vein grafts (Fig. 1). The preoperative angiogram shows diffuse severe hypokinesis of the anterior, apical and inferior walls of the left ventricle (Fig. 2, *A* and *B*). The postoperative angiogram, on the other hand, illustrates the marked improvement in the contraction of all segments and also an important reduction in the volume of the

TABLE V

POSTOPERATIVE MODIFICATION OF VENTRICULAR ASYNERGY ACCORDING TO THE SITE OF ABNORMAL CONTRACTION

(23 patients)

Site of Abnormal Contraction		Preoperative Ventriculograms		Postoperative Ventriculograms	
Inferior wall	6	Akinesis	4	unchanged	4
		Hypokinesis	2	unchanged	1
				return to normal	1
Anterior wall	17	Akinesis	11	unchanged	3
				improved	8
		Hypokinesis	6	return to normal	6

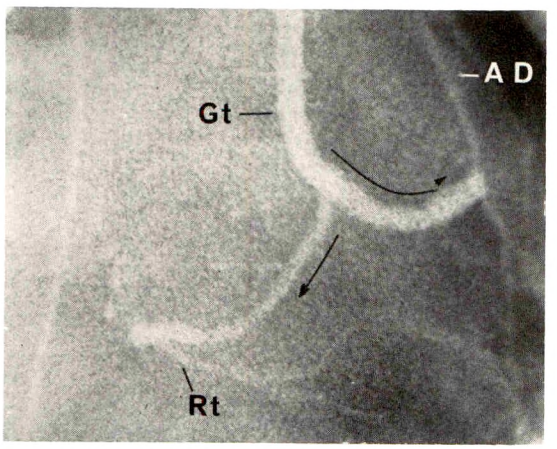


Fig. 1. Case 1. Aorto-coronary venous graft (Gt) to the anterior descending artery (AD) and distal right coronary artery (Rt).

left ventricle (Fig. 2, *C* and *D*).

Figures 3, *A* and *B* and 4, *A–D* show the case of a 46 year old white male with severe typical angina of 1 year duration. There was no evidence of myocardial infarction clinically and on the electrocardiogram or vectocardiogram. The selective coronary arteriogram demonstrated a severe 85 per cent stenosis of the main trunk of the left coronary artery and also a 60 to 65 per cent stenosis of the proximal third of the anterior descending coronary artery (Fig. 3*A*). A saphenous vein by-pass was placed distal to the obstruction of the anterior descending coronary artery and, at re-evaluation, the angiographic contrast medium was seen to give good opacification not only of the distal segment of the artery but also of the proximal part, with good opacification of the circumflex and diagonal arteries (Fig. 3*B*). The preoperative left ventricular angiogram showed severe hypokinesis of the anterior and apical segment

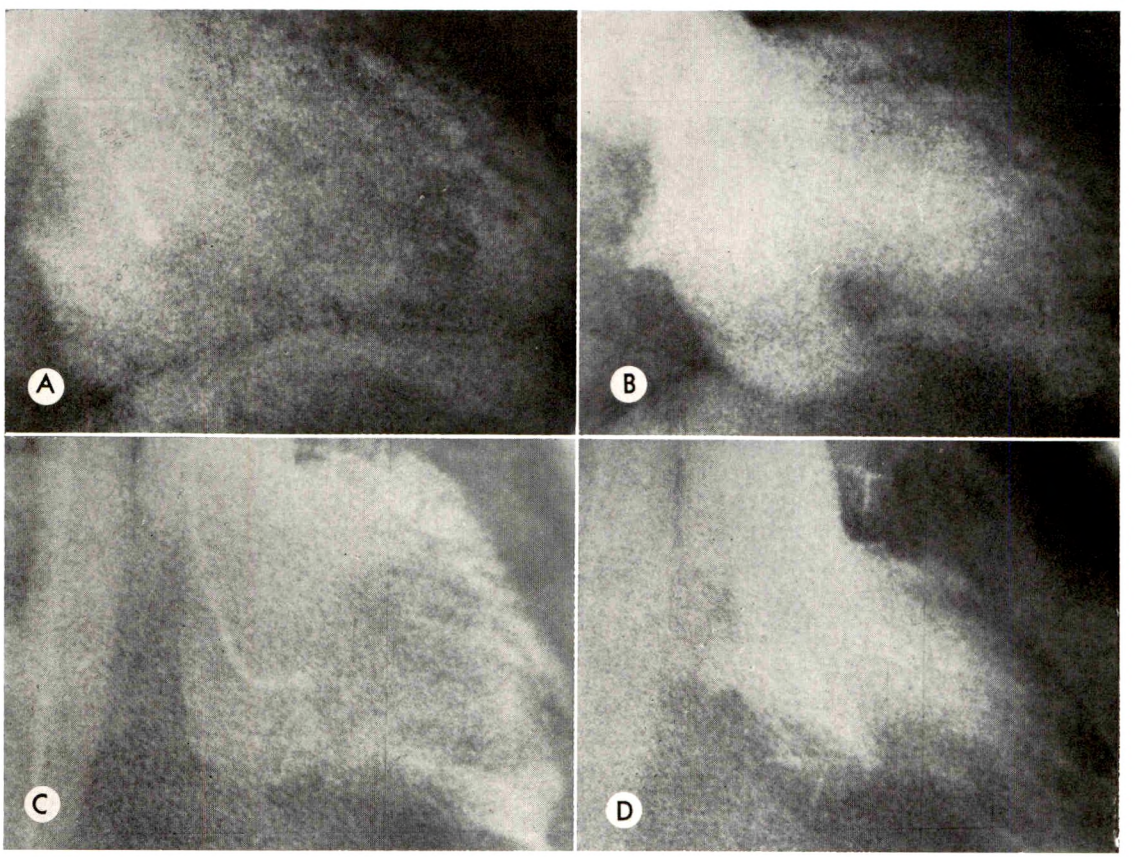


Fig. 2. Case 1. (*A*) Preoperative end diastole and (*B*) preoperative end systole left ciné-ventriculograms (RAO projection). Severe hypokinesis of anterior and inferior walls; enlarged left ventricle. (*C*) Postoperative end diastole and (*D*) postoperative end systole ciné-ventriculograms. Marked improvement of contraction and size of the left ventricle.

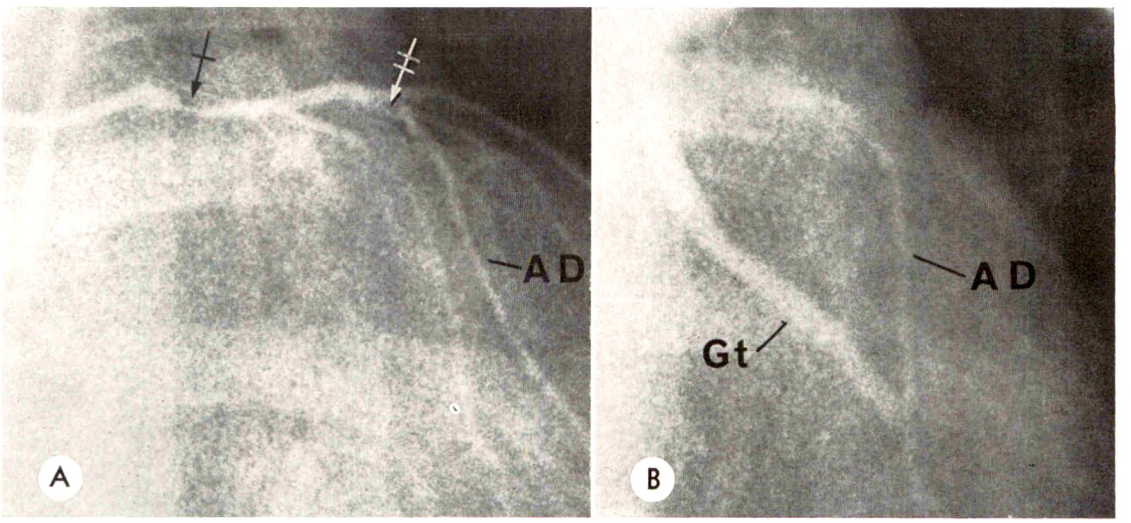


FIG. 3. Case II. (*A*) Selective ciné-coronary arteriography. Severe stenosis (85 per cent) of the main trunk of the left coronary artery (⊣→) and a 65 per cent stenosis of the proximal segment of the anterior descending artery (⫣→). (*B*) Aorto-coronary bypass graft (Gt) to the middle third of the anterior descending artery (AD).

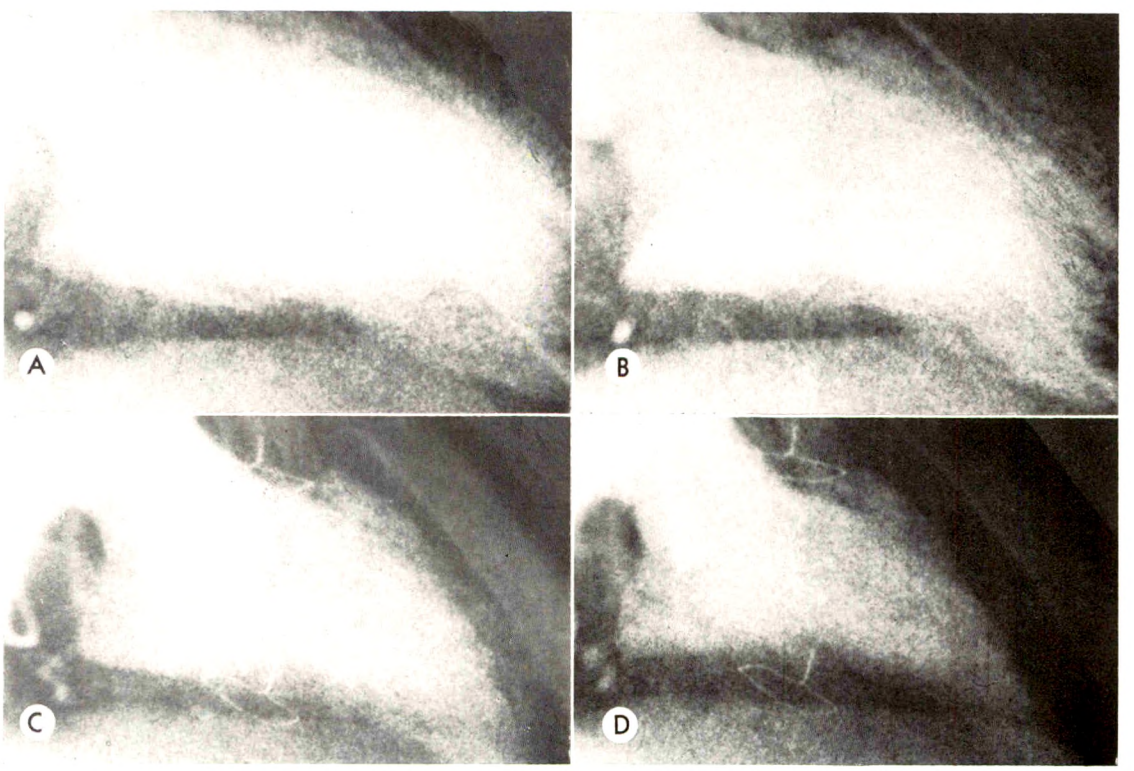


FIG. 4. Case II. (*A*) Preoperative end diastole and (*B*) preoperative end systole left ventricular ciné-angiograms. Important hypokinesis of anterior and apical segments of left ventricle. (*C*) Postoperative end diastole and (*D*) postoperative end systole angiograms. Excellent recuperation of left ventricular contraction with reduction in the size of ventricular cavity.